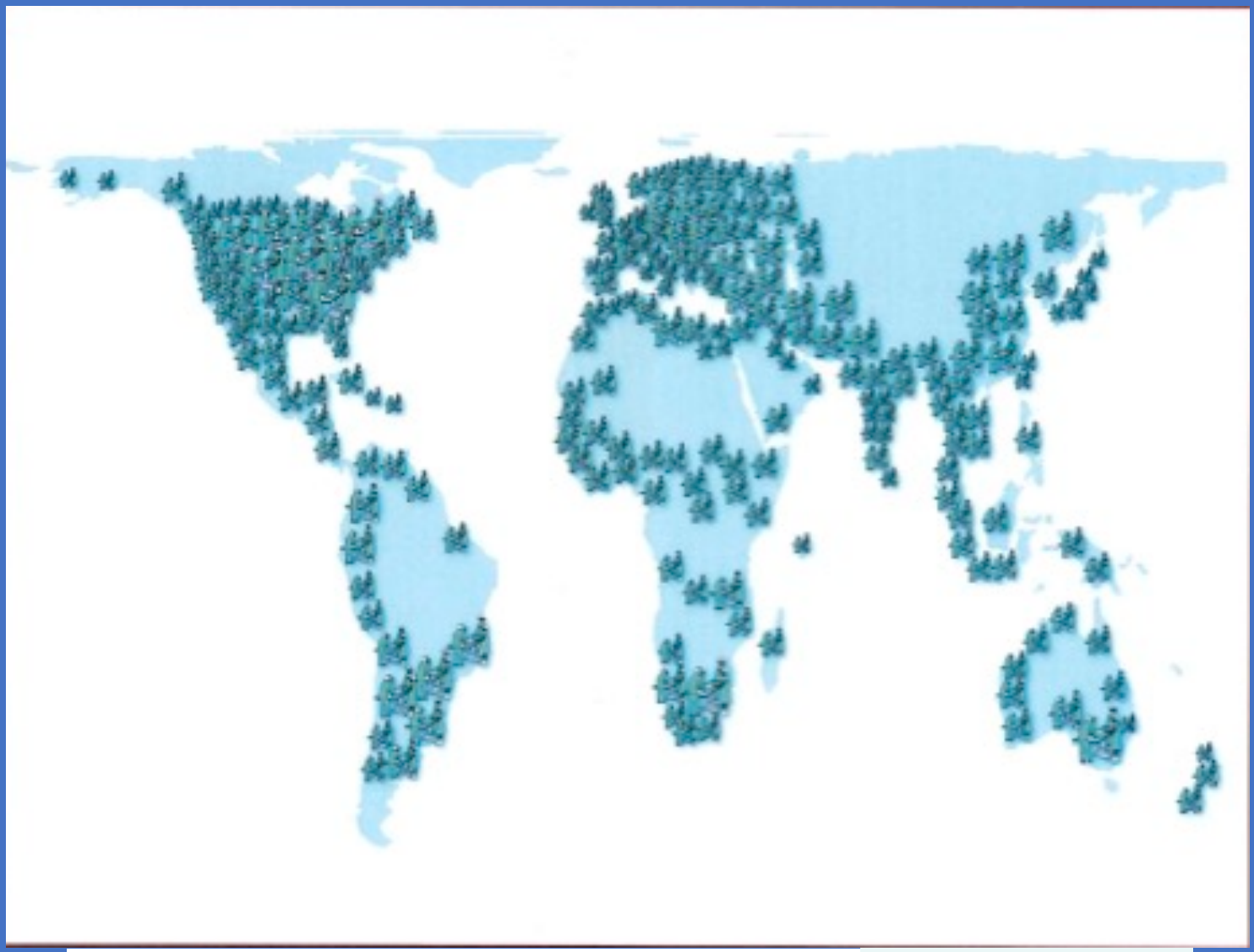
Primary Surgery

[Volume One: Non-Trauma]

2nd edition (revised)

Chief Editor: Michael Cotton



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Third World Network

Deutsche Gesellschaft für
Globale und Tropenchirurgie
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GLOBAL HELP

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Volume One: Non-Trauma

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Volume One: Non-Trauma

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The cover illustration attempts to show (with some artistic licence) the maldistribution of surgeons around the world, depicted with the famous Gall-Peters projection which better demonstrates land mass than the more traditional Mercator projection.

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Primary Surgery
Volume One: Non-Trauma
(Second edition, revised)

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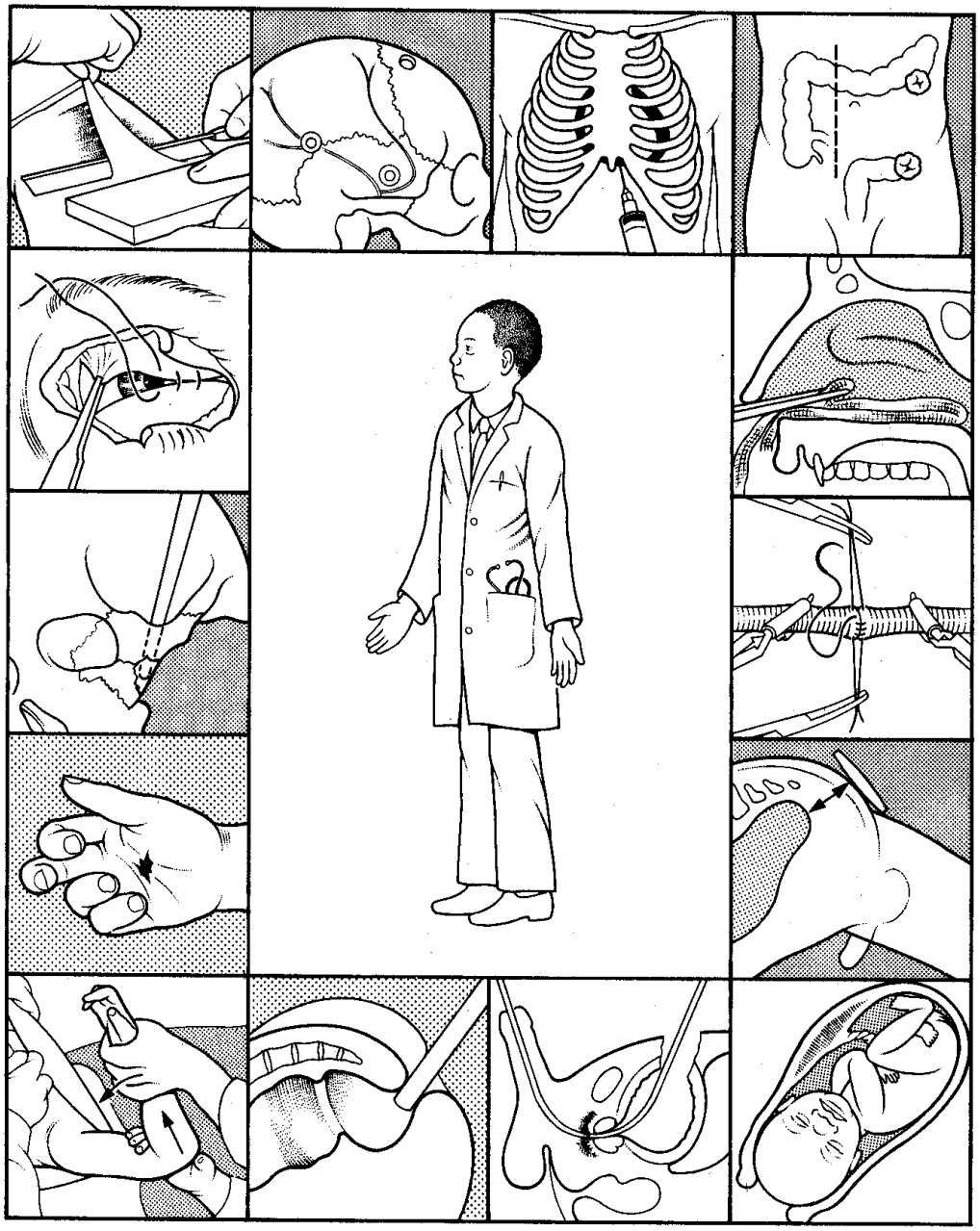
Appendix A. Grades of operation.
Appendix B. Difficulties with the index.
Appendix C. Abbreviations.

Note on numbering

1.1 Refers to a written section in the text with its own subject heading.
1-1 Refers to a figure in the text.

Chapters are numbered with dots (e.g., 8.3) and illustrations with dashes (e.g., 4-12). An A at the end of a number, for example 2-7A, refers to the first illustration in a particular figure.

AT LEAST 20 SURGEONS IN ONE



So, here you are, one of our readers, faced with the difficult problem of helping a surgical patient in all these fields, and unable to refer him to an expert. Reading from the top left in a clockwise direction, you may need to be: a plastic surgeon, a neurosurgeon, a thoracic surgeon, a GI surgeon, an ENT surgeon, a vascular surgeon, a paediatric surgeon, an obstetrician and gynaecologist, a urologist, a proctologist, an orthopaedic surgeon, a hand surgeon, a maxillofacial surgeon, and an ophthalmic surgeon.

This drawing does not include your role as a trauma surgeon, a dental surgeon, a leprosy surgeon, an HIV specialist, an oncologist, an anaesthetist, and an 'intensivist', in addition to doing everything else you have to do in medicine, paediatrics, psychiatry, and management! We hope this manual will help you in some of these varied and exacting tasks.

Remember though the famous wise words of the London surgeon Sir Astley Cooper:
"A surgeon should have an eagle's eye, a lady's hands and a lion's heart."

You must be humble; surgery is a craft that makes use of the scientific method of Popperian falsification. The art of surgery consists of judgment and the beauty of an operation well done, done gently, with respect for living tissue, for every cell, with reverence for form and function, carried out with compassion, always remembering that the only justification for invading the body of another individual is the intent to restore homeostasis.

Imre J.P. Loefler, Surgery in the Post-Colonial World (Rahima Dawood Oration),
E & Centr Afr J Surg 2002; 7(1): 53-8

No person is so perfect in knowledge and experience that error in opinion or action is impossible. In the art of surgery, error is more likely to occur than in almost any other line of human endeavour; and it is in this field that it should be most carefully guarded against, since incorrect judgement, improper technique, and a lack of knowledge of surgical safeguards may result in a serious handicap for the rest of the life of the patient, or may even result in the sacrifice of that life. For the surgeon, perfection in diagnostic skill is of equal, if not more, importance than operative skill.

Max Thorek, *Surgical Errors and Safeguards*, J.B. Lippincott, USA, 1960

Any doctor who has worked in a developing country will not easily forget the widespread and pathetic evidence of surgical neglect in the villages. Huge hernias and hydrocoeles, unsightly lumps on the faces of women and children, and the compound fractures infected with maggots bear testimony to the failure of so many countries to provide even a basic level of surgical care for their people.

Samiran Nundy, How might we improve surgical services for rural populations in developing countries?,
BMJ 1984; 39(10): 71-2

Patients should be treated as close to their homes as possible in the smallest, cheapest, most humbly staffed, and most simply equipped unit that is capable of looking after them adequately.

Maurice King, *Medical Care in Developing Countries*, Symposium from Makerere, Uganda, OUP, 1967

Foreword

When Michael Cotton invited me to write the Foreword to this second and significantly changed edition of *Primary Surgery*, I was delighted, and I have three reasons.

First, I greatly respected Michael's work as a frontline surgeon and a most dedicated teacher of surgery during his many years in Bulawayo, Zimbabwe. I was often asked by colleagues in training where they should go to learn operative surgery in Africa; Michael was always the one who came to mind first, because I knew that he would take the trouble to teach sound, careful and relevant surgery. I knew also that his colleagues would be working with a man of resolute integrity. This book is the expression of all his work as a surgeon at the front line. It was a further delight when I found that he had recruited Olive Kobusingye to be his assistant editor. I have been with Olive on take-in evening ward rounds at Mulago Hospital, Kampala; I witnessed teamwork, clear thinking and the practice of excellent clinical surgery. Thus this book has editors who have been proved as teachers and surgeons.

My second reason for delight is that the book will be a real help to those who have to practise surgery at the front line. For too long such colleagues, whether surgical clinical officers or medical officers, have not had a book which was written for them, to enable them to treat rural patients, the injured and those who are unable to meet the costs of travel to, and accommodation at, a regional or national teaching hospital. I believe that if the book's sound common sense and clear practice are followed, the victims of injury will be treated early and acute emergencies will be dealt with before they progress and complications develop. Patients will thus be able to get back to work and families will not suffer socially and economically.

Finally, I am certain that, where good life-saving and worker-restoring surgery is done, people who may have been afraid to bring their family member to hospital will lose that fear. Good surgery will be a great advocate and foundation for the public health of a community, now assured that disease and injury which previously could not be treated are not only treated but treated successfully. Surgery will no longer be forgotten by the administrators and those who are responsible for providing a nation's health service; it will take its rightful place in health care. This book, properly used, will help to accomplish this and will be blessed by many whose surgical needs have been met by the skills which it has helped to develop. I wish it well as I confidently expect its readers to enjoy successful and fulfilling primary surgical practice.

The late Sir Eldryd Parry, OBE
Visiting Professor and Honorary Fellow, London School of Hygiene and Tropical Medicine
Chairman, Tropical Health Education Trust
Former Professor and Dean of Medicine, Accra, Ghana; Addis Ababa, Ethiopia; and Ibadan, Nigeria

DEDICATION

This book is dedicated to the world's poor, so that when they need surgical help, they may get it, and it may be done well.

ACKNOWLEDGEMENT
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Preface

The first edition of this work by Maurice King appeared in 1990 and has established itself as the gold standard of do-it-yourself guides to surgery in the up-country hospital. Some 50,000 hours of work went into compiling the mass of expert contributions from many varied and far-flung individuals, all enthusiasts with a first-hand indigenous experience of surgery in poor-resource environments. The need for such a book has been amply justified, and 34 years on, its usefulness is in no way diminished. Indeed, there is even greater urgency for such appropriate basic surgical guidelines to be disseminated in parts of the world where people's access to surgery has been difficult or well-nigh impossible. To this end, it is envisaged that this manual – which comprises two volumes – will be translated into French, Spanish, Portuguese, Russian and Chinese, and also produced electronically as compact discs. Publication on a freely accessible website will allow more readers access throughout the world.

In this revised edition, more is included on pathology seen in different parts of the world, *viz.*, Chagas' disease in South America, hydatid disease in Asia, *schistosomiasis* in Egypt, and so on. There will remain gaps, as different hospital environments will always differ hugely: suggestions for alterations and inclusions will always be gratefully received, and incorporated in future editions, which can now be updated electronically much more easily than heretofore.

The single most dramatic change in the practice of surgery in much of the developing world, and in Africa in particular, has been the rampant, inexorable spread of HIV disease since the early 1980s; this has seen the appearance of new pathologies, and the requirement that new strategies be developed not only to combat its spread, but to deal with its effects. By the new millennium, antiretroviral medication was still seriously beyond the scope of most government health systems, but this is changing. Whilst the advent of therapies to combat HIV effectively may still remain out of reach for very many, it will offer hope to the young and those yet unborn that this scourge may be controlled, if only by encouraging victims of the disease to be tested. Until recently, so much obfuscation around the disease, and slavish following of individualistic ideologies, has prevented much community openness concerning this epidemic; it is fervently hoped that with cheaper and successful antiretroviral treatment, the exceptionalization of HIV may disappear.

Much therefore of the changes since the First Edition has concentrated on the impact of HIV disease; however, other changes are noted: for example, the inclusion of ultrasound and flexible endoscopy, which, though the equipment is expensive (it might be sourced through donor agencies), are highly cost-effective in diagnostic yield. Further, thyroid surgery is no longer excluded, as its performance is considered no more complex than much else described. The inclusion of grading of difficulty of operations, as mooted in the First Edition, has been carried out: this scale is inevitably idiosyncratic and is offered simply as a guideline, especially for surgical technicians. Furthermore, various procedures which are in danger of being lost to the experience of Western-style practitioners and their trainees but are eminently useful in poor-resource settings, have been described in some detail.

It is rare that a book tells its reader what not to do, and what to do when things go wrong! This is such a book, whose aim, essentially, is to encourage surgery in the districts and remote areas, if necessary by non-specialist, even non-medical, practitioners. The realization that surgery is not an expensive luxury but a cost-effective intervention is slowly dawning on health planners; however, to remain viable, such surgery must remain relevant and relatively low-cost. It is estimated that 80% of surgery necessary can be covered by 15 essential procedures. If even only these are mastered, the surgical contribution offered will be substantial.

The editors' view is that laparoscopic surgery is not at this stage a generally viable adjunct, and it is therefore not described. Where special arrangements have been made, and a surgeon with appropriate skills is available, the benefits should not of course be denied patients in rural environments. Nonetheless, the greater danger is that surgery is not done simply because of the unavailability of highly trained individuals or of high-technology equipment presumed essential, and this must on all counts be avoided. Appropriate technology has been described, and inventions made known through the practical insights of many in poor-resource settings have also been included. This must be further encouraged; indeed, the principles thus discovered should be exported to the so-called knowledgeable rich world, which groans under the ever-increasing cost and bureaucratic complexity of delivering high-technology medicine.

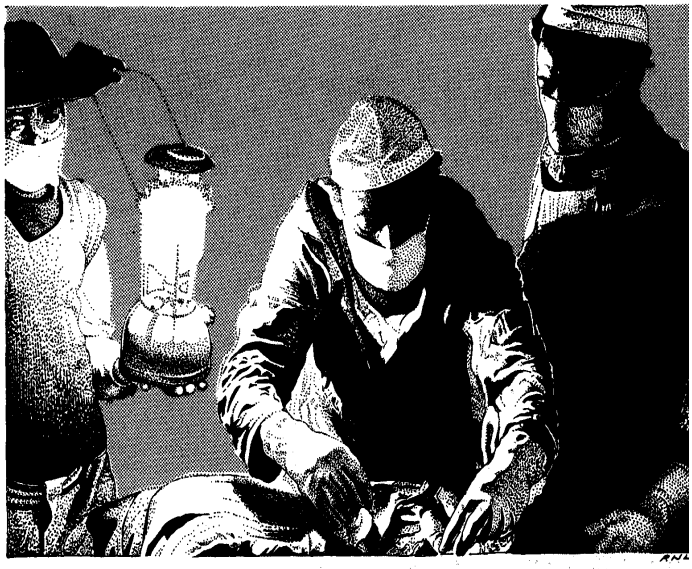
It is the fervent hope that this Second Edition (revised) will bring relief and benefit through surgery to millions to whom it might otherwise be denied. The fact that some 2 billion people in the world don't have access to any surgery must be seen as a scandal, and this book will do its part in correcting this tragedy.

This updated Second Edition corrects some errors, and introduces advances, particularly regarding Caesarean section. It must be noted that antibiotic use and availability (and HIV regimes) vary from country to country; our recommendations are only guidelines, and not absolutes. Local traditions, however, are often much less scientifically justified! The patient remains the priority!

1 The background to surgery

You have just arrived at your hospital and have not yet unpacked, when the ambulance arrives with a note from the sister-in-charge to say that there is a patient with a strangulated hernia waiting for you. You have never done one, because you were left doing the paperwork when you did your internship and your senior wanted to do as much operating as he could himself. So most of the time you assisted and were occasionally allowed to suture the skin. All your seniors have now left and have gone into private practice, so there is nobody to help you. If you refer this patient, he may die on the way.

This manual is addressed to 'you'. The personal reminiscence above was contributed by Dr Michael Migue of AMREF, as describing the type of scenario for which the manual is needed.



ONE OF OUR READERS. You may have had very little surgical experience and yet have to operate on severely ill patients. In an emergency you may even have to operate by the light of a hurricane lantern. The light will attract insects, and these will fall into the wound, but even so they are unlikely to influence the patient's recovery. However, an LED head torch will be preferable in such situations, and ketamine anaesthesia is virtually always feasible! *Kindly contributed by WHO.*

1.1 The unmet need for surgical care

The attraction for patients and practitioners alike of surgical cures is that they are a 'once-only' phenomenon. For the patient, surgery is therefore something that can reasonably be borne stoically, and for the practitioner, surgery can be a source of intense satisfaction.

Both can witness an often dramatic transformation of a critical to a normal situation. Health planners are beginning to realize that surgery is socially and economically cost-effective. This is true for elective as well as emergency interventions, but especially so for trauma (the subject of Volume Two of this manual). Surgery need not be complicated, and should not be made unnecessarily expensive.

Surgically treatable diseases may not be as numerous as the great killers of small children in the developing world – malnutrition, pneumonia and diarrhoea – but are soon to overtake them! They represent 11% of the global burden of disease. Surveys suggest that in low- and middle-income countries (LMICs), 8% of all deaths, and almost 20% of deaths in young adults, are the result of conditions that would be amenable to surgery in the industrial world. If even very simple surgical services were available, two-thirds or more of these deaths would not have occurred. What is more, for every person who dies of an accident, there are at least eight who were permanently disabled. Estimates are that the maternal mortality rate (MMR) is >340,000 per year, and probably <10% of mothers who need a Caesarean section get one done. Only 1 in 10 who need an inguinal hernia repair get it done, and since a strangulated hernia is almost always fatal unless it is treated, this is a mortality rate of nearly 90%. For emergency laparotomies the situation is worse: of 50 who need such an intervention to save their life, only one gets it done!

These are just some statistics of the surgery that needs doing and is not done. It is estimated that <3.5% of all surgical interventions done worldwide are done in low-income countries. Since most of these procedures will be minor ones, it is probable that <½ million major operations are done per annum in these countries.

All this unmet need means that there are many unnecessary deaths from strangulated hernias and obstetric disasters, as well as from vesico-vaginal fistulae (VVF) and from foetal cerebral injury or anoxia at birth. They illustrate the fact that hospitals are only coping with a fraction of the burden of surgical disease in the communities around them. The result is that millions of people, whom surgery might help, get no help. Too many people still die from obstructed labour or obstructed bowel, or are disabled by untreated osteomyelitis, or burns contractures, much as they were in the industrial world a hundred years ago.

If we wait till services are available to prevent the killing diseases of childhood, the simple surgical services described here will not become available for a very long time. They can do much to improve the quality of life of the poor.

Although much of this manual has a rural orientation, 44% of the people of the developing world are now living in towns, so the surgical care of the urban poor is almost equally important. As at 2010, 9 nations in sub-Saharan Africa (Angola, Botswana, Cameroon, Congo, Gabon, Gambia, Ghana, Liberia, Nigeria) have >50% of their population living in towns. There were no such countries in Africa in 1950. Practically all South American and Far Eastern nations have a majority of people urbanized. There is therefore an urgent need for 'district hospitals' in towns, leaving only specialized care to the central institutions. Furthermore trauma presents an increasing burden of morbidity and mortality in the developing world, and as the success of its management depends mainly on early rapid appropriate surgical care, this onus falls on the district hospital in the first place.

Surgery has an importance in the public mind that medicine does not have. It is also the most technically demanding of the tasks of a district hospital doctor or clinical officer, and is thus a good measure of the quality of his medical education. If this has not been adequate, either because it never was adequate in the medical school, or because the quality of its teaching has fallen, he will be very loath to do much surgery, and may do none. This is why many rural hospitals and several district hospitals in some countries do little surgery. When this happens, patients soon realize that it is no use going to such hospitals, with the result that they soon have empty beds. So if you see a hospital with empty beds, one of the first questions to ask yourself is: "What is the quality of the surgery here?" There is thus a qualitative aspect to the unmet need for surgical care as well as a quantitative one.

The constraints on the provision of surgical care are formidable, but some hospitals have succeeded in increasing their workload and their operations tally despite rising costs and scarce manpower resources.

1.2 The surgical scene

The countries of the Third World and the surgical scene within them differ widely. Ethiopia and Paraguay, for example, are about as different as two countries could be. Typically, the people of low-income countries are poor, hungry, and rural, although they are rapidly migrating to the towns. The population of sub-Saharan Africa is increasing at an inexorable 3% annually, although in some countries there was a negative growth rate due to deaths from HIV disease. Meanwhile its *per capita* food production and its already meagre gross national product even if increasing remain hugely unevenly distributed, whilst costs on the military and socially dislocating wars multiply.

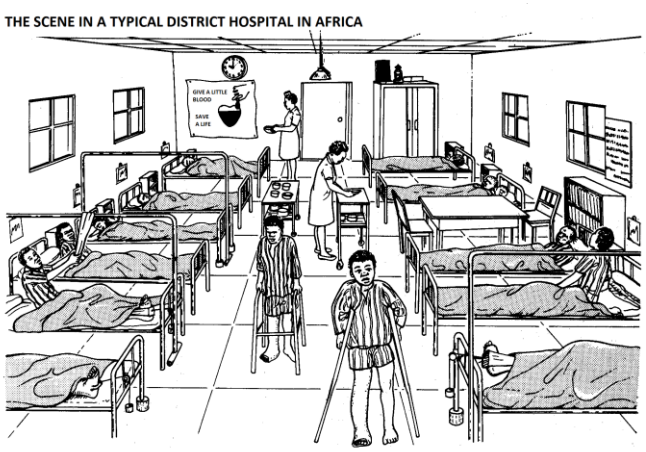


Fig. 1-1 THE SCENE IN A TYPICAL DISTRICT HOSPITAL IN AFRICA. This is *somewhat better* than the average conditions for sub-Saharan Africa at the time of writing. Note the blood transfusion poster. You will see that there are several patients on traction, two with long leg casts, and that one of the beds contains 2 patients.

It is obscene that the richest 1% own half the world's wealth. One feature developing countries do have in common is that much of the surgery should be done in 'district hospitals'. These typically have between 60 and 200 beds and are staffed by 2-4 doctors, assisted by nurses and auxiliaries. Fortunately, the 'one-doctor hospital', which was common until recently, is now unusual. Each hospital typically serves about 150-250,000 people living in an area which may be as large as 3,000 square miles.

Over the world as a whole these hospitals range from the excellent to the indescribable. At one end they provide care which anyone would be fortunate to have, at the other the few patients brave (or foolish) enough to enter them lie largely untended. Nonetheless these hospitals are the local focus of health care in the community and have an important place as such, as well as being a major employer of labour. How much your hospital is valued by the Government can be measured by whether the Minister of Health or his officials would be willing to be treated at your institution, or whether they will use scarce resources for treatment in a richer country with 'better' facilities.

If you work in a hospital in the middle or at the lower end of this spectrum, expect to find your wards overcrowded, with more than one patient in a bed. 'Clean' and infected cases may not be separated, so that a patient with an open fracture may lie next to one with a perforated typhoid ulcer. Your maternity ward is likely to be particularly overcrowded and resist all your attempts to decongest it. Cultural reasons may make it impossible to restrict the number of visitors to the wards. Defects in their construction will make keeping them clean and tidy a major task. Your equipment will be limited and poorly serviced. When it does break down, it may take years to replace. Trees may be so scarce that your staff have to go a long way to collect firewood.

If your hospital is at sea level on the equator, expect to operate at 30^0C in 95% humidity, with your clothes wet, and everything which can go rusty or mouldy doing so. Only insects enjoy such conditions, and you will find plenty of them. If your hospital is at high altitudes, expect problems with sterilization (water boils at lower temperature) and with smoke from numerous fires.

You may have to rely on locally trained staff with only primary education who have not had training relating to the idea of sterility. Most of them will experience considerable hardship, and be so poorly paid that they will have to grow the food they need. Their ability to monitor a patient post-operatively on the wards may be so poor that you may be forced to assume that, once a patient has left the *theatre*, he is on his own as far as recovery is concerned.

Your anaesthetic facilities will vary greatly. If you are lucky you will have 2 or 3 anaesthetic assistants, trained to do most of the methods described in *Primary Anaesthesia.* You may have the services of well-trained surgical technicians who, without formal medical training, can carry out most of the surgical procedures required very adequately. You will rely on them more and more! Your laboratory facilities will usually be minimal.

Although HIV has made it much more dangerous in many areas, blood transfusion should always be possible, if you can put enough effort into organizing it. Often, relatives will give blood for a patient, but for nobody else, but *don't ignore the HIV risk just because the blood comes from a close relative!* You may have to try to make your own IV solutions or rely on relatives to purchase essentials outside the hospital.

So be prepared to find everything, or nothing. You may have expensive equipment given by charitable organizations: some of it may well be lying idle, because no one knows how to use it, what it's for, or how to maintain it. However, on occasion expect to find no water, no steam, no linen, no gauze, no bandages, no sutures, no local anaesthetics, no gloves (or only gloves with holes in them), no plaster (or only plaster that does not set) or no intravenous fluids. When you need to prepare for a laparotomy, expect that no instruments have been prepared beforehand. When you go into the maternity ward late one night, be prepared for the last sphygmomanometer to be missing. Try not to blame your staff too harshly, they may not be responsible; and even if they are, their families may be starving. Try to examine where things need to be changed and call meetings to get these things done. If you do have electricity, be prepared for it to fail at 3am, just when you are in the middle of a Caesarean section. Try not to blame cultural differences, and above all respect your patients' confidentiality.

Even when you have your 'normal' supplies, you will not have solutions for parenteral nutrition, or plasma, and probably no plasma expanders.

You may, however, have more than the teaching hospital: it too may be without water, electricity, spirit, or linen! You may be cherished, supported, praised, and congratulated by your Ministry of Health, or you may not. You may be in a health service which is steadily improving, or in one which seems to be getting steadily worse, if that were possible.

You may be in a culture which encourages you to be an entrepreneur, or you may be in a system ready to direct blame if you do something wrong, and ignore the truth if you do nothing! Expect that you may be cut off from the rest of the world for 4 months of the year. On top of everything else, HIV may now be endemic in your district. Finally, your greatest blow may be that your predecessor, who was promised that he would be posted to your hospital for only a short time, never ordered any stores.

But you have great blessings. In coping with all this, in creating and caring and leading and serving, you will have done something that your colleagues in the more comfortable circumstances of *private* practice will never have done. You are an all-rounder, and have one of the last remaining opportunities to practise the totality of medicine, rather than some infinitesimal corner of it. If you are one of few doctors, lack of continuity of patient care will not be an issue. *Sub specie aeternitatis* (in the mirror of eternity), you are a hero and will surely be recognized and remembered as such.

You will need:
(1) A willingness to learn from the culture of your patients, and learn their language. This will enrich you greatly, whether you are a national from the urban elite or a foreigner, and will greatly increase their trust in you.
(2) An almost pathological desire for hard work under conditions which are not conducive to it.
(3) An unfailing ability to improvise and make the best of things.
(4) The capacity to withstand prolonged periods of cultural and maybe financial isolation. If your morale is high, so soon will be that of your staff also. Your patients will be grateful for anything you can do for them, and it is likely they will not yet have learnt to litigate against you.

DIDIMALA (4 years) was severely burnt. You worked for hours to put up a reliable drip and took great care to ring up for a bed in the referral hospital. When you pass by the ward 2 hours later, you find that she has indeed been sent there by ambulance, but the drip is lying on the bed, and the vein is thrombosed. You ask, "Why is this?", to which you get the reply, "There was no hook in the ambulance."
MARIA (6 months) presented with intermittent vomiting and abdominal swelling and was diagnosed as having intussusception. Unfortunately, the first hospital she went to had run out of anaesthetic gases and so could not operate. Her mother had to take her through three states stopping at four hospitals before she found one which could anaesthetize her.
LESSONS (1) Anaesthesia is often the limiting factor in surgery. (2) There is no need to have to rely on a supply of nitrous oxide. (3) Some cases might not need anaesthesia if treated early (12.7).

If you serve your hospital and the community round it for >5yrs, you will earn a unique place in its affections. Just to prepare you, we describe the kind of situation you may have to cope with.

THE SCENE IN A TYPICAL POOR HOSPITAL

Fig. 1-2 THE SCENE IN A TYPICAL POOR HOSPITAL.
An improvised ward in a small hospital in Madhya Pradesh, India, in the 1960s. Most patients are accompanied by members of their families or by friends. If they are away from their villages during the planting and harvesting season, they will go hungry. *After Howard GR. Socio-economic factors affecting utilization of a rural Indian hospital. Tropical Doctor 1978;8(4):210-9 with kind permission.*

If you subsequently move to work in the hospitals of the affluent world, you may well miss the sense of purpose and achievement that you found when treating patients in low-resource settings. Your experience and your practical knowledge may not be highly esteemed, or at worst ignored. You will be shocked by the wastage of resources and the lack of a clinical acumen that you have tuned carefully over much time and painful experience but that seems to count for little in the corridors of modern high-technology hospitals.

However, no one will be able to take away the pride that you have done what so many of your colleagues wished they had done, and the gratitude of so many of your patients, who, without your help, would have suffered long or died.

1.3 Twenty surgeons in one & medical superintendent?

As a doctor in one of the hospitals we have just described, you are unlikely to find a fully qualified specialist surgeon with 6-8yrs of postgraduate training. But somehow you have to care for the sick in *all* of the 20 specialist fields shown in the frontispiece, into which surgery has fragmented in recent years. The chance of your being able to refer patients to specialists is remote. There may be no maxillofacial surgeon or hand surgeon in the country, and if it is a small one, there may not even be a specialist anaesthetist. Even your own teaching hospital may lack the complete range of specialists. Nor, despite present training programmes, is the situation in many countries likely to improve much in the near future. Even your nearest regional hospital may only have one or two general surgeons, or none at all! But surgery will be only part of your work; you may also have to be a physician, and a paediatrician, *and* manage the hospital as chief executive.

This will be especially true if you are an emergency surgeon flown in to help in a disaster situation, such as an earthquake; the first operation you are likely to have to do is a Caesarean section! *Try to set up a system of triage (even in non-emergency settings) so urgent patients don't wait.*

As a leader, or even district medical superintendent, you may have to deal with everything and everybody. When you arrive, make note of what you see (you easily forget your first impressions and fail to improve things which could have been altered). Beware of the subtle temptations of corruption: the bribes offered for preferential treatment, the back-handers for unnecessary or sub-standard equipment, the requests for unsecured financial advancements from hospital funds, the persuasive salesman for unrecognized drugs, the falsifying of records and so on…. *Don't get bogged down in an office and let clinical work take second place*: this should be your priority. Organize a regular timetable for yourself and stick to it.

The method of a good leader is to observe, listen, learn, discuss, decide, communicate, organize, encourage, facilitate and participate. It is necessary to have a critique of your activity: this is audit. Be sure to set goals, evaluate them, get feedback, coordinate efforts of others, recognize achievement and accept responsibility. Most problems will have as their root causes: poor leadership, poor relationships, poor pay, poor morale and working conditions, poor administration, and poor supervision. How you handle a crisis is the best test of your managerial skills; try to think beforehand what might go wrong, however, to avoid such a crisis in the first place.

Ordering supplies in advance and organizing repairs are most important. Keeping good records is essential, both of managerial decisions and patients. Don't forget aspects of hygiene, the use of toilets, disposal of garbage, the problems of overcrowding and relatives' accommodation within hospital premises, and the problem of excessive noise!

Inevitably you will have to hold meetings, usually as chairperson; set clear objectives and outcomes, set an agenda, keep a strict eye on time, and allow everyone to have their say, but keep folks to the point and avoid letting the subject drift. Afterwards make sure you get feedback.

You will inevitably have to write death certificates, and medical reports, and do much other paperwork. Get a secretary to help you, and limit this sort of activity to a particular short period in the day. Take care when disclosing medical information: it may be confidential.

Education is the key: daily morning reports, bedside teaching, grand rounds (especially for visitors), morbidity and mortality (M&M) meetings and rehearsing critical care practices should be the norm. Clinical audit is healthy: look at, for example, rates of wound infection, success of skin grafts, incidence of HIV+ve patients, mobility scores for femoral fractures, delays getting equipment repaired etc. Remember, though, that M&M should not be an occasion to apportion blame: it is a way to examine how you can avoid errors of omission or commission, or poor judgement or poor technique.

For you to keep up to date, *don't miss out on your own education*: try to encourage specialists to visit your hospital, subscribe to journals (especially *Tropical Doctor*), establish distance learning (by e-mail if possible), and promote a hospital library. However, beware of spending excessive time at workshops, which may leave your hospital stranded and be of little educational use to you. Try to visit your rural clinics and other hospitals in your district on a regular basis.

So you will have to do your best in *all* these fields simultaneously, as well as being 20 surgeons in one! To help you, we have collected from among the armamentarium of diverse experts:
(1) Some easier methods which you could use. Fortunately, many of them, despite the fact that they are normally only part of an expert's expertise, are not too difficult. For example, the position of safety in a hand injury is within the competence of any doctor or technician.
(2) Those methods, either easy or difficult, which you will have to use to save a patient's life.
(3) Those difficult, disability-preventing but non-urgent methods for which you should refer a patient, but may not be able to, such as sequestrectomy for osteomyelitis (7.6).
Many countries don't even have enough general duty doctors to do all the surgery that needs doing, let alone specialists.

Typically there is only one doctor for 50,000 people, and only 4% of a severely depleted gross national product is spent on health services. Many countries in the world have recognized that essential surgery could be done by specially trained medical assistants (clinical officers), and several have trained them to do this. Such surgical technicians are the backbone of surgical delivery in several countries.

How nice it was to see how well the Assistant Medical Officer (AMO) was managing his tasks; he seemed to be well in control. He had done several Caesarean sections, 2 laparotomies for intussusception, some hydrocoelectomies, and fracture reductions. He was treating 3 cases of fractured femur with skeletal traction in a very satisfactory way. His management of burns did not give cause for criticism. He had not had sufficient experience of hernia operations, so we operated on 5 collected cases together, after which he wishes to do them himself. To go to Kiomboi was an inspiration for our AMO training programme. *(Isaakson,G. Report of visit to Kilamanjaro Medical Centre)*

We quote this to emphasize that, not only must much surgery be done by non-specialists, but that it is often excellently done by surgical technicians. Perhaps there is no such teaching programme in your country, and yet you are hopelessly overworked. Try to train an auxiliary to do the simpler operations, such as hernias, Caesarean sections and exploratory laparotomies. Write out a simple-to-follow scheme so that they can follow a regular work-path. This will relieve your burden, and ensure the work carries on when you are not there!

Remember that there may be a large turnover of staff: don't resent this but be welcoming of new faces and new ideas!

Beware 'burnout', where you get so exhausted and irritable, you cannot function properly. Take a break, leave the place and go on a well-deserved holiday, so you can come back refreshed and revitalized (and bring some vital supplies back)!

1.4 Your surgical work

Of all your hospital admissions, 10-15% will probably be surgical, but because operating is time-consuming, and as some patients remain in bed for a long time, surgery may take 30% of your time, and fill half your beds. How much you will do will depend on how good you are. Patients will travel hundreds of kilometres to a doctor with a good surgical reputation. A bad one will soon do little surgery.

Look carefully at the ages and sexes of the patients in your wards. When modern medicine first reaches a community, the first patients to present are usually the men, followed by the women and children. Only when medicine is well established will you see a proportionate number of older women. You will see a few hypochondriacs, but some may just come to see you because of your novelty value, and there are likely to be comparatively few repeat visits to the outpatient department because travel is so difficult.

You will see many of the diseases that are common in the industrial world, but in different proportions, a major difference being that so many of them present late (1.6).

'Western diseases' such as aortic aneurysm, carcinoma of the colon, gallstones and varicose veins may not be very common at all in rural practice, but are diseases arriving in the cities. Urethral strictures, pelvic infections, fibroids and hernias are usually common, as are some diseases that are almost extinct in the industrial world: acute haematogenous osteomyelitis, for example.

You will probably see tuberculosis of the chest, lymph nodes, abdomen, and bones, many manifestations of HIV disease, amoebiasis and other 'tropical' illness. Sepsis is frequent.

But you may seldom see carcinoma of the bronchus, or the thromboembolic complications of surgery that are so common in the West; you may probably never see diverticulitis.

No branch of surgery will differ more starkly from that in the industrial world than orthopaedics, where contractures and deformities are commonplace.

You may be presented with many kinds of operation to do, but 50% of your workload is likely to be in obstetrics and gynaecology. The rest will be divided almost equally between sepsis and trauma, the nature of which will depend on where your hospital is situated.
N.B. Trauma is discussed in Volume Two of this manual.

Unfortunately many times you will *not* be able to refer a patient (1.6). *Never refer someone just to get him off your hands!* Always think what would, in the current circumstances (*not in the ideal world*), be the best for your patient.

This was a consecutive list of surgical cases seen over 3wks in a 50-bed mission hospital in Mandritsara, Madagascar:

(i) a 9yr-old girl with osteomyelitis of the tibia
(ii) 2 Caesarean sections for failure to progress
(iii) a 50yr-old lady with intestinal obstruction and carcinomatosis
(iv) a 24yr-old lady with an ectopic gestation
(v) an 18yr-old girl with gas gangrene of the uterus
(vi) a 48yr-old man with an unresectable cologastric mass
(vii) a 58yr-old man with a pertrochanteric femoral fracture
(viii) a 46yr-old woman with a large fibroid uterus
(ix) a 37yr-old man with Fournier's gangrene of the scrotum
(x) a 36yr-old man with necrotizing fasciitis of the whole right leg
(xi) a 16yr-old girl with septic arthritis of the left shoulder
(xii) a 15yr-old boy with a urethral fistula
(xiii) a 35yr-old G9 P5 woman with pelvic impaction of the fetal head
(xiv) a 45yr-old lady with pericardial tamponade
(xv) a 9yr-old girl with 5 distal ileal typhoid perforations
(xvi) an 8-month child with a huge 25cm-sized hydronephrosis
(xvii) a 93yr-old man with a right inguinoscrotal hernia
(xviii) a 31yr-old man with a plexiform shoulder neurofibroma
(xix) a 56yr-old lady with 3 days of adhesive small bowel obstruction
(xx) a 17yr-old girl with retained placenta for over 24h.

Fig. 1-3 TABLE OF SURGICAL ADMISSIONS IN A RURAL HOSPITAL.

Always think whether what you can do to a patient will probably benefit him; if you cannot refer him, or the distance is too great for him to reach the referral hospital alive, your choices are much clearer. Decide whether his problem is urgent (and therefore needs your intervention) or whether it can be alleviated by an operation within your scope (even if something else has to be done later), or whether it can wait for the specialist. Try to get a specialist to visit you to teach and advise: he may well enjoy a trip away from the daily grind!

KALPANA (46yrs) presented with mild abdominal pain for several days, severe for 4 days, and diarrhoea with two loose stools tinged with blood daily for a week. She had a tender, fluctuant mass in her right lower quadrant, and a marked leucocytosis. At laparotomy she had a patchy necrosis of her caecum with a localized perforation. A right hemicolectomy was done for suspected necrotizing amoebic colitis (14.5). The operation was a nightmare. Her colon came to pieces in the surgeon's hands and there was gross faecal contamination. She died. LESSONS (1) Expect a different spectrum of disease from what you might be used to where a fluctuant mass in the right lower quadrant may be most likely to be an appendix abscess. (2) Avoid doing a right hemicolectomy for amoebiasis if you can.

The late Imre J.P. Loefler, one of our editors, in a wide-ranging lecture on the failure of the medical profession to deliver surgical care in much of the developing world, stated: "You must be humble; surgery is a craft that makes use of the scientific method of Popperian falsification. The art of surgery consists of judgment and the beauty of an operation well done, done gently, with respect for living tissue, for every cell, with reverence for form and function, carried out with compassion, always remembering that the only justification for invading the body of another individual is the intent to restore homeostasis."
Loefler IJP. Surgery in the Post-Colonial World (Rahima Dawood Oration). E & Centr Afr J Surg 2002;7(1):53-8.

Remember, it is not only possible, but usually mandatory, to perform surgery without every modern convenience. Such surgery is by no means necessarily worse than that done in a high-technology centre with every available gadget.

BHEKUMUZI (10yrs) was lying in a district hospital with an obviously angulated fracture of the left forearm sustained when climbing a tree to fetch fruit. When a visiting doctor came to do a surgical round, he asked when he was admitted and was told, "Three days ago, just after it happened." The reason given as to why the fracture had not been reduced was that no radiograph could be taken because the X-ray machine was not working! LESSON It may seem obvious that you don't need a radiograph to tell you that an obviously angulated fracture needs reduction. Think whether you really need a laboratory to correct potassium loss in diarrhoea, or an abdominal radiograph for a gross sigmoid volvulus, or a CT scan for a head injury.

1.5 Your patients

In many of the villages of the developing world, the burdens of chronic disadvantage, poverty, ignorance, and insanitation are the background to life. A surgical disease on top of this may be the last straw.

As a result, patients often present late. If yours is a really disadvantaged community, tapping a hydrocoele may yield litres rather than mls of fluid. An elephantoid scrotum may have progressed so far that it hangs to the ground (27.34). If a patient has a urethral stricture, he may leave it until he has multiple fistulae or massive extravasation (27.11). If he has carcinoma of the penis (27.33), he may wait until much of it has been eaten away. Most carcinomas of the breast (24.4) and cervix (23.8) present too late for any hope of cure.

Too often, patients only present when complications have made their lives unbearable. When even the struggle to stay alive may be a losing battle, the fact that surgical disease is normally treatable is irrelevant.

There are usually good reasons why a patient presents late. The family may have had no money for the operation or for transport, or there may be no transport. Perhaps it is the planting season, or there is nobody to look after the children or the goats? Perhaps the disease is painless, and symptoms can be tolerated, so that illness remains unrecognized? Perhaps the tolerance to pain, disability, deformity, and misery is so high that help is only sought as a last resort? A patient may only come to you when he has exhausted local remedies and the services of traditional practitioners. He may not come to you because he doubts whether you can provide any assistance, or that he can afford it. Transport, which may have been difficult before the rainy season, can become an insurmountable problem when roads become quagmires, and rivers even more perilous. Acute surgical emergencies, in particular, may only come when patients are in the direst straits.

You are unlikely to be able to send patients for extensive series of investigations before you start treatment. In fact you should rely more and more on your clinical skills. Many patients will arrive with classic presentations or advanced disease, and the diagnosis may be obvious. However, we wish to present a guide on how to deal logically and effectively with patients *without* sophisticated technology. *Don't fail to treat a patient simply because you don't have the means you may be used to!*

Expect to find that the patient has other diseases also. Studies in Nepal, for example, showed only 15% of operations were done in otherwise healthy patients; in Zimbabwe over 30% of operations were done on patients with HIV. So expect your surgical patients to be poor, malnourished, immunosuppressed, anaemic, malarious, tuberculous, or worm-ridden, or all of these things. These illnesses make a patient weak, wasted and a poor operative risk. Anaemia increases the risks of surgery, and in some communities the *average* haemoglobin may be only 8g/dl. Some patients may still be walking around with 4g/dl or even lower. Apart from a little breathlessness on the hills of Nepal, one 12yr-old girl with an Hb of only 20g/l had no other complaints.

So try to prepare your patients for surgery before you operate, especially if the cause is readily treatable. *But beware the dangers of blood transfusion* (5.3).

Pain and disability are unlikely to rate highly when there is rice or maize to be planted, or when there are festivities and holidays. Although the local economy may be poor, certain obligations may be compelling.

Some cultural objections may exist, to orchidectomy, for example, and may be so firm that a patient is unlikely to agree. Mastectomy or colostomy may be similarly abhorrent.

A PRIVATE WARD

Fig. 1-4 A PRIVATE WARD in a rural hospital. For a village family an illness is more than a biological disorder: it may be a social and economic crisis. *After Howard GR. Socio-economic factors affecting utilization of a rural Indian hospital. Tropical Doctor 1978;8(4):210-9 with kind permission.*

Death is the great enemy of doctors and evidence of our failure. But a patient may have faced up to his own mortality long before you have, and may not always share your view. He may have learnt to live with death since childhood, and both his own attitude to it and that of his closest relatives may be very accepting. *Never lie to a patient when you know he is dying:* he probably knows it also, and realizes you know it too!

One of the greatest mistakes you can make is to offer a useless operation, which will use up much of his own resources and those of the hospital in an unsuccessful attempt to produce a cure. Theodor Billroth, a pioneer of surgery, famously commented, "To operate without having a chance of success is to prostitute the beautiful art and science of surgery." In some cultures it is important for a patient to be buried at home, so consider sending a terminally ill patient home early while he can still travel.

1.6 Referral is mostly a myth

A patient with a surgical disease has first to refer himself to you, and if you cannot care for him, you must consider referring him to someone else. Referral onwards from a community health worker (CHW) normally takes place at all the five steps (1-5). Although surgery is done in other parts of this system, we are concerned with the district (or mission) hospital, and the critical referral steps from C to D and from D to E.

Although 'referral systems' exist in all health services, the difficulties put in a patient's way are often insurmountable. Unfortunately, for many patients referral is a myth. In many resource-poor countries the possibilities for referral appear to have got worse during the last decades rather than better. Too often, there is just no petrol for the hospital's ambulance to take a patient to a referral hospital, or no money to buy it; furthermore the roads may be impassable; he may not have money to pay the referral hospital fees, or the bribes necessary to gain admission, and he may be very reluctant to travel so far away from home. Alas, in many countries the future does not seem any more hopeful.

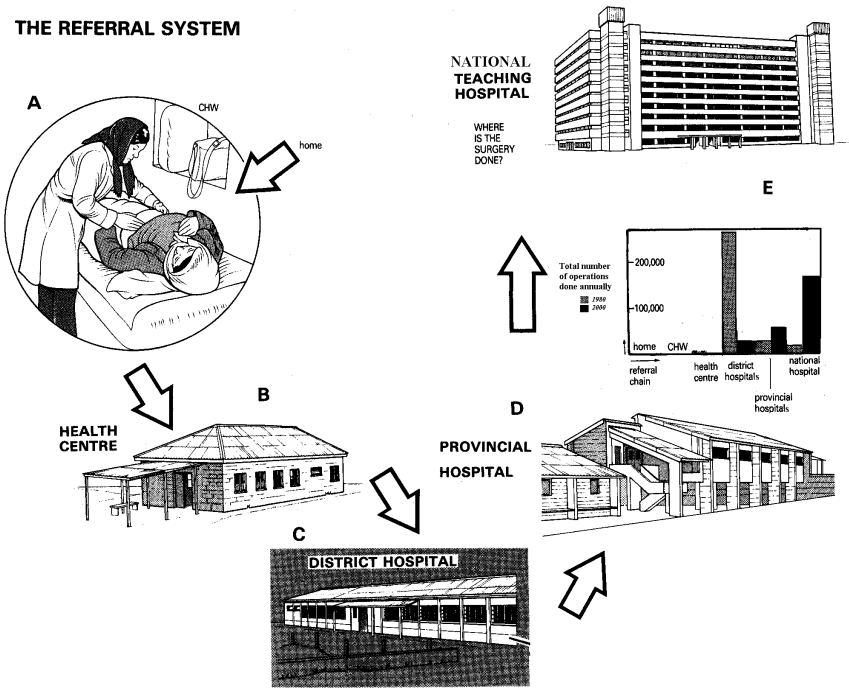


Fig. 1-5 THE REFERRAL SYSTEM.
Each of these steps in the referral chain has its difficulties. A, from the patient's home to the community worker. B, from the community worker to the health centre. C, from the health centre to the district hospital. D, from the district to the provincial hospital. E, from the provincial to the teaching hospital. The histograms show a typical change in the total annual number of operations done at each stage in the referral chain in 1980 and 2000. Little surgery is now done in many of the district hospitals, but *it is often not being done in the provincial or national hospitals either.*

Only too often a patient reaches a referral hospital with great difficulty, only to return no better than before he went. Because there are so many uncertainties, assess the chances for each patient individually. Try to find out what happens to each of the patients you send. Just what cases is it useful to refer, how, when, and to whom? If there are referral services, be sure to use them, both to refer patients properly and to learn from yourself.

In the following pages, we assume that you *usually can't* refer the patient. There may be procedures you don't feel confident to do; obviously if you do have the opportunity for referral, use such help. Consider carefully if the patient may end up worse off than if you had not intervened. However, don't back out of a life-saving procedure through false modesty. This volume exists to help you in just such a situation.

Don't overburden the referral hospital with minor cases; take the opportunity to spend time there to learn surgical procedures if you need more experience. The important factor is the degree of urgency that exists: balance this against the feasibility of referral.

N.B. Some surgeons working in referral hospitals have a false idea of the practicalities of referral. They see only the 'tip of the iceberg' (or the 'ears of the hippopotamus'), the patients who reach them successfully: they may think that referral is easier than it is.

There are however certain cases which referral hospitals should accept without question, and district hospitals should know what they are. One example is the management of intestinal fistulae (11.15).

Remember, referral hospitals also have their problems:
(1) They may be overcrowded with simple cases that you could care for in your district hospital.
(2) When the time comes to discharge a referred patient, it may not be done because the bills have not been paid.
(3) Their system of communicating information may be very longwinded, so you may not get proper feedback unless you yourself enquire: that is why direct contact is so useful!

One of the purposes of this manual is *to make sure that any surgery that can be done in a district hospital is done there*, so that referral hospitals can fulfil their proper function, and life-saving surgery is not deferred till after a long journey to the referral centre. Another purpose is to train cadres (not necessarily doctors) to acquire special skills to deliver surgical services relevant to situations. A third reason is that referrals are expensive (to both the patient and the health system).

REFERRAL: IS IT WORTHWHILE?

The chances of being able to refer a patient vary greatly, and are apt to change. They depend on the answers to these questions:
(1) Does he have a disease for which the referral hospital has no effective treatment (*e.g*. hepatoma or advanced HIV disease) or equipment (*e.g*. advanced osteoarthrosis of the hip needing hip replacement)?
(2) Does he have a condition (*e.g*. cleft lip and palate) best left till a later date?
(3) Does he have a condition which will kill him before he gets there (*e.g*. ruptured spleen) or be untreatable by the time he gets there (gastroschisis)?
(4) Will he be able to get there and look after himself when there? What about his family?
(5) Will he be sure of getting any better treatment than yours? Try to contact the surgeon for advice before sending the patient, especially if distances are long and the case is not an emergency.

PATSON (49yrs) was in a Land Rover when it rolled over in deep sand, causing an open fracture of the right humerus and injuring the radial nerve. He was still able to walk, so he eventually reached a district hospital, where the wound was carefully toileted, and left open for delayed primary suture. The radial nerve injury was recognized, the arm was put in a collar-and-cuff sling, the wrist in a cock-up splint, and he was asked to return in 48h. The wound being clean, it was closed. So far he had received ideal treatment. It was decided to refer him to the provincial hospital 40km away, across a river and a flood plain, 8mins by air, a day by boat, or 2 days by Land Rover. There was no radio, and the telephone was not working, so there was no way of telling the provincial surgeon that he was coming. He was able to get a seat on a barge and was in the provincial capital 24h later. It was dark but he was able to find a relative who had a room for the night. The next day he sat in the outpatient queue and handed the slip to the medical assistant.
Unfortunately, the provincial surgeon had left the previous day to attend a planning meeting at the Ministry of Health. He would not be back for 2 days. The provincial surgeon returned and saw him, but decided that the training had not prepared him for posterior exploration of the humerus, plating the fracture and perhaps secondary suture of the radial nerve. Also, he had no 6/0 monofilament. So Patson was given a bus warrant, and a note to the orthopaedic surgeon in the teaching hospital in the capital city.
Unfortunately, he had no money, no food, and no clean clothes for the journey, so he went home. The Land Rover had been partly dismantled by thieves, but his partner had towed the wreck back to the village, and hired a lad to help him with the fishing. The family were already deeply in debt. They debated whether he should go 800km to the capital, but the limp wrist decided it for them. He started on the long journey with a pack of food, a few clean clothes, and a bus warrant, but very little money.
Four days later he arrived at the orthopaedic clinic on a Friday. He had no appointment, and the surgeon to whom the note was addressed had held the clinic on the previous day. The harassed sister, busy with another clinic, found that he had no relatives in the city, and no money, so she sent him to the orthopaedic ward in the hope that they might have a bed for him over the weekend. They did.
On Monday the surgeon saw him. The wound had healed and he was fit for surgery, and the necessary screws, plates, adhesive drapes, and sutures were in stock. But there was a three-month waiting list, so he had to wait 8 days, even for operation as a semi-emergency. A silent cheer went up from the hospital staphylococci, as they began to colonize the skin of this provincial patient.
His radial nerve was freed from compression in its spiral groove, and the fractured humerus was successfully plated. Two weeks later he returned to the provincial hospital with suggestions for physiotherapy (a 2-day journey for each session) and instructions to return in 1yr for removal of the plate.
He was lucky. He was one of the minority for whom the referral system 'worked'. The radial nerve palsy recovered. A friend paid for nearly 4wks in a teaching hospital, and 1600km in transport. He was in debt, and the family were hungry, but he did not have to sell the boat, or the remains of the Land Rover. It could have been much worse.

TOPNO (41yrs) fractured his ankle in a bus accident. The very competent doctor who saw him had learnt that difficult ankle fractures should be referred. He could manipulate fractures, but he thought that an expert would do better, so he sent the patient with a letter to the referral hospital 70km away. After a long journey, the patient arrived too late at the fracture clinic. He was able to reach the next fracture clinic in time, only to find that the surgeon was away at a conference. So he hung around hopefully for some days, but in the end he was advised to return to the original hospital. Meanwhile, he had had no treatment except the original 'first aid' plaster. When he eventually returned to the doctor who first saw him, the fracture had partly united in a very bad position. It was now too late to manipulate him, so he now has a stiff painful ankle and is waiting to have it fused.
LESSON A patient may be better in your hands, if you learn those procedures that you can reasonably do, in your own set-up.
*Jellis JE. Chairman's Address, Proc Assoc Surg E Africa 1981;**4** 53-6.*

ASSESS EACH PATIENT'S CHANCES OF EFFECTIVE REFERRAL

Make sure you know the specialist's timetable, and his contact details including mobile phone numbers. You can often use the messaging system on mobile phones better than the voice; with newer mobile phones, you can send pictures of a radiograph, histology slide, or even a patient's lesion. You can create a clinical WhatsApp group. *Beware, though, of possible leaks of confidentiality.*

Can the patient get himself to the referral centre? In some districts, for example, the roads and airstrips are closed for weeks at a time during the rainy season. Is he prepared to leave the family and the fields or the job? Has he or the hospital got money for transport and for lodging when he gets there? Often, neither of them does. If he does arrive, will he arrive on the right day, find the way to the right clinic, wait in the right queue and be seen and admitted? Will there be an empty bed? Will the surgeon you send him to actually be there when he arrives, or will he have gone on holiday, or to a conference in America?

Investigate him first if you can, and state the procedure that you think he needs. If a biopsy is necessary, do it, and refer him with the report. Often this takes time to obtain or may have to be sent to the referral hospital anyway. You might then send the patient with the biopsy specimen already taken. If referral is urgent, *don't wait for the report*, but give sufficient details so the report can be traced.

Inform the surgeon that the patient is coming. Make sure that the patient knows exactly what to do, and where to go when he arrives. Send a careful letter with him, including all necessary information. If there are any particularly good referral facilities, such as those for artificial limbs, for example, be sure to use them.

Finally, *don't refer patients unnecessarily*. No surgeon likes to be sent plantar warts.

1.7 The limits of this system of surgery

Detail, especially in surgery, is important, but you can get bogged down in details. The quotation "*Le bon Dieu est dans le detail*" (God is in the details), attributed to the French writer Gustave Flaubert (1820-1880), must be balanced by the German proverb "*Der Teufel steckt im Detail*" (The devil hides in the details)!

In view of the common impossibility of referral, we have tried to describe in this book everything that you, our readers *as a whole*, might have to do: both the emergency procedures and the less urgent elective cases.

As you will see in the next section, you *individually* should not necessarily do everything we describe. We take for granted that personal tuition from an expert is the best way to learn anything. But, what if there is no expert? *A manual is surely better than nothing.*

Somehow, we have had to find a balance, so we have considered each procedure on its merits. Our task has been made no easier by the wide range of the abilities of our readers. You may range from being a highly trained surgeon, doing unfamiliar operations for the first time, to an inexperienced technician doing your first job. We have tried to serve all your needs.

Although learning something from a book is not for many as good as learning it first-hand from a good teacher, the very *raison d'être* of this text is to provide relevant information in a situation where you have no such teacher. These books, though, are of no use if they are kept unused on the bookshelf!

As books are expensive, we have made this text available *gratis* electronically on the internet, which we hope will further disseminate the accumulated wisdom gathered herein.

It has not always been easy to distinguish the tasks which are obviously impossible for you (oesophageal atresia for example) from those which may be possible (duodenal or jejunoileal atresia). We have had to balance benefit, risk and urgency. This has led us to include methods for removing the prostate, for example, but not a meningioma.

We have tried to grade the difficulty of the operations described (Appendix A). If you can refer the more difficult cases and the patient is likely to obtain a better result if you do so, this is obviously preferable. We have stressed, though, that some operations are only for the *careful, caring operator*. These include Girdlestone's arthroplasty (7-21), closure of a Hartmann's procedure (12-15), Roux-en-Y anastomosis (15-14), and closure of a meningomyelocoele (33-15).

Although the common conditions may comprise perhaps 60% of your work, the rest will include many rarer ones. *In aggregate, the rarities are common.* So we have tried to describe as many of the comparative rarities as we can, in the hope that you will find about 98% of the conditions you could hope to treat surgically described here. The edges of this large collection of appropriate methods are inevitably blurred, and it has not been easy to know which rare, or which difficult, procedures we should include. For example, you will find much on HIV-related pathology (5.6), and there is even mention of cystic hygroma (33.12).

We shall probably be criticized for including oesophagoscopy (30.2) and bronchoscopy (29.14), and some cancer chemotherapy. But it is better to include slightly too much rather than slightly too little – there is no obligation for you to do things you don't feel able to do, but a crisis may force your hand! Thyroidectomy (25.7) is our *tour de force*, and the great detail in which we have described it should enable our more experienced and caring readers to do it. Some methods, such as methods of haemostasis, are classical, in that no textbook of surgery would be complete without them. Inevitably, some parts of the 'system' are tidier than others.

We have excluded all procedures which appear too sophisticated, but the range of facilities available is always very wide. In some cases we may have made false assumptions. We have often assumed that you have an X-ray facility, and ultrasound for example.

Uncertain sterilizing procedures and limited nursing care have also guided our selection.

Although we write mostly for hospitals which are short of both money and skill, there are some, such as those run by mines and plantations, or supported generously by outside agencies, where money is less scarce. These might be able to procure even comparatively expensive drugs for cancer chemotherapy, for example. For them all the equipment we list (even bronchoscopes and oesophagoscopes) should not be a problem. *However, beware the notion that expensive methods are necessarily the best!*

Overall:
(1) We have tried to describe a system of practice which includes all the basics, but is *ahead of the practice of many district hospitals*, so that even comparatively advanced ones have something to aim for.
(2) We have tried to cover most of the range of the 'general surgeon' working in the districts.
(3) We have tried to describe this system in complete detail, and in doing so would agree with both the quotations with which this section started.
(4) We have in our mind's eye a concept of 'quality' at the district hospital level; even simple things can and should be done well.
(5) We have tried to give guidance when things go wrong.

This last is most important. Many texts tell you what you should do (in the author's view); few explain what to do with complications. If you can correct these, you will often avoid a catastrophe and gain much satisfaction. You will also build a base of great wisdom for the future.

1.8 Should you operate?

Although the era of 'furor operandi' *has passed, one still has almost daily evidence of the disastrous effects of major surgical procedures, attempted lightly by young, or even inexperienced older, surgeons. The author would in no way dampen the ardour of the neophyte, or check the ambition to acquire skill. Still, it is well to suppress the feelings of cocksureness and egotistic pride.* (Thorek M, Surgical Errors and Safeguards, JB Lipincott, 1932)

Whether or not you should operate on a given patient will be the most important question you will have to answer. Put yourself in the patient's place. What would *you* like to happen if you were the patient? Several factors will influence your decision. We have already discussed one of them: can you refer him?

Would his operation be better done elsewhere? On the whole we think that for every doctor who operates when he should not, there are many more who don't operate when they should. So one of our aims has been to get more surgery done, on the *correct* indications! The mature surgeon is one who knows when *not* to operate! On the other hand, if you are always too cautious, you will never learn and some of your patients will never benefit. Remember to keep records (2.12).

So beware of what Max Thorek describes as *furor operandi*, the furious urge to operate, and ask yourself:

What will happen if you don't operate? If a patient is likely to die or become disabled if he is not operated on quickly, you will have to operate. We have therefore included all the more practical emergency operations, whether difficult or not. For example, you must drill immediately for acute osteomyelitis, but a patient who needs a sequestrectomy for chronic osteomyelitis can wait.

How difficult is the operation?
At least three factors determine this:
(1) your technical knowledge,
(2) your experience,
(3) your skill.

We can provide you with the knowledge, and bring you some of the experience of other people, but only practice will improve your manual skill. A score is given for your guidance (Appendix A). Grades 1.1-1.5 describe simple procedures that you will definitely have to master. Grades 2.1-2.5 describe straightforward operations without serious difficulties or complications that would not pose much of a problem for basic surgical trainees. Grade 3 represents more difficult operations, with increasing complexity up to 3.5.

Those procedures of even greater difficulty may be mentioned in passing but not described, as they are thought to be unsuitable for the situations pertaining where this book will be useful.

How good is your post-operative care?
It may be a good idea to have a special ward for the serious post-operative cases: the advantages are concentrating staff where they are needed, giving them experience and training, and making it easier for you to visit and monitor these patients. If you can separate a section or unit for intensive care (ICU) for the really serious cases (11.9), so much the better.

How safe is the operation? What disasters may happen?
Little can go wrong with draining most abscesses, or manipulating most fractures, but disaster lurks if you decide to close an intestinal fistula, dilate a difficult urethral stricture or do a block dissection of the groin.

Do you have the instruments, materials and staff needed?
Even if you don't, you may be able to improvise. Check that the electricity is working, the blood can be cross-matched, the necessary staff are present. *Don't be over-ambitious initially with staff whose expertise you don't know*; assess the capability of the hospital to handle certain procedures. Try to build on your experience, and teach the staff (and yourself) accordingly. Check the instruments and equipment before you start. Discuss the case with your anaesthetist colleague (if any). Is he experienced enough to administer the GA you require? Is there an alternative?

Are you yourself inclined to operate too readily, or not readily enough?
Cultural attitudes to operating vary. In Indonesia, for example, the common failing is to be too timid, and not to operate when necessary. The reverse is true in some parts of Africa, where inexperienced operators are much too bold. So be aware of your own personal and cultural bias and try to correct for it. *Don't operate out of bravado!*

Is the reason for operation unclear?
If the indication is vague, wait! *Don't be dragooned into operating by enthusiastic nursing staff or insistent relatives*. Treat the lowliest patient the same as an important politician.

What is the known or probable HIV status of the patient?
Take a social and sexual history. Look for tell-tale signs of immune deficiency (5.6).
N.B. You should try to move toward routine HIV testing especially if antiretroviral treatment is available.

What is the general condition of the patient like?
(1) Check the Hb level (and sickle test if this is common in your area), and the level of malnutrition and dehydration.
(2) Assess the respiratory reserve (11.13).
(3) Measure the Peak Expiratory Flow if you can.
(4) Can you improve pre-op hydration or nutrition?

Assess the risks of complications. Remember you will cut, saw, burn, bruise, traumatize and violate your patient, exposing his tissues to the cold and hostile external environment, spilling his blood and body fluids, but the patient's own healing mechanisms need to repair the damage. You can only assist this process.

Decision. If you have difficulty knowing what to do and can contact anyone who might know, *don't hesitate to do so*. Try to invite a surgeon to your hospital for a period to give you instruction first-hand.

Have this book available in theatre.
WRITE THESE RULES UP IN YOUR THEATRE:

RULES ABOUT DECIDING WHEN TO OPERATE:
(1) You must be certain of the indication to operate, even if it is only exploratory.
(2) When life is in danger, take risks and act fast.
(3) If a case is hopeless, *be prepared to say "No!"*
(4) *Don't do difficult elective surgery*, especially if the expected outcome is likely to be of limited value to the patient.
(5) Take trouble to make sure the time is correct to operate, and all the preparations for surgery are in place.

RULES BEFORE OPERATING: Inform the theatre of your operation list well in advance, if possible. Book your children, clean cases and diabetics first.
(1) Go over the history, examination and investigations yourself to confirm it is the right patient: ask him his name yourself! Confirm the correct diagnosis, and that the need for surgery still exists.
(2) If there is a lump, make sure you can feel it. Mark it. Make sure the bladder is empty.
(3) Ask the patient what operation he expects to be done and explain the nature of this operation, its purpose and consequence to the patient: this is *informed consent*. You need not scare him or confuse him with medical jargon, but *don't keep him ignorant* and make sure he and the relatives understand. Use diagrams, stories or even cartoons. Mention alternative treatments, and possible complications.
(4) Mark the side to be operated upon with indelible ink.
(5) Make sure the patient bathes the night before surgery, and that especially the operative area has been cleaned. Trim his nails, clean the umbilicus, scrub the feet, remove studs and jewelry. (There is no need to remove all nail varnish or bangles and threads of religious or cultural significance, but take down an elaborate hairstyle which may prevent extension of the neck.) *Never use blunt razors to shave the skin:* do minimal shaving. Remove any loose or false teeth.
(6) Check for any allergies.
(7) Check that the patient is starved for 4h (less for babies), but warm, well-hydrated and fit for a GA, fluid-loaded for a spinal anaesthetic, and that diabetes, hypertension, asthma, epilepsy, and coagulation are controlled. *Don't starve patients for long periods waiting for theatre!* Make carbohydrate drinks available up to 2h pre-op to avoid hypoglycaemia. Remember deep vein thrombosis and antibiotic prophylaxis if indicated.
(8) Check that blood is cross-matched if required, and blood results available.
(9) Make sure especially that suction, laryngoscopes, airways, ambu-bags, masks, endotracheal and nasogastric tubes, stethoscope and diathermy are available. Make sure the patient comes to theatre with the notes, investigation results and radiographs, and properly signed consent for the proper procedure (with the correct side, if any, noted).
(10) Familiarize yourself with the operation to be performed if you are uncertain of any details.

SPECIAL CONSIDERATIONS
Some patients take routine medicines: *don't stop these just because they are starved before operation!* This applies especially to anti-hypertensives, bronchodilators, steroids, anticonvulsants, anti-Parkinsonian drugs, cardiac medication, anti-thyroid drugs and thyroxine.

Steroid-taking patients should get extra amounts: add 100mg hydrocortisone at the start of a major operation and then reduce slowly: 100mg tds on day 1, 50mg tds on day 2, 25mg tds on day 3.
(N.B. 100mg Hydrocortisone = 20mg Prednisolone = 4mg Dexamethasone = 3mg Betamethasone = 16mg Methylprednisolone = 5mg Triamcinolone)

Oral contraceptives: stop these *1 month before* a major operation, especially involving the pelvis, where she is not ambulant immediately post-operatively. Advise about alternative barrier methods or you may be blamed for an unwanted pregnancy!

Anticoagulants: stop these 3 days before a major operation; an INR <2 is ideal if you can measure it. *Avoid spinal anaesthesia and the use of tourniquets.*

Antidepressants can give problems (*e.g.* tricyclics) with anaesthesia: stop these 2wks before a major operation.

Alcohol: many people drink large quantities of alcohol. This may affect the liver, and cause slow metabolism of anaesthetic agents, bleeding disorders, and produce post-operative withdrawal symptoms.

Diabetics need careful handling. Check glucose levels regularly. Make sure dehydration is corrected.
If control is not good, start a sliding-scale regime of soluble insulin 6-hrly:

Glucose Level	Soluble Insulin Needed
0-4 mM	0 IU
4-8 mM	0 IU
8-12 mM	4 IU
12-16 mM	8 IU
16-20 mM	12 IU
>20 mM	16 IU

N.B. It's best to err on the side of mild hyperglycaemia!

If control is by oral hypoglycaemics, omit them on the day of operation; if the operation is small, they can simply be restarted the next day. If the operation is major, convert to a sliding scale.

If control is by insulin, reduce the dose in the evening pre-operatively (if any) by 20%. Administer no insulin on the day of surgery and set up a 5% dextrose IV infusion; make sure the operation is done early in the day.

If it is a minor operation and the patient is eating normally afterwards, restart the insulin at the normal time. If he is not, start a sliding scale: you may need to adjust the sliding-scale insulin doses if these were previously high so that the total given per day for a level 4-8mM equals the normal total pre-operative dose, *viz.*:

For a patient on 30 IU am and 18 IU pm (total 48 IU), start with 48 divided by 4 (number of times glucose is checked/day) = 12

Glucose Level	*Soluble Insulin Needed*
0-4 mM	*0 IU*
4-8 mM	*12 IU*
8-12 mM	*16 IU*
12-16 mM	*20 IU*
16-20 mM	*24 IU*
>20 mM	*28 IU*

If it is a major prolonged operation, use 16 IU soluble insulin with 20mmol KCl IV in 1 litre of 5% dextrose at 100ml/hr during the operation provided the blood glucose level is >4mM and check it 2hrly. If it is >16mM, add another 16 IU soluble insulin to your infusion. After the operation, continue with a sliding scale.

If ketoacidosis is present in an emergency, administer 10 IU soluble insulin IV *and* 10 IU IM immediately, and then 6 IU IM hourly; infuse 5l normal saline, the 1st in 30min, the 2nd with 20mmol KCl in 1hr, and the 3rd to 5th with 20mmol KCl in 2h each. Then when the glucose level is <15mM, start a sliding-scale regime, and alternate normal saline with 5% dextrose.

You may need to sedate an alcoholic with large doses of diazepam, chlorpromazine or chlomethiazole, especially post-operatively.

If you have not done any surgery before, or only very little, start with the easier operations (Grade 1). You should at least be able to open abscesses (6.2). However, in an emergency, consider what you can do, and *don't be frightened to do it:* you may well save lives!
N.B. Limited surgery, leaving advanced procedures to an expert, is now accepted practice in damage control (11.3). In an emergency, do all you can to save lives: *you are not expected to make a perfect repair of everything!*

Note that in many cultures, operative consent involves the whole family, and not just the individual patient!

Johann Wolfgang von Goethe (1749-1832) in his *Maxims and Reflections* wrote: "The most fruitful lesson is the conquest of one's own error. Whoever refuses to admit error may be a great scholar but he is not a great learner. Whoever is ashamed of error will struggle against recognizing and admitting it, which means that he struggles against his greatest inward gain."

Winston Churchill (1874-1965) said, "Success is not final, failure is not fatal: it is the courage to continue that counts."

(a) Rules about operating
(1) You must be familiar with the anatomy; if necessary consult an anatomy book during the operation. *Don't be embarrassed to do so!*
(2) You must have someone familiar with anaesthesia giving the anaesthetic. If this is yourself, there must be someone else who can monitor its progress and record the patient's vital signs. You should also have someone available who can assist during complications, and have airway accessories to hand. Try by all means to get a pulse oximeter to monitor the patient.
(3) There must be a reliable system of sterilization, preferably an autoclave.
(4) You must have a good light, preferably adjustable. A headlamp is useful.
(5) You must have the necessary equipment and supplies for resuscitation (infusions, giving sets and cannulae, a laryngoscope, tracheal tubes, adrenaline, atropine etc) and haemostasis (swabs, suction, ligatures, clips).
(6) Have the highest regard for living tissue and be gentle and circumspect. Operate at your own speed. Use the technique you know best, *not one for which you don't actually have the experience.*
(7) Remember to give pre-operative antibiotics *before you start operating*, if indicated.
(8) Finally, *don't be too elated over your successes, or too despondent over your failures*. If you do fail, forgive yourself, *don't give up!* A bad spell during which 2 or 3 patients get complications may be followed by another in which none of them do.

CAUTION! Remember also that with elective operations, disasters are more difficult to justify than with emergency procedures, both to the hospital staff and to the general public, and that accusations that the doctor is experimenting on patients can do much harm.

(b) WHO safety checklist
Apart from having the above rules in your theatre, you should use the checklist recommended by the World Health Organization (WHO). You may have to adapt this according to your local conditions. One single person should be responsible for checking *verbally with the theatre team* each box on the list. The checklist is not something to be done by one individual alone, but openly with everyone involved present, much like checking procedures before take-off of an aeroplane.

There are 3 phases:
(1) **Sign In** before anaesthesia,
(2) **Time Out** before skin incision, and
(3) **Sign Out** before the patient and surgeon leave the theatre.
(If a box cannot be ticked, leave it blank.)

The anaesthetic safety check includes examination of airway equipment, breathing system (oxygen and gases available), suction, drugs and devices, and emergency medications and equipment, particularly for a difficult airway or aspiration risk, as well as the patient's fitness. Significant blood loss is >500ml in an adult or >7ml/kg in a child. *Don't be blasé about how little blood you are likely to lose!* Check if you need blood *at the start* of an operation!

The Time Out allows the team a moment to double-check the patient's identity and operation. You can then mention critical steps that you, as the surgeon, may encounter and so warn the rest of the team. The anaesthetist and nurse can do likewise.

The checker should complete Sign Out *before* you leave the theatre.

<table>
<tr><th>SIGN IN</th><th>TIME OUT</th><th>SIGN OUT</th></tr>
<tr><td></td><td>Confirm all OT staff introduced</td><td></td></tr>
<tr><td>Patient has confirmed:</td><td>Surgeon, Anaesthetist and Nurse confirm:</td><td>Verbally confirm:</td></tr>
<tr><td>IDENTITY</td><td>PATIENT NAME</td><td>PROCEDURE PERFORMED</td></tr>
<tr><td>SITE</td><td>SITE</td><td>COUNTS CORRECT</td></tr>
<tr><td>PROCEDURE</td><td>PROCEDURE</td><td>SPECIMEN LABELLED</td></tr>
<tr><td>CONSENT</td><td></td><td></td></tr>
<tr><td>Site marked</td><td>Surgeon review of critical events</td><td>Review of equipment failures</td></tr>
<tr><td>Anaesthesia safety check done</td><td>Anaesthetic review of concerns</td><td rowspan="3">Recovery concerns review: Nurse, Surgeon Anaesthetist</td></tr>
<tr><td>Pulse oximeter OK and on</td><td>Nurses' review of equipment etc.</td></tr>
<tr><td>Allergy: YES/NO</td><td></td></tr>
<tr><td>Difficult airway or aspiration risk: YES/NO</td><td>Antibiotic prophylaxis: YES/NO</td><td></td></tr>
<tr><td>Severe blood loss risk (blood available?) YES/NO</td><td>Radiographs present: YES/NO</td><td></td></tr>
</table>

Make sure you do this for emergency operations as well as elective procedures!

(c) Checks at the end of an operation
You should be satisfied at the end of a surgical procedure that you have done everything that needs to be done. *Don't do things that don't need to be done:* often complications from those 'extra' jobs done will come back to haunt you!
Make sure you have:
(1) Secured haemostasis
(2) Washed the operative wound or cavity
(3) Checked any anastomosis
(4) In the abdomen,
 (a) made sure no hernia orifices remain open,
 (b) placed the small bowel carefully,
 (c) secured omentum between bowel and skin,
 (d) checked a nasogastric or jejunostomy tube is properly in place (if required)
(5) Secured a drain (if needed)
(6) Made sure the swab, needle and instrument counts are correct.

None of these checks will guarantee that you avoid mistakes, but they go a long way to minimizing them. Try to establish a 'no blame' culture amongst your staff, so that when something does go wrong, you can find out what happened, and take corrective measures.

(d) Rules after operating
WRITE THESE RULES UP IN YOUR THEATRE!
(1) Ask your staff if you've forgotten anything (see above).
(2) Make sure the patient is nursed semi-recumbent in the recovery position.
(3) Check the airway. Suction any secretions.
(4) Make sure there is a post-operative regime of monitoring vital signs, fluid balance, and drugs given.
(5) Write neat, concise operative notes, preferably with diagrams:
 Name of operation
 Persons present
 Incision
 Findings
 Procedure
 Closure; drains inserted
 Time taken
 Estimated blood loss
 Specimens properly labelled and removed
 Post-operative orders
(6) Make sure nurses looking after the patient understand your instructions, especially with regard to IV fluids, drains, and pain relief.
(7) Indicate how to deal with possible problems and complications.
(8) Visit your patient at the end of your operating list, or some time after an emergency case.
(9) Encourage breathing exercises and early mobilization*: this will often go against local culture.*
(10) Provide good nutrition, skin and oral care.
(11) Explain the nature of the operation to the patient.
(12) Organize appropriate follow-up.

IT IS NOT THE AIM OF SCIENCE TO OPEN A DOOR FOR INFINITE WISDOM, BUT TO SET A LIMIT TO INFINITE ERROR

Bertolt Brecht, *in The Life of Galileo, 1939, scene 9, l.74*

1.9 'Oh, never, never let us doubt what nobody is sure about'

Inevitably, such manuals contain a huge quantity of didactic detail with few reasons as to 'why' you should do anything, and few references to the original papers. We have tried to select the best methods for your needs. Even so, remember that accepted methods change, that few have been rigorously evaluated by controlled trials, and that some which were widely accepted only a few years ago have now been completely abandoned or reversed.

Here are some examples of how fallible medical practice can be:
(1) Tension sutures used to be used to close a difficult abdomen, but are now thought to make things worse.
(2) Complete immobilization was and often still is considered to be the ideal treatment for all long bone fractures. It is now increasingly realized that many of them benefit from early controlled movement.
(3) It used to be standard practice to separate mothers from their babies immediately after birth. Now, this is completely reversed and their close contact immediately after delivery is considered essential for bonding.
(4) Shaving a patient the day before an operation, which used to be standard practice, has now been shown to increase the incidence of infection.

This list could be expanded. So be prepared to 'doubt what nobody is sure about', even while you follow the didactic instructions we give. There is little justification for much of what is traditional practice in surgery. There is no justification for the 'arrogance, arbitrariness, stagnation, imitation, hypocrisy of political correctness, loss of sense of reality and resulting pretentiousness one finds among professionals in the universities, medical schools and departments of surgery.'
Loefler IJP, Surgery in the Post-Colonial World (Rahima Dawood Oration). E & Centr Afr J Surg 2002;7(1):53-8.

Remember 2 other Winston Churchill aphorisms:
"It is no use saying, 'We are doing our best.' You have got to succeed in doing what is necessary."
"Criticism may not be agreeable, but it is necessary. It fulfils the same function as pain in the human body. It calls attention to an unhealthy state of things."

1.10 Creating the surgical machine

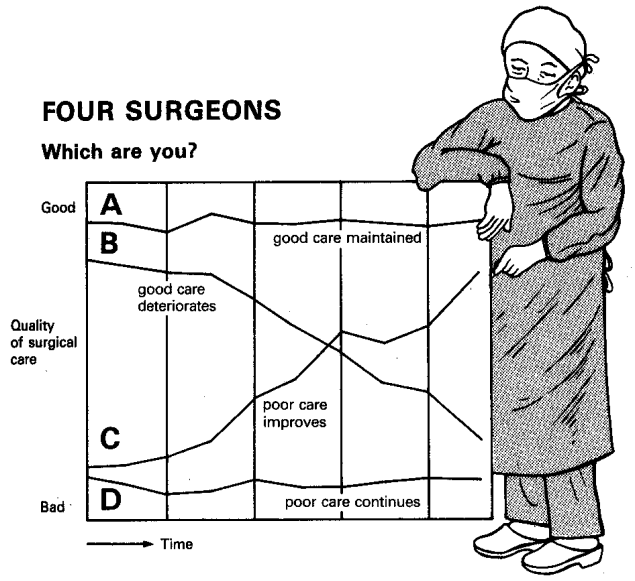


Fig. 1-6 WHICH OF THESE SURGEONS ARE YOU?
Doctor A found a nearly perfect surgical system and stepped in and out of it without needing to change it. Doctor B found a moderately functioning system and slowly let it deteriorate. Doctor C found a poorly functioning system and with great effort was able to improve it considerably. Doctor D found and left chaos.

If you are lucky, you will arrive at a hospital where your colleagues and your predecessors have created a smoothly running surgical system. Or, you may arrive and find almost nothing. More likely, you will arrive and find a system which is working somehow but which badly needs improvement.

The presence of pressure sores on the wards will tell you a lot, as will the frequency of wound complications after elective clean procedures. As well as actually treating the sick, you may have to try to make the hospital as a whole, and particularly its surgical services, more efficient.

To do this you will have to improve:
(1) The morale and training of the staff: congratulations are likely to be much more effective than reprimands. Explanation of the purpose and value of observations, history taking and examination is likely to be more effective than forceful teaching by rote.
(2) The fittings and equipment.
(3) The administrative arrangements.
(4) Your own skills. In doing this you must be prepared to do *any* task yourself, no matter how humble and how unfamiliar. There is no place for the attitude, "Oh, but it's not my job." Our jobs, wherever we are, are to create the 'machine' and make it work (1-6).

ALL FOR A PIECE OF CHALK. There was once a professor of surgery who found to his astonishment that the operating list had been cancelled. When he asked why, his junior assistant replied, "Because there is no chalk with which to list the cases." The professor was furious and dismissed the houseman on the spot. The District Medical Superintendent pleaded with him, "... such a nice boy..."; even the Minister pleaded, but the professor insisted that he could not have such a person as his junior. So he continued to clerk his own cases. Finally, weeks later the repentant houseman came to him and said, "About that chalk, Sir, I think I made a mistake..."
LESSON *Failure to improvise, where this is at all possible, is never an adequate reason for not doing something.*

When you arrive inexperienced in a new place, study it carefully and list the things that need changing. Then, cautiously and steadily, try to implement them during the next few months or years. If you don't note them when you first arrive, you will soon take them all for granted and do nothing. Beware of constant change, because the staff will not accept it. Get to know them and accept their advice before introducing 'improvements'. Identify keen and active members of staff, and communicate through them.

Above all, when you operate, start with familiar cases at first, and look out early for complications. *Don't blame others for your mistakes!*

Then, after 2-3 months, when you have the feel of the place and its problems, visit the nearest hospital where they do things well, stay a week or two and learn whatever they can teach you in a short time. Then come back and put what you have learnt into practice.

Remember the golden rules:
1. Use an aseptic technique.
2. Get adequate exposure.
3. Cut under tension and counter-tension.
4. Ensure adequate haemostasis.
5. Handle tissues gently.
6. Remove devitalized tissue and foreign bodies.
7. Obliterate any dead space.
8. Make sure the tissue blood supply is good.
9. Avoid excess tension on any suture line.
10. Check the swab and instrument count.

Many problems arise when patients are sedated but not properly observed: this is one of the most important things you can teach nurses in post-operative care.
Use the Ramsay scoring system:

1	Anxious, agitated, restless
2	Cooperative, oriented, and tranquil
3	Sedated but responds to commands
4	Asleep; brisk response to glabellar tap or loud auditory stimulus
5	Asleep; sluggish response to light glabellar tap or loud auditory stimulus
6	Asleep; no response to deep painful stimulus

Fig. 1-7 DOCTOR 'C' TEACHING THE TEAM.
When Doctor 'C' (1-6) **arrived, he found the obstetric wards in a deplorable state, and its beds so overcrowded that rupture of the uterus occurred in the corridors almost unnoticed. He soon got to work, and here you see him explaining how to put on gloves. Soon, the obstetric services were so efficient that he had empty beds.**
Kindly contributed by Holly Quinton.

It may all be summarized in the words of Denis Burkitt, the famous African epidemiologist, when asked for an autograph on his book: *"Attitudes are more important than ability, motives than methods, character than cleverness and perseverance than power, but above all, the heart takes precedence over the head."*

1.11 The surgical care of the poor

The purpose of surgery is to heal the sick. What is the use of surgery if the sick cannot afford it? The rapid growth of the populations of many countries requires that we care for ever more people every year, on a health budget which is not only low to begin with, but is *static*, or in some countries is *even declining in real terms.*

Despite this, many patients now know what surgery has to offer, so that their expectations increase steadily. It is deplorable how poor some are. Of the US$2-$6/yr per head that is available in many developing countries for all forms of health care, half or more is spent in the cities, so only US$1 a head or even only a few cents are available in the rural areas for both hospital and health centre care.

The *per capita* income in the rural areas in many places of the world where 80% of people live may be <US$50/yr; the cash income is even lower than that. Estimates as to how much an Indian villager can spend on health care range from US36c-$6/yr. It is however less the cost in cash which devastates the family, than the complete disruption of their earning power.

Fortunately, the kind of surgery we describe is remarkably cheap and cost-effective compared with the high-technology surgery of the industrial world. But it is not so cheap in terms of a villager's income. If you work in a government hospital, such funds as you have may be provided for you, but increasingly patients or their relatives have to source the wherewithal for their own treatment, often on the black market. The reliability and suitability of such practice is obviously small, and the opportunity for corruption great. If you work in a voluntary agency hospital, your patients probably have to pay, and if you really want to care for them, you will have to keep your costs low. Complicated methods can easily lead to rising costs, and so gradually drive the most needy away. Instead, your hospital may fill with richer patients, who could, if they wished, seek care in the towns. You may become too busy even to notice this! Your high standing in the community may cause you to befriend the elite, and you end up neglecting the poor.

(a) Pulling a hospital 'out of the red'

Here is some advice principally from Tumutumu PCEA Hospital in Kenya which was able to turn a substantial deficit in its accounts into a surplus in two years. Try to make the containment of costs, or their reduction, an activity which all your staff share. They and you should know how much everything costs. If you can make your financial decisions by mutual consensus, they will be implemented.

Form an action committee consisting of all the spending departments: the medical superintendent, the administrator, the matron, and the senior medical assistant. Meet weekly and pass all decisions involving money through this meeting. A good time to start holding such meetings is after some crisis has occurred, for example, being told to cut your budget by 40%. A crisis atmosphere makes people more cooperative and more willing to change their ways.

Examine all funds coming into the hospital and all funds going out of it, scrutinize all bills and orders. Discuss demands from each department, and reject any unnecessary ones. Scrutinize all expenditure and expect to make some savings on almost everything. No single item is decisive, but collectively they make the big difference. Look at the large items first: salaries, transport, drugs and food; even small percentage savings here will have a big overall effect. Rationalize the use of drugs, especially antibiotics. Look at your establishment figures. You may find that your hospital has got fat and that you should let it get a bit leaner by not recruiting after natural staff wastage. You may find that you have to return to the staffing ratios and technologies (such as making your own plaster bandages) of earlier years. For example, you will probably find that most patients with pneumonia can be treated without a radiograph and so can most extension fractures of the wrist. Economize with sutures, IV fluids, lubricant jelly, stationery and so on. Use IV drugs only when you have to; remember to use the rectal route (PR) if the oral route is impossible.

Hospital meetings may often be critical. They will ensure the cooperation of the leaders of all sections of the hospital, who will transmit the sense of urgency to everyone else. They will also help to create an awareness of the economic implications of a decision, to establish priorities, and to ensure the continuation and extension of your economy drive. Follow up your decisions; someone must check that the fire is extinguished once the water is hot, or that the right weight of the right cabbages has been supplied. Make sure that the staff know how much money is running through their hands, and that the viability of the hospital depends on how they use dressing materials, gas, and equipment. A public chart showing hospital income and expenditure monthly will give employees, and potential donors, an understanding of your situation.

Money coming in is no less important than money going out. So try to keep your beds full. Work out a policy to reduce costs to the patient, and to make your services affordable to as many people as you can. Think about what they can pay and be prepared to lower some charges. However, you may be able to offer special treatment, for example in a private ward, to paying patients, especially if they have medical insurance. Such patients may prefer to come to your hospital for more individual attention than a large teaching hospital in the city. Consider income-generating projects: a restaurant at the hospital, a vegetable garden, a dairy, a maintenance service, a garage, a hairdressing saloon etc.

You may find it financially more reliable and less stressful to lease such activities out to a local entrepreneur. Engage your long-stay patients in making handicrafts or using their skills for the hospital (*e.g.* carpentry, electrical work, or sewing). Persuade the major players in the community to invest in the hospital, *e.g.* the bank or post office; a branch at the hospital will be very popular with staff and a big time saver. This requires marketing and data collection: make a survey of local demands and needs. One hospital in India had considerable success producing CDs of elective operations and selling them to the patients concerned!

Try to twin your hospital with an institution you know in a richer part of the world: the benefits of such contacts are not just economic!

Your greatest asset is the pathology arriving at your door: use it! Even simple, but carefully carried out, research is valuable and will attract funding to your institution from outside agencies.

Valuable contributions to the surgical care of the poor have been made in South America. In Colombia, it was found that 75% of all the operations were simple enough to be done on outpatients with a single anaesthetist supervising 2 patients simultaneously in the same theatre, mostly using local and epidural methods, and adequately supported by assistants. Operating theatres were only used for 40% of working hours, surgeons only did 120 operations per year and 'physicians' only 18.

In most hospitals, services are limited less by resources than by motivation. So expect to be able to do much more, even with what little you think you have. The rest of this section shows what can be done even when resources seem to be already stretched to their limit. If you think that checking the stores is not your responsibility, remember that it is critically important for the financial viability of the hospital, on which your whole surgical endeavour depends.

(b) Economical surgery

STAFF. You may unfortunately have no control here, but your influence is great. If possible, try to reduce staff to the bare minimum by not replacing unnecessary personnel, and make sure they do a full day's work. Keep existing staff busy with additional duties. Junior staff are often willing to have more responsible jobs such as filing and typing, or even preparing IV fluids. Try to lay off consistently dishonest and inefficient staff. Encourage punctuality, tidiness and cleanliness. Employ inexpensive ungraded staff where you can, to relieve more expensive staff of routine tasks. Employ multipurpose workers, such as a laboratory technician who can take radiographs. Employ married couples where both partners are gainfully employed. *Don't forget training programmes*, and encourage success by certificates and ceremonies. Take advice across the board: anyone may have a good idea! This is a strong motivator for staff as they feel involved.

(c) Savings on consumable materials

Dressings. If necessary, you can treat most wounds without dressings. *Clean closed surgical wounds don't need them.* Use gauze and cotton wool economically. *Don't make dressings larger than is necessary*. Re-sterilize all dressings which have not been soiled.

Avoid using strapping, but if you do use it, use narrow strips and *don't allow it to be used anywhere except on the human body*.

Hold dressings on with bandages, socks, caps, bras, tight vests, pants etc.

Wash gauze sponges, immerse them in water to remove stains, dry them and re-sterilize them. If necessary, cut up an old polyurethane foam mattress or cushion into small squares and use these as swabs and sponges. They absorb blood well. Cut up and sterilize old linen. Sterile toilet paper can be used as an alternative to swabs for some purposes.

Make up laparotomy pads. Use a sewing machine to join enough pieces of gauze 20x25cm together to make a 5mm layer; attach a tape to one end, and when you operate attach a large haemostat to the tape and leave this hanging out of the wound. Laparotomy pads are a more convenient and economical way of washing and reusing gauze than using it as swabs, and can replace them for some purposes.

Keep an open wound wet with water. Keeping a wound dry uses many more dressings than treating it wet. The water need not be sterile, and need not contain salt (except where sodium loss is important as in burns). Use large quantities of water: soak, wash, shower or spray the wounds!

If a wound is suitably sited to be immersed, as with the arm, leg, or buttocks, immerse it in water for 3h bd. Put a leg in a bucket, an arm in a long arm bath, and let a patient with a buttock wound sit in a hip bath.

If a wound is not suitably sited for immersion, keep it wet all day.
N.B. Dressings in these situations only serve to protect the environment.

Disinfectants. *Don't fill gallipots to the brim*. Use cotton wool, not gauze, for scrubbing the skin. *Don't use disinfectant for the preliminary 'scrub' to remove dirt*; use soap and water. One gallipot of disinfectant will then be enough to prepare the skin. You can use it all day: it is self-sterilizing.

Disposable items. Avoid these and replace them with permanent equipment. If you buy plastic equipment which is intended to be thrown away, choose the kind which you can autoclave or boil. Recycle everything you possibly can, and try to throw nothing away.

Buy the kind of gloves you can re-sterilize 3-4 times. Re-use clean sterile gloves as disposable gloves. Re-use clean disposable gloves for general cleaning work.

Use nylon syringes, such as the French KIGLISS pattern, which you can sterilize indefinitely, and which have a rubber ring to seal the plunger which you can purchase separately.

Don't use disposable urine bags; instead, use bottles and tubing from old intravenous sets. Re-use endotracheal tubes after thorough washing and cleaning with 'Cidex' (2.5).

Catheters. Use simple Jacques catheters if they are less expensive than Foley catheters; if you want to leave them *in situ*, secure them with strapping. *Consider carefully if the catheter is necessary anyway.*

IV fluids. Make your own for 7% of the price of the

commercial ones. Where possible, use rectal rather than IV fluids. These are not suitable for rehydrating patients, but they may be adequate for maintenance. If IV fluids are scarce for post-operative patients who have had major gastrointestinal or other surgery, insert a nasogastric tube for drainage and a naso-jejunal tube for feeding. In this way you will greatly reduce your need for IV fluid.

Oxygen is only necessary for such indications as pulmonary oedema, asthma, shock, or coma, but *not* for moribund patients. If you use it for patients with no hope of survival, relatives may come to believe that when you switched it off, it killed them! Get hold of oxygen concentrators: the economy is well worth the initial expense.

Drugs. Use cheaper drugs instead of expensive ones. For curettage of the uterus use pethidine with diazepam instead of ketamine; use aminophylline instead of salbutamol, aspirin instead of paracetamol, nitrofurantoin instead of ampicillin for urinary tract infections, and morphine instead of pethidine for many applications. Look carefully at the prices you pay for drugs. One supplier may be 100 times cheaper than another, *but beware counterfeit products!* Always consider if antibiotics are really necessary: they are often overused! *Don't practise poly-pharmacy!*

Sutures. Where possible, use surgical suture material bought in bulk on reels, or use nylon fishing line (4.6). Only use atraumatic sutures when they are absolutely necessary. With more expensive suture materials, use continuous sutures rather than interrupted ones. The application of warm moist gauze packs (especially if soaked in dilute adrenaline) to a bleeding surface will drastically reduce the number of bleeding vessels that you need to tie. Use sewing cotton for simple ligatures.

Scrubbing up. Use ordinary soap not special fluids, if the first costs less.

(d) Saving kitchen supplies

Find the cheapest supplier and buy at the right season. Find out if buying in the market may be better. Watch tenders carefully, change suppliers when necessary, and insist on good quality. *Don't let them supply you with old, rotten, or small potatoes*. Buy boneless meat, especially offal (liver or heart). Adjust the number of meals cooked to the bed state. Provide high-protein diets only on genuine indications. Reduce waste. Fill plates moderately and vary helpings according to the appetites of both patients and staff. Keep your own livestock to feed on waste and run your own vegetable garden if possible.

(e) Energy savings

Washing. Use the timers to set minimum times for washing and spin drying carefully. Avoid tumble dryers unless the climate is very wet; they use much electricity.

Petrol or diesel. Diesel vehicles may be cheaper to run but need more careful maintenance. Use the smallest economical vehicle for a given job and avoid unnecessary trips. Keep logbooks and use vehicles for hospital journeys only. Drive at economical speeds and use moderate engine revolutions in all gears. Use public transport wherever possible. Encourage a style of driving that is considerate for the vehicle, especially when carrying heavy loads on bad roads.

Gas. Put lids on pots. Reduce the flames when the pot has boiled. Use pressure cookers. Control cooking times. You may be able to insert a system that utilizes gas from compost or sewage, which is very cost-effective, although expensive to install.

Electricity. Switch off lights when unnecessary (*e.g.* in daylight!). Use fluorescent tubes instead of bulbs. Heating is much more expensive than lighting, so make sure it is used only where really necessary. Make sure you have universal connectors so you don't waste time and expense on adaptors. LED lights (*e.g.* on a headband) are extremely effective and use minimal power, so are useful if you have to rely on solar energy.

Air conditioning. In hot humid climates, a cool air environment makes life and work much more comfortable. Electrically driven air conditioners are expensive and frequently break down, and heat up surrounding areas outside the room they are cooling down! If you insert a system of PVC pipes 3m below ground, where the earth temperature is virtually constant, and blow air through these pipes with a simple fan, you can cool the room temperature by about 10ºC and reduce humidity by 40%. This system also avoids the dust that regularly contaminates electrical air conditioners.

Solar lighting is practical, virtually maintenance- and cost-free and its initial installation is becoming less expensive. Solar heating, by allowing the sun to warm black pipes, is very effective for producing hot water. Solar refrigerators are available, but their initial cost is high. Invest in inverters to convert solar 12v to 240v, but *beware that you don't overuse your batteries*. Use solar or hand-cranked batteries.

(f) Other savings

Use the space fully on all case sheets, use paper on both sides. Make your own forms with a stencil. Minimize the use of paper for internal correspondence. Use scrap paper for messages. *Don't use so much detergent that it causes foaming in the laundry and when scrubbing floors*. Register and charge for private phone calls. Send letters with your hospital transport if possible. Use e-mail or electronic messaging if you can.

Control all items that could be used in private homes, including torch batteries, soap, matches, pens, toilet paper, female sanitary pads, food and medicines. Be firm on discipline when it comes to theft. Remember theft probably accounts for your greatest 'expenditure': inventories and

security are mandatory. Proper accounting systems are also essential: otherwise money will just 'disappear'! Also, *don't delegate ordering of stock or equipment to a junior*; not only will you get the wrong things but you will be conned into buying expensive varieties of cheap things. Beware offers of 'new' equipment from unknown dealers: it is probably stolen or cheaply repaired and will *not* last.

Practise regular maintenance. Keep an eye open for breakages and organize repairs early. Establish a climate of accountability. Remember to order stock with sufficient time to allow for delivery and delays: *don't wait till the last X-ray film is in the hospital before ordering new supplies!* Otherwise you will be forced to improvise with expensive items because the cheaper ones have run out (*e.g.* using 3-way catheters when ordinary ones are actually needed).

THE PATIENTS ARE OURSELVES

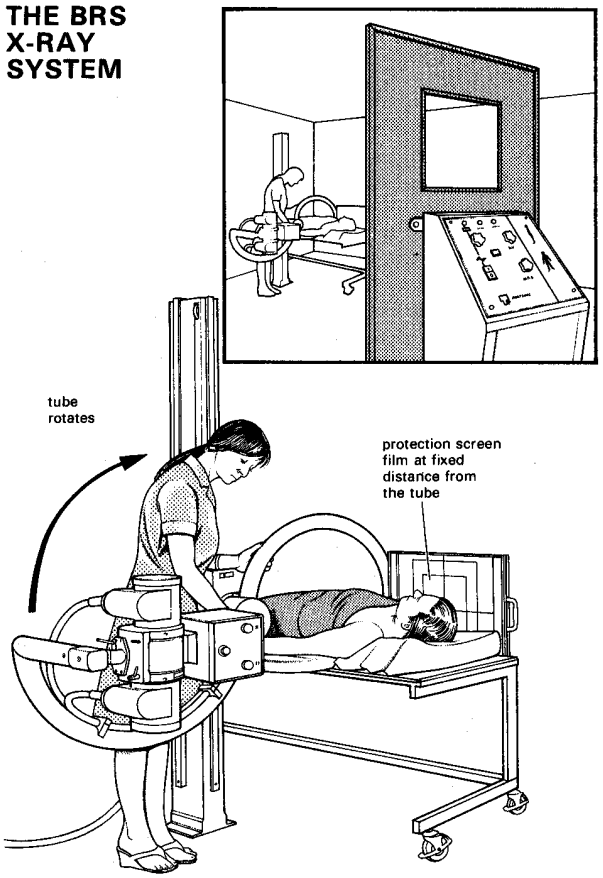


Fig. 1-8 THE BRS X-RAY SYSTEM was developed by WHO to make essential cost-effective radiology available safely and reliably all over the world. If you want one of these machines, order one made to WHO specifications. Note the screen protecting the operator.
Kindly contributed by Philip Palmer.

WHO has made a great advance in the X-ray departments of the world's district hospitals by developing the BRS (Basic Radiological System). The BRS machine shown (1-8) is made by several manufacturers to WHO specifications. If you are thinking of buying an X-ray machine, this is the one to get. If you don't have electricity all day, you can run it on a battery which you charge when you turn your generator on. It is so simple that a radiographic assistant can easily work it, but if you have a radiographer who has been trained to use a more sophisticated machine, he may not like this one because it does not give him enough freedom to adjust the settings.

The BRS machine is based on the assumptions that: (1) A good chest radiograph needs a short exposure, and a substantial distance between the patient and the tube. (2) A radiograph of the lumbar spine will be one of the heavier exposures required. It has therefore been designed to produce at least 80mA at 18kV, not one or the other, but both simultaneously. It has a fixed tube-to-film distance of 140cm, which gives satisfactory chest films and is the ideal distance for most other investigations. The tube is fixed so that it can use an accurately focused grid of high quality. The tube and the film are always accurately focused on one another and cannot be angled independently. This makes it easy to position the patient and makes routine views exactly repeatable. The supporting arm of the tube and the film can be rotated through at least 270º, so that horizontal and vertical projections are easy, and angled views are possible. Erect views of the skull, sinuses, shoulders, or abdomen are as easy as routine views of the chest. A radiographer's manual is available; so is a manual of radiography to go with the machine.

ECONOMY IS ESSENTIAL TO SURGERY

1.12 Primary care imaging

Radiology uses X-rays which provide much useful information, particularly about bones, but ultrasound (38.2) can replace radiographs for very many indications, especially in obstetrics except for X-ray pelvimetry. Think carefully if a radiograph is likely to give you essential information. Remember quite sophisticated interventional radiographs can be taken with simple means (38.1).

Ultrasound is an extremely useful modality, and you should really not be without this useful tool. Ideally, it should be portable, and must be suitable for obstetric evaluations. You don't need many types of probes, but it is almost essential to have a trolley where the probes can be safely placed so they are not damaged. You certainly don't need the extra gadgetry (freeze control) for taking still pictures, or on-screen measurements (though this is helpful). In fact, the fewer the knobs, the better and more consistent are the images you will obtain.

A computer attached and key console are not essential. The more features the system has, the less transportable it will be. If you intend to take it to distant clinics, make sure it is robust and comes with a specially padded case. Make sure it runs on rechargeable batteries.

A TALE OF FOUR PEOPLE, Everybody, Somebody, Anybody, and Nobody. There was an important job to be done and Everybody agreed that it could be done by Anybody. It was agreed that Somebody should be detailed off to do it, but although Anybody could have done it, it eventually got done by Nobody. Somebody got angry about it, after all (he said) it was Everybody's job. But, while Everybody thought that Anybody could do it, Nobody realized that Everybody was going to assume that Somebody was going to do it. It ended up that Everybody blamed Somebody when Nobody did what Anybody could have done.
LESSON This book is written to benefit Everybody, so that Anybody who is put in charge of surgical patients will know that Somebody cares enough to write down methods of surgery in a way that a 'Nobody' can find that he or she can do Something even if tucked away in the middle of 'Nowhere'.

1.13 How to use this manual

You will notice that after 5 chapters on 'the basics' there are 5 on draining pus. Then come chapters on the abdomen and hernias, followed by obstetrics, gynaecology, and the breast. After this there is the surgery of special areas (thyroid, proctology, urology, etc.) then finally terminal care and imaging.

After dealing with general principles of trauma management, Volume Two deals with various regions in turn.

In writing this manual, we have tried to make both language and the typography work for you. You will notice that we use the imperative, avoid the passive voice, and refer to 'the patient' and then mostly to 'him', which does in fact usually mean both 'him and her'. *Alas, English, unlike French, has no personal pronoun which includes both sexes and we have refused to use the grammatically awkward 'them'*. Our use of 'he' or 'him' to include both sexes improves clarity, and shortens the text, but we owe our apologies to our lady readers!

We have used 7 degrees of approximate commonness: very common, common, not uncommon, uncommon, unusual, rare and very rare. This is based on the experience of the authors and *may not apply exactly in your situation*.

Inevitably, we are mostly concerned with technology but *behind all this lies the patient himself.* The boy with the fractured radius and ulna waiting at the end of the queue might be your own son, that paraplegic your brother, that old lady with the fractured femur your mother. Tomorrow, you might yourself be that comatose patient with the extradural haematoma in the end bed. These patients are ourselves! Perhaps the thing that we most often miss is any explanation of what is going to happen to us, and any indication that anyone really cares. Believing the compassionate and devoted care of the sick to be one of the noblest human activities, and something of ultimate value for its own sake, we stress this!

We trust that this volume will enormously improve medical care as it did in St Francis Hospital, Ifakara, Tanzania. This showed that this manual had been put to good use. It contains much detailed factual information, and we have done our best to make it as easily understandable as we can. Feel free to disseminate this knowledge as you see fit: we make no restrictions on your keenness to photocopy the text. If, however, you would like to translate the text into your own language, please contact the editors, who will be happy to hear from you!

Take this book to the wards, clinics, and operating theatre. How does the treatment you see given differ from that described here? The methods of examination we give are summaries only; practise them on a fellow student.

We are all students, and should never give up learning new things. *Don't be overwhelmed by the mass of detail you find here. Don't panic,* and *don't think you need to read cover to cover!* These pages *differ enormously in importance.* Try to distinguish between what you should know, and what you can look up.

You will notice that much of the writing is didactic. This guidebook is a distillation of the cumulated experience of very many dedicated surgeons and physicians working in challenging environments. Also, there are very few references, because adding these would have hugely increased the volume of the text, and they cannot readily be looked up by our readership. Many references are old, but are still very relevant in low-income situations, again reflecting how advances made in the rich world are often not translatable to the poor world.

If, however, you find something really does not work in your set-up or you have good practical suggestions, please write and let us know.

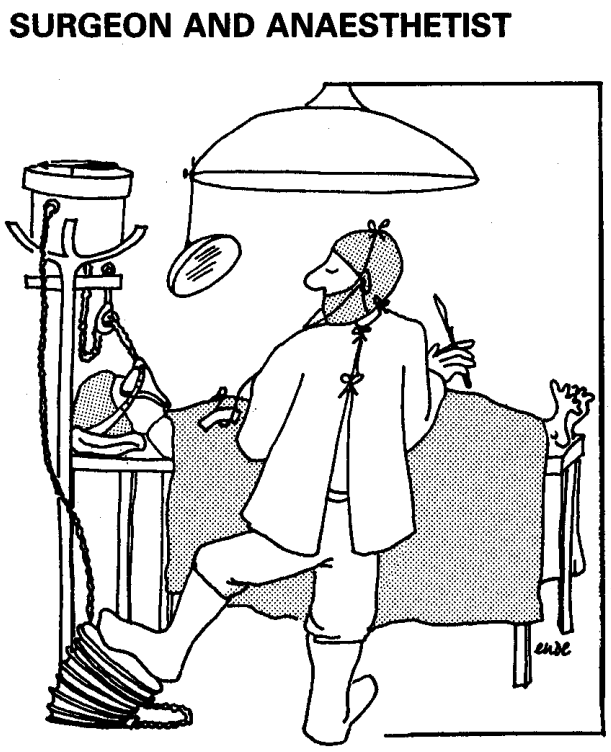


Fig. 1-9 YOU MAY HAVE SIMULTANEOUSLY TO BE SURGEON AND ANAESTHETIST.
Kindly contributed by de Glanville N. Proc Assoc Surg E Afr.
N.B. This cartoon is no longer very up-to-date: now you are much more likely to use ketamine than inhalational anaesthesia; also we recommend that you train a nurse or clinical assistant to monitor the patient during the operation, to warn you if there is a problem.

Certain drugs have been re-named in English usage according to European regulations; whilst generations of readers will probably still use and write the old names, the new ones are

given for correctness. Nonetheless, you should liaise with your pharmacy as to your own local usage! It goes without saying that prescriptions MUST be legible.

New Name	Old Name
Amoxicillin	Amoxycillin
Cefalosporins (all types)	Cephalosporins
Chlorphenamine	Chlorpheniramine
Diethylstilbestrol	Stilboestrol
Furosemide	Frusemide
Indometacin	Indomethacin
Levothyroxine	L-Thyroxine
Lidocaine	Lignocaine
Procain Benzylpenicillin	Procaine Penicillin

(a) A suggested initial reading list
Start by reading the whole of this chapter. In those which follow, read only the introductory passages, and merely glance at the detailed didactic instructions which follow. Read these carefully later when you need them to guide you in a specific situation. Start with the common things first.

Read particularly the first section of each chapter and the following: the major theatre (2.1), aseptic theatre technique (2.3), autoclaving (2.4), antibiotics in surgery (2.7 to 9), the control of bleeding (3.1 to 2), bloodless limb operations (3.4), the instruments (4.1 to 5), suture materials, sutures and needles (4.6 to 8), drains (4.9), instrument sets (4.13), 'pus' (6.1 to 24), pyomyositis (7.1), osteomyelitis (7.3), septic arthritis, especially the positions of rest and function (7.16), hand infections (8.1), empyemas (9.1), peritonitis (10.1), abdominal surgery (11.1 to 15), the acute abdomen and intestinal obstruction (12.1 to 16), appendicitis (14.1), inguinal and femoral hernias (18.1 to 8), and PID (23.1).

(b) The main anatomical drawings are the following: mandibular region (6-7), parotid (6-8), tongue (6-9), anorectum (6-13, 26-1), anterior thigh (7-18), hand and tendon sheaths (8-4,7), pleurae (9-1), peritoneal cavity (10-5), anterior abdominal wall (11-1), broncho-pulmonary segments (11-23), biliary tract (15-3), inguinal region (18-3,4), uterine blood vessels (22-14), relations of the ureter (23-20), pelvic ligaments pelvis (23-21), eye (28-1), auditory pathways (29-2), carotid artery (29-7), tonsil (29-10), ventricular system (33-18).
There are also the following transverse sections:
forearm (7-8), thigh (7-9, 35-18), calf (7-11), hand (8-1), ankle (32-18), wrist (32-35), lower leg (35-20).

(c) If you are a general duty medical officer, *don't be ashamed to refer to this manual*. A patient will be more grateful for being correctly treated than for being wrongly treated because you could not remember something and had to guess! For example, you cannot possibly remember all the steps in the general method for a spinal injury, or a hand injury, so why not refer to them in front of a patient until you have examined so many patients that the necessary clinical routines become automatic? If the condition is difficult to diagnose, ask to wait until the end of the clinic, and then use the routines we give here to try to reach a diagnosis.

Keep this manual in the theatre. If a procedure is long or difficult, sit in an armchair and study it in peace before you try to do it. Then study it again after you have done it. *Don't expect to be able to do everything we describe immediately*. Progressively extend your practice, little by little.

Don't let things you cannot do, because you don't have the necessary equipment or drugs, prevent you from doing the things you can do.

Whenever you refer a patient, try to learn from the person you refer him to. If possible, be there when he is examined. In the same way, if someone refers a patient to you, he should be there so that you can teach him.

What methods are your staff using? For example, if medical assistants treat fractures in your hospital, study the methods they use and encourage them to use those described here. If they might find this manual useful, see that they have a copy and go through it with them.

If a patient dies and you are not sure of the diagnosis, try to get permission for a postmortem examination.

Make good use of the endpapers and charts you find in this manual:
WHO safety checklist (1.8), endoscopy form (13-10), partogram (21-2), fundal height chart (22-15), baby head circumference chart (33-17), and foetal growth centiles (38-6,7,8).
Where convenient, photocopy them and stick them up on the wall, or have them printed.

(d) If you are a surgical teacher, try to integrate this manual into your teaching, and base your examination questions on it. Aim, less that the students should know this manual, than that they should know their way around it and be prepared to use it.

(e) Hospital records
In-patient hospital records often provide life-saving information which cannot be found elsewhere; they are a medico-legal obligation, and should contain all the important details of patients. *There is no real need for nurses and doctors to keep separate records*. Both could write in the same set of notes!

A proper hospital filing system is essential; notes are best stored by number (not name, as patients may use different names on occasion) using the last two digits, thus:

……236000, 237000, 238000….259100, 269100, 278100….243200, 252200, 255200….etc…..209800, 243800, 246800etc…..256001, 264001, 265001….201002, 222002, 265002….etc

A patient's ID number could be used if necessary.

Patients' social details should indicate: name, date of birth, address, next-of-kin, and mobile phone number.

If you can get your hospital records digitalized, so much the better, but remember that your hospital 'memory' will need constantly to be upgraded!

(f) Medical notes should be accurate, legible and comprehensible. There should be an admission note (with history and physical findings), continuation notes (with results of relevant investigations) commenting on progress and giving instructions, and finally a discharge note.

It is good practice to provide patients with their own outpatient cards: brief notes are made on clinic visits, and inpatient summaries are included:

(1) Hospital number,
(2) Date of admission,
(3) Diagnosis with relevant signs,
(4) Operation done,
(5) Complications,
(6) Lab results (especially histology),
(7) Date of discharge and review.

Never ever be tempted to alter the notes of a patient.
You may, however, add a comment later (with a date) if you feel it appropriate.

IF YOU ARE A STUDENT, LEARN THE IMPORTANT THINGS FIRST

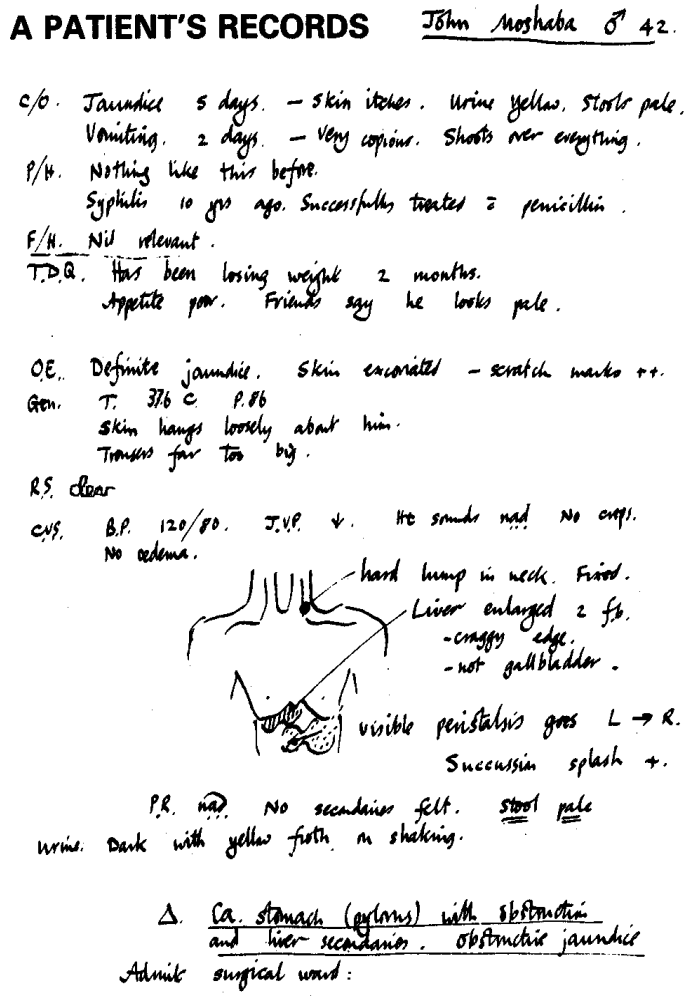

A PATIENT'S RECORDS
John Moshaba ♂ 42.
c/o. Jaundice 5 days. – skin itches. Urine yellow. Stools pale.
Vomiting. 2 days. – very copious. Shoots over everything.
P/H. Nothing like this before.
Syphilis 10 yrs ago. Successfully treated ∑ penicillin.
F/H. Nil relevant.
T.D.Q. Has been losing weight 2 months.
Appetite poor. Friends say he looks pale.
O.E. Definite jaundice. Skin excoriated – scratch marks ++.
Gen. T. 37.6 C. P. 86
Skin hangs loosely about him.
Trousers far too big.
R.S. Clear
C.V.S. B.P. 120/80. J.V.P. ↓. Hrt sounds nad No murs.
No oedema.
hard lump in neck. Fixed.
Liver enlarged 2 fb.
-craggy edge.
-not gallbladder.
visible peristalsis goes L → R.
Succussion splash +.
P.R. nad. No secondaries felt. Stool pale
Urine. Dark with yellow froth on shaking.
Δ. Ca. stomach (pylorus) with obstruction
and liver secondaries. Obstructive jaundice
Admit surgical ward:

Fig. 1-10 A PATIENT'S RECORDS, *as kept by Peter Bewes (adapted).* **Good notes are an excellent indication of quality of care. You may like to indicate the reasons for admission and orders: in this case, *"Prepare for Gastrojejunostomy. Check Hb. Rehydrate IV N/Saline 1L 3hrly with 1 ampoule KCl with each litre. Pass NG tube. Check clotting time. Inj. Vit K 10mg IM. Discuss with relatives. Get consent."***

2 The surgical infrastructure

"It is one thing to operate with the chief at your elbow on a patient whose vital functions are being monitored by an expert anaesthetist at the head of the table. It is quite another to be almost alone at midnight, struggling with a patient in shock from a ruptured ectopic pregnancy, as the light fades in and out while a superannuated generator tries to function on adulterated diesel oil. Then is the moment of truth when you realize that an excellent theoretical foundation is not the only thing you need." Hankins GW, the former Shanta Bhawan Hospital, Kathmandu, Nepal, Annals Roy Coll Surg Engl 1980; 62(6):439-44

2.1 The major theatre

Although aseptic surgery has been done in a tent, under a tree, or on a kitchen table, it is safer if it is done in a room which has been designed to preserve the sterility of the surgical field, to make surgical routines easier, and to prevent mistakes. The difficulty with aseptic methods is that they require an autoclave. If you don't have one, we describe an antiseptic method that you can use instead (2.6).

Do all you can to prevent nosocomial infections, *i.e.* those reaching the patient whilst he is in hospital. Such infection may come from himself, other staff, equipment and instruments, linen, furniture, floors, walls, water, toilets and insects. Things that come in contact with sterile internal parts of a patient need **sterilization** (2.4) whilst those that come in contact with intact mucous membrane need **disinfecting** (2.5). Other items need simple cleaning.

You will need 2 theatres at least: a major one and a minor septic one (2.2). We are concerned here with the major one. When you start work in a theatre, look at it carefully. How many of the desirable features that we are about to describe does it have? Is there anything which you could do to make it safer or more efficient?

The operating team should be as small as possible. It consists of:
(1) Yourself, the surgeon.
(2) Your assistant(s), when you need one or two.
(3) The scrub nurse responsible for the instruments.
(4) The circulating nurse to fetch and carry.
(5) The anaesthetist.
(6) His assistant, if he has one.
Two other people are important:
(a) the theatre charge nurse responsible for organizing the theatre, and who in a smaller hospital will usually take turns being on call,
(b) the 'theatre assistant' who, unlike the nurses who come and go, may have spent his whole career in the theatre and in that case will know its routines and where things are.
In an emergency, roles (2) and (3) can be combined in an efficient nurse or medical assistant, and so can roles (4) and (6). The first 3 members of the team are clothed in sterile gowns, the last three are not. An important part of the drill in theatre is to prevent the last 3 from contaminating the surgical field and the first 3.

Two zones in the theatre ensure this:
(1) A sterile zone which includes the operation site, the first 3 members of the team, and that part of the theatre immediately around them.
(2) An unsterile zone which usually includes the head end of the patient, separated from the surgeon by a towel rest, and the remainder of the theatre. The last 3 members of the team can move freely within this zone. The patient's entrance and the access to the sluice room are continuous with it. A separate room for scrubbing up is not essential; scrubbing is possible in the theatre in two domestic pattern sinks with draining boards. They should be fitted with elbow taps which are very highly desirable, although you can, if necessary, scrub up from a bucket or use spirit-based cleansing solutions. The boiler, autoclave, preparation room, and store rooms should be outside the theatre.

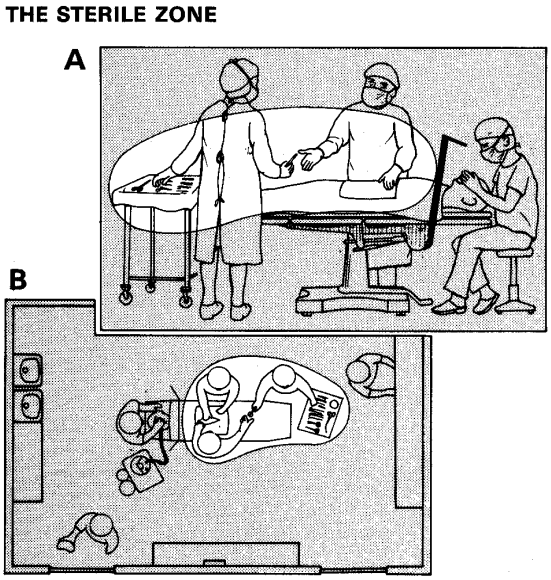


Fig. 2-1 STERILE AND UNSTERILE ZONES IN A THEATRE.
A, sterile zone (white) in a vertical dimension. B, sterile zone in a horizontal dimension. The sterile zone contains the operation site, the instrument trolley and the three scrubbed-up members of the surgical team. The unsterile zone comprises everything else in the theatre. The sterile zone is separated from the unsterile zone by a towel rest. The great danger, when technique is poor, is for the sterile zone to become smaller and smaller as the operation progresses.

Adequate space is essential, so that staff can move freely within their zones, and without touching one another. Space is needed for manoeuvring and parking the patient's stretcher next to the operating table, and for parking trolleys without congestion. The absolute minimum is $25m^2$; a room 5x6·5m ($32m^2$) is better, and $42m^2$ is ideal. The more equipment you have in the theatre, the more space you need, and in ideal conditions $64m^2$ is normal.

If the case load is heavy, a 2nd theatre is more useful than making the 1st one unduly large, unless you plan to run 2 operating tables in the same theatre; this is useful if you have only one anaesthetist to supervise cases. *A large operating theatre with areas not used will not be properly cleaned.*

Straightforward physical cleanliness is important. Sophisticated methods are unnecessary. Sluicing the floor between cases, washing the walls weekly and mobile equipment daily will ensure a high enough standard without using antiseptics on the theatre itself.

The floor is important. The most dangerous sources of infection are pus and excreta from the patients, which must be cleared away between every operation, and must not be allowed to contaminate the theatre. To make this easier, it should have a terrazzo floor, but a smooth concrete finish is almost as good and much cheaper. To make it easier to wash down, it should have a 1:1000 slope towards an open channel along the foot of the wall at the unsterile end of the theatre. This channel should have a plugged outlet leading directly outside to an open gulley. Fit a sparge pipe to the wall at the sterile end 150mm above the floor, so that the whole floor can be flooded by turning a tap. A little dust on trolley wheels or shoes, or from open windows, is less dangerous than is generally believed, but remember hair, fibres and fluff tend to get caught in wheels and need to be periodically removed.

The walls of the theatre should be smooth, but they need not be tiled. A sand and cement backwash application painted with one coat of emulsion and two coats of eggshell gloss is adequate. Gloss paint is satisfactory for the walls, and the fewer the doors, sills, ledges, crevices, mouldings, architraves, and window boards, the better. The main point is that the walls must be washable preferably up to 3m.

Every time a door is opened, dust from the floor is whirled into the room. There is no need for a door between the changing rooms and the theatre. A door is only needed between the sluice and sterilizing room, if these rooms will be used when the theatre is not.

The ceiling should be at least 3·5m high and the roof timbers solid enough to support an operating light. It should also have a pair of 2m fluorescent tubes or LED lights. The ambient level of illumination should be high, so make the windows big enough. They may enable most operations to be done by daylight. A suitably placed mobile mirror to catch the bright sunlight is very useful. There should be a window of $5m^2$ at the head and the foot ends, facing north and south shaded by a roof overhang of at least 800mm. Even better are windows on three sides. Fit ordinary low windows, and frost only the panes below eye level, so that the staff can look out (which improves morale), but that anyone looking in can only see their heads, not the patient.

In the tropics avoid windows in the roof. You may need fans to reduce the temperature, but *remember they can blow dirt and dust into wounds! Electrical air conditioning is notorious for collecting dust and transmitting bacteria*: it should not be a high priority; use an alternative (1.11). Make sure the theatre still remains warm enough: hypothermia due to exposure still occurs in tropical regions, especially at night. *It is comfort of the patient, who is practically naked, not of the surgeon, that is important.*

Don't have more shelves than you need, but keep the things you need daily nearby; use trolleys where you can. When shelves are needed, set them 50mm away from the wall on metal rods, so that they can be lifted away for ease of cleaning. All shelves should be at least 1m high so that trolleys can be pushed under them. The glove shelf should be at least 1·2m high, so that you can keep your hands higher than your elbows to prevent water running back down over your now dry hands. The anaesthetist needs a lockable cupboard, a trolley, and a worktop near the patient's head.

Electric sockets should be 1·5m above the floor to minimize the danger of igniting explosive gases, and damage from moving beds and trolleys. Make sure your electric sockets are uniform, and you have equipment working with the hospital voltage. You can easily overload the system if you have lights, a sterilizer, suction machines, lights, fans and diathermy all working at the same time. Make sure you have an emergency power source. A foot suction pump and hand-torches are useful in a crisis.

Basic requirements are:

OPERATING TABLE, simple pattern. The minimum requirements of an operating table are that: (1) you must be able to tilt the patient's head down rapidly for the Trendelenburg position, and if he vomits (2) you should be able to adjust its height. This table does these things at a fraction of the cost of the standard hydraulic ones, which need careful maintenance and are useless when their hydraulic seals perish. However, if a simple general purpose hydraulic table is well maintained, it lasts a long time. A really sophisticated one can cost as much as the entire building of the theatre. A dirty table is a menace, so make sure yours is kept clean.

If the head of your table does not tilt head down, get one that does. Meanwhile, in an emergency, you can put a low stool under the bar at its foot. If it does not tilt from side to side, make a wooden wedge to fit under the mattress. If it does not have a kidney bridge and you need one, use folded plastic covered pillows.

Locally made 'Chogoria' supports (19-3) are a useful addition to a standard table. They are made of 2 suitably bent pieces of pipe which fit into the holes for ordinary stirrups and keep the patient's hips widely abducted, and the hips and knees moderately flexed, so that the lower legs are horizontal. The legs rest on boards attached to these pipes. These supports are more comfortable than stirrups and are particularly useful for such operations as tubal ligation.

ALTERNATIVE OPERATING TABLE, Seward minor or equivalent. This is slightly more versatile and considerably more expensive than the table above.

MATTRESS, for operating table, with three or more mackintosh covers. A dirty mattress is a potentially serious source of infection. So swab the cover after each patient, and replace it regularly.

ARM BOARDS (2), for operating table, locally made. These are simply pieces of hardwood about 20x120x1000mm, which you push under the mattress to rest the patient's arm.

STOOLS (2), operating, adjustable for height, local manufacture. If you do much operating, a chair with a padded seat, wheels, and a back greatly reduces fatigue.

LIGHT, operating theatre, simple pattern, preferably with sockets to take bayonet or screw fitting domestic pattern light bulbs, in addition to special bulbs. Most operating theatre lights take bulbs which are irreplaceable locally, and may cost US$70 each, so find out what bulbs your light takes, and try to keep at least three spares. Record their specification and catalogue number somewhere on the lamp casing. When new lights are ordered, they should have fittings that can, if necessary, take ordinary domestic bulbs. An LED operating or head light is a very useful help or alternative.

The preparation room should lead off the theatre. A big one is desirable, because it needs to contain 2 autoclaves, a large and a small sterilizer, sterile packs, instrument cupboards and space to lay out instrument

trolleys. Ideally, it should be 64m^2 and serve 2 theatres. About 25m^2 is the absolute minimum, with a terrazzo shelf round most of two walls, a sink, a draining board, a single vertical autoclave (preferably two), a large boiling water sterilizer standing on the floor, and a small one on the bench.

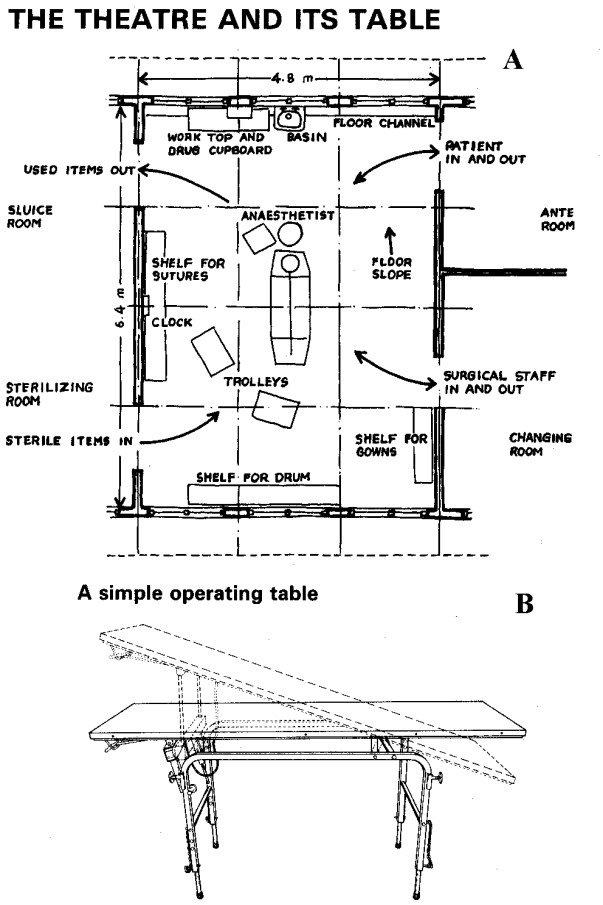


Fig. 2-2 A SIMPLE THEATRE AND ITS TABLE.
A, this is about the smallest practical theatre possible. B, simple pattern operating table described.
A, adapted from Mein P, Jorgensen T, Design from Medical Building, AMREF, Nairobi, 1975 with kind permission.

SPOTLIGHTS (2), free standing on the floor, 'Anglepoise' type, to take ordinary domestic pattern bulbs. Also, *high efficiency internally reflecting bulbs (5) to give a parallel beam*. These are necessary, both as a standby to the main theatre lamp, and to illuminate positions that the main theatre light cannot reach. A normal spotlight can direct an undesirable amount of heat into the wound, so, if possible, get LED lights which produce little heat. These are more expensive initially, but have a longer life. You can improvise a spotlight by removing the headlight of a car, especially the sealed beam type, and attaching it to a drip stand in the theatre. Connect it with a long lead to the battery of a car outside. Or use a slide projector held by an assistant. If the level of illumination is not enough, especially for eye surgery, you can increase the contrast by blacking out the theatre.
SOLAR PANEL, charger, and battery. A single solar panel will collect a useful quantity of electricity and enable you to light two wards in the evenings.
BATTERY CHARGER for the common sizes of rechargeable dry batteries, and five rechargeable batteries of each size. This will enable you to recharge batteries for your torches and laryngoscopes etc.

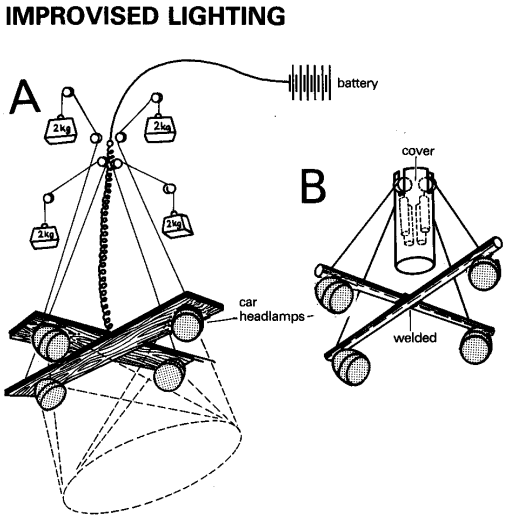


Fig. 2-3 IMPROVISED LIGHTING.
A, if you have to make a light locally, suspend 4 car headlights on a cross, and suspend each end of it on a pulley counterbalanced with a weight. B, better, put the counterweights in a metal casing which will be easier to keep clean. Or, less satisfactorily, hang three fluorescent tubes from the ceiling in the form of a triangle. This is basic but significantly better than nothing!

CLOCK, wall, electric, with second hand. This is essential; you must have a proper awareness of time, especially when you apply a tourniquet (3-6), and without a clock you can readily forget it. The instructions given here for controlling bleeding by applying pressure sometimes tell you to wait 5mins by the clock.
INSTRUMENT CABINET, glass door, sides and shelves, 1300x600x400mm, local manufacture.
RADIOGRAPH VIEWING BOX, standard pattern, local manufacture.
INSTRUMENT TROLLEYS (4) without guard rail, with two stainless steel shelves, antistatic rubber castors, (a) 600x450mm and (b) 900x450mm. Glass shelves ultimately break, so stainless steel ones are better. A larger table will make it easier to lay up for larger cases, especially orthopaedic ones.
STAND, solution, with antistatic rubber-tyred castors, complete with two 350mm stainless steel bowls, side by side. Put water in one bowl, and use the other for spare instruments and the sucker. The bowls can be sterilized in the autoclave or in a boiling water sterilizer.
DRIP STANDS, telescopic. Or, less satisfactorily, use long wire hooks suspended from the ceiling near the head of the table. Hooks for drips sticking out from the wall are useful above some beds in the wards.
SUCTION PUMP, operating theatre, electric with 2 1l unbreakable plastic bottles and tubing. These are always breaking down, so the model chosen must be easy to service and spares should be available. If you are going to depend on an electric sucker, *make sure it can actually suck before the operation starts.* A sucker which makes a noise may not necessarily suck.
SUCTION PUMP, foot operated, with 2 wide mouthed 1l unbreakable plastic bottles, rubber bungs and metal tubes.
This is an automobile pump with the valves in it arranged to suck instead of pumping. Both the surgeon and the anaesthetist need a sucker, so *you need 2* at least. A hospital workshop may be able to make one of these suckers by altering the valves of a truck tyre pump.
A foot sucker is much more reliable and more easily repaired than an electric one. *If you use an electric sucker, make sure you have a foot sucker also.*
SUCTION TUBES, metal, Poole's abdominal, wide bore, with guard. The standard laryngeal suction, the Yankauer type, is used by the anaesthetist at almost every operation, but not so useful for the surgeon. A small Gilles suction tube is useful for fine operations. Connect it through a piece of sterilized rubber tube to one of the suction pumps.
(*If you don't have a suction tube*, suck using the rubber end on its own, but beware of damaging bowel if the suction pressure is high.)
DIATHERMY. Bipolar diathermy is only useful for fine surgery; otherwise a simple unipolar diathermy is sufficient.
MONITORING EQUIPMENT. A pulse oximeter is very useful; a continuous ECG monitor is valuable but less essential. Expensive continuous blood pressure recording equipment is desirable but unnecessary, and if faulty may give a false sense of security.

SUITS, theatre, cotton, with short-sleeved shirt, and long trousers, assorted sizes, local manufacture. The purpose of these is to make sure that nobody enters the theatre in ordinary clothes, or in clothes worn elsewhere in the hospital. Everyone entering a theatre should put on a theatre suit in the changing room, having taken off their outside clothes. These suits should be laundered, and if possible ironed, but need not normally be sterilized each time they are used, unless they have been used for septic cases. You should discourage the habit of staff who have been out of theatre in their theatre suits, coming back without changing.

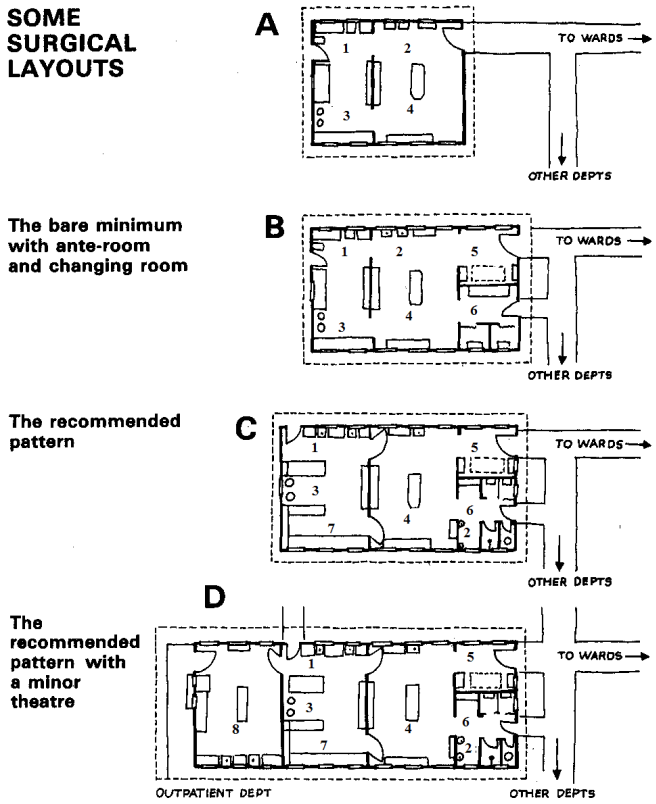


Fig. 2-4 SOME SURGICAL LAYOUTS.
This incorporates the theatre in 2-2 in progressively more developed settings. A, the absolute minimum. The changing is done in the sterilizing room. B, similar but has an anteroom and staff changing room. C, the arrangement recommended, which is 2-3x the cost of A. (1) sluice. (2) scrub up. (3) sterilizing room large enough to prepare sterile items for the rest of the hospital. (4) theatre table. (5) anteroom. (6) changing room with shower and toilet. (7) cupboard. D, further addition of (8), a minor (clean) theatre. *Adapted from Mein P, Jorgensen T. 'Design from Medical Buildings' AMREF, Nairobi, 1975 with kind permission.*

CLOGS, assorted sizes. Rubber boots are outmoded; sandals are less easy to keep clean and as they are open, provide inadequate protection. Use them only at the barrier between the theatre and the rest of the hospital.
APRONS, mackintosh, assorted sizes, local manufacture. These protect the suits and are worn under a theatre gown. If they are merely hung up in the changing room after use, they become progressively more contaminated and more dangerous. So, make sure that they are at least washed and regularly swabbed down with an antiseptic solution, and are always swabbed after septic cases. Keep two for special clean cases only.
CAPS, cotton. Put on a cap before you enter the theatre, and make sure it completely covers your hair (scalp and beard)!
MASKS, theatre. The use of these is controversial: if the surgeon has a bad cold, he should better not operate. *Most masks don't prevent passage of air-borne bacteria, and cotton muslin masks are useless*. They do protect against blood splashes, and should be used to cover a surgeon's beard!
GOGGLES, wrap-around, industrial. These should be used when drilling or splashes are expected.
GOWNS, cotton. These should go right round the wearer and cover the back. They should have long enough sleeves to reach the surgeon's wrists. Before sterilization they must always be folded so that the inner surface on the wearer is exposed to the outside in the drum.

GLOVES, operating, sizes 6 to 8. Remember that gloves are designed to protect the surgeon as much as the patient. The type of gloves you buy is critically important, and so is the relative number of the various sizes. It is useful if they can be re-sterilized, when not soiled by their first use. Most females wear sizes 6 to 7 and most males sizes 7 to 8. Pack each pair in a cloth or paper envelope, one glove on each side with its cuff turned outwards. Gloves are more useful to protect you and the next patient, than the patient you are actually operating on. Long arm-length gloves are useful for septic or bloody laparotomy cases.
GLOVES, industrial. These are useful for picking up hot objects and cleaning floors and surfaces; used on the correct indications, they will save many pairs of surgical gloves.
N.B. Avoid glove powder, especially starch or talc because it causes granulomas particularly in the abdomen, and also is prone to produce allergic reactions. Never use it when preparing equipment for auto-transfusion.
SOAP, hexachlorophene, carbolic. If necessary, the cheapest soap that does not irritate the skin will do. A liquid soap dispenser may prove not only more efficient but more economical. Spirit disinfectants between clean cases are effective and save on soap.
BRUSHES, nylon, nesting, autoclaveable. Autoclave several of these each operating day and store them between cases in a bowl of antiseptic solution. They will last longer if you merely keep them clean and immerse them in an antiseptic solution.
TOWELS, cotton, green, theatre. (a) Hand towels 25cm square. (b) Theatre drapes 100x75 cm. (c) Abdominal sheets. An abdominal sheet covers a patient completely from head to foot and has a slit in it through which the operation is done. The upper end acts as a guard which keeps the patient's head and the anaesthetist out of the operative field.
GASES. Cylinders need to be re-filled; if this is not possible, for a reliable oxygen supply, an oxygen concentrator, which extracts the gas from the air, is very useful. Note that oxygen cylinders are black with a white top, whereas nitrous oxide cylinders are completely black.
ANAESTHESIA DELIVERY SYSTEM. A 'draw-over' low pressure system which is leak-tolerant and uses air is far more reliable than a sophisticated Boyle's machine. *Make sure equipment for airway management (ambu-bag, mask, Guedel airways, ET tubes, laryngoscope with working batteries and bulbs, and stethoscope) is always available.*
HEATER to warm the theatre when it is cold (especially at night), and to warm IV fluids and lavage fluid. *Even in tropical climates, patients (especially babies) can become hypothermic!*
Other supplies: *(1) Pyjamas and pyjama trousers. (2) Dresses. (3) Macintosh drapes, 75x100cm. (4) Squeegees. (5) Bucket and mop.*

N.B. Make sure extra staff can be found and called in case of complications or emergencies.

2.2 The minor theatre

A minor theatre for septic cases will help to maintain the sterility of the major theatre. Use it for draining all abscesses, and for the closed reduction of fractures. It will need a simple operating table which tips, and a second set of basic anaesthetic equipment, including especially a sucker and the equipment for resuscitation. It will also need at least two minor sets (4.13), three incision and drainage sets. If possible, the minor theatre should have its own instruments and not be supplied from the main one.
Don't use this minor theatre for general anaesthesia (GA) cases.
N.B. Remember there is really no such thing as minor surgery for the patient!

2.3 Aseptic safe theatre technique

In order of importance, the most serious sources of infection in a theatre are bacteria from:
(1) the pus and excreta left behind by previous patients, especially on equipment or towels, etc.
(2) the clothes, hands, skin, mouths, or perineal regions of the staff; the bacteria on them may have been derived from other patients.
(3) the patient himself.

Minimize the risk of infection by:
(1) following the design rules (2.1) as far as you can,
(2) washing your hands between patients,
(3) keeping the theatre as clean as possible, so that the pus and excreta of previous patients are removed,
(4) making sure that all the autoclaving is done conscientiously,
(5) following the rules about the indications for operating, the timing of operations, wound closure, and careful tissue handling,
(6) creating and maintaining the sterile zone in 2-1.

This sterile zone has to be created anew for each patient in a theatre in which the risk of infection has been reduced as much as possible. Its creation starts when a nurse swabs the top of a trolley with antiseptic, puts two sterile towels on it and lays out sterile gowns and gloves. The sterile zone grows as the surgeon, the assistant and the scrub nurse put on their gowns. The operation site joins the sterile zone as it is prepared with an antiseptic solution and draped. Thereafter, nothing which is contaminated must touch anything in this zone until the end of the operation. If the technique of the team is poor, the sterile zone becomes smaller and smaller as the operation proceeds.

If you work on two sites on the body at the same operation, start on the less septic site, and preferably use a separate set of instruments for each procedure.

As well as protecting the patient from sepsis, be sure to protect yourself! Hepatitis B and C and HIV (5.3) are serious risks, and transmission of these infections cannot be prevented by screening every patient or using special precautions in individual 'high-risk' cases. Always adopt danger-free zones for sharps. Be sure there is no direct handling of sharps nurse to doctor, or *vice versa*. Place knives and needles on syringes in a kidney dish in a 'no-man's land' where scrub nurse and surgeon *never* put their hands at the same time. Remove sharps by instruments and not by hand, and dispose them in specially designated containers for incineration. You should try to avoid using sharp retractors, skin hooks, and cutting needles wherever possible. *Don't use your hands as retractors. Don't try to find a needle lost in the tissues with your fingers*.

Handle needles with instruments; cut the needle off before tying a suture, or hold it at its sharp point with the needle-holder.

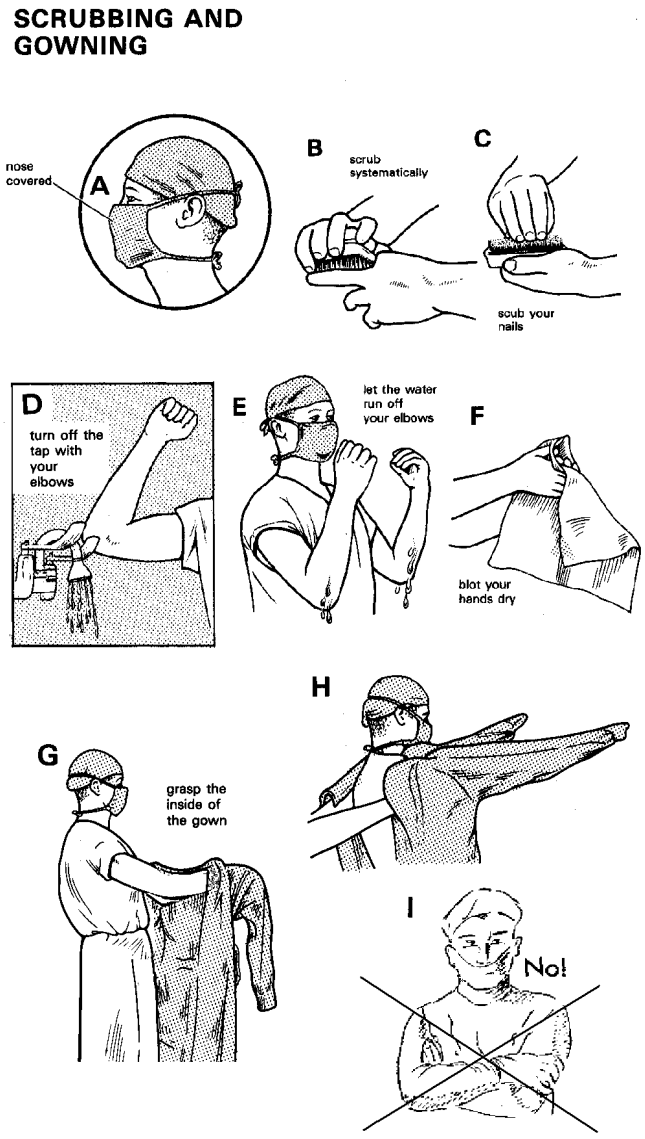


Fig. 2-5 SCRUBBING AND GOWNING.
A, make sure your mask covers your nose *(if you wear one at all)*. B, scrub your hands in a systematic manner. C, scrub your nails. D, turn off the taps with your elbow. E, while your hands are wet, hold them higher than your elbows. F, blot your hands on one corner of the towel, then dry your forearms. G, hold the gown away from your body, high enough not to touch the floor. H, ask the circulating nurse to grasp the inner sides of the gown at each shoulder and pull it over your shoulders. I, how *not* to wear your mask! *Don't put your hand in your axilla:* it is *not* a sterile area, even after gowning!

Wear wrap-around goggles when using high-speed drills, and where large quantities of contaminated fluid are expected.
Double-gloving decreases the risk of needle-stick injury, but does not eliminate it. You can use re-sterilized gloves for the first layer to reduce costs. Some surgeons prefer to put on one pair ½ a size larger on the outside, or on the inside.
Different coloured gloves may show up an accidental perforation more easily.
You can wear special Kevlar needle-proof gloves inside, but they tend to be cumbersome, especially for fine surgery (5.3).

(a) Entering the theatre. Anyone entering the theatre must change, in the changing room, into clogs and into theatre pyjamas or dress. This is important also when someone has left the theatre (in theatre attire) for the wards or casualty (accident and emergency) department, and returns. (Many hospital routines concentrate on putting on overshoes, gowns etc. on leaving the theatre; *more important is to change again on re-entering*.)

You must insist that *theatre clothing is not just worn over ordinary outside clothes*. Clogs are better than boots, which become sweaty and smelly. Tennis shoes are an alternative to clogs but get soaked by fluids. However, you can likewise soak them to clean them!

There is no proof that masks are helpful, except in protecting the surgeon (or nurse) from splashes. A sneeze passes through all masks; *a person with a bad respiratory infection should not be in theatre at all!* Masks are an unnecessary expense.

(b) Positioning the patient

Do this carefully before you scrub, so that you don't have to disturb him by altering the drapes or lights during the operation. Make sure IV lines, catheter and nasogastric tube are in place and functioning. Check that there is sufficient room for you, the anaesthetist, the scrub sister and an assistant (or two).

If you use diathermy, place the earth plate in contact with the skin of the buttock or leg *before* draping. Make sure it has been tested, *e.g.* on a bar of soap.

Pay close attention to pressure points, particularly in emaciated patients, and when legs are put in lithotomy position.

If a patient is in the lithotomy position, make sure he is pulled down sufficiently so that the perineum is then quite free from the end of the bed. *Make sure the legs don't fall out of the stirrups!*

If a patient is prone, make sure the abdomen is free to move with respiration. Fold the arms under a pillow on which the head, turned to one side, is resting.

If a patient is in the lateral position, make sure he is cushioned and supported, and there is a pillow between the knees.

Make sure the theatre lights are directed correctly once you have pumped the theatre table to an agreeable height.

(c) Scrubbing up. Remove any jewelry. Open a gown pack without touching the inside of the pack. Check that it is properly autoclaved. Adjust the taps to deliver water at a comfortable temperature. In most tropical countries only a cold water tap is necessary. Wet your hands, apply a little soap or detergent, and work up a good lather.

Rub your hands and forearms to 5cm above your elbows thoroughly. Wash your forearms and your hands. Then take a sterile brush and put soap on it. Scrub your nails (2-5C), thoroughly for the first case in the day.
N.B. Make sure all surgical staff keep their fingernails short, and have long hair tucked away!
Rinse the suds from your hands while holding them high, so the water runs off your elbows (2-5E).

Turn off the taps with your elbows, if this is possible (2-5D); otherwise ask someone else to do it. Blot your hands dry on one corner of a sterile towel (2-5F), taken from the gown pack without contaminating the gown itself. Then dry your forearms, using a different (dry) part of the sterile towel.

If you can get disinfecting spirit for the hands, you only need wash with soap initially or after septic cases; it is easy to become slack with any method.

(d) Gowning. Hold the gown away from your body, high enough to be well above the floor (2-5G). Allow it to drop open, put your arms into the arm holes while keeping your arms extended. Then flex your elbows and abduct your arms. Wait for the circulating nurse to help you. She will grasp the inner sides of the gown at each shoulder and pull them over your shoulders, and tie it at the back (2-5H). *Don't touch the outside of your gown till you have sterile gloves on.*

(e) Gloving. *Try to avoid using glove powder even if you are using re-sterilized gloves.* Be careful to touch only the inner surface of the gloves. Grasp the palmar aspect of the turned-down cuff of a glove, and pull it on to your opposite hand (2-6A). Leave its cuff for the moment. Put the fingers of your already gloved hand under the inverted cuff of the other glove, and pull it on to your bare hand (2-6B). Holding the sleeves of your gown tightly folded against your body, pull the glove over the wrist. Then do the same for the other hand.
N.B. If you do use powder, always wash it off your gloved hands with sterile water to remove it completely.

Now help the next person who has gowned on with the gloves.
(If you wear 2 pairs of gloves, you may prefer to put the first pair on before gowning. The first pair could be one that has been re-sterilized.)

You may prefer to ask the already scrubbed, gowned and gloved theatre nurse to hold open the gloves, with the cuffs everted, for you to slip your hands inside. This is easier and a safer method, but relies on the scrub nurse's gloves being sterile!

***MAKE SURE YOU HAVE FOLLOWED THE CAUTIONS LISTED* (1.8)**

It is a good idea if using local anaesthetic to infiltrate before scrubbing, in order to allow it time to take effect.

(f) The operation site

Make sure the patient has bathed before the operation and the operation site is clean. Remove any jewelry or skin piercing.
Check the side to be operated upon. *Make sure it is marked with a permanent marker.* If not, confirm the side with the anaesthetist *and* scrub nurse.
Put a septic limb to be amputated in a plastic bag already on the ward and seal the bag with wide tapes onto the leg. In the theatre cover the bag with sterile towels. Take the amputated limb out of the theatre before recovering the towels.
Check the position of the patient on the table yourself.

(g) Shaving. The operation site should be socially clean before the operation, and you may have to check this. There is usually no absolute need to shave a patient. If you shave or clip the hair, do so on the morning of the operation, or as part of the operation, and limit this to a narrow zone (2-5cm) around the planned incision. Make sure you remove the cut-off hair (this can be done with an adhesive tape and washing); otherwise the hair will end up in the wound.
If you do the shaving a day or two before, minute abrasions in the skin will become infected and the risk of wound infection will increase. Betadine shampoo, especially of the head and groin, is particularly useful after shaving.

(h) Skin preparation. Do this as soon as the patient is anaesthetized. Use an alcohol-based solution, preferably iodine, if possible: check for the patient's sensitivity. Take a sterile swab on a holder, start in the middle of the operation site, and work outwards. Be sure to prepare a wide enough area of skin, including any additional areas needed for example in skin grafting. In an abdominal operation this should extend from the patient's nipple line to below the groin.

N.B. Make sure the alcohol-based solution dries because of potential burn hazard if you use diathermy. Avoid spillage under towels and seepage under a tourniquet where it may remain in contact with skin for a long time and cause irritation.

N.B. There is no justification for using skin preparation twice.

(i) Catheterization. For major abdominal and pelvic operations, catheterize the bladder using an aseptic technique (27.2) before draping. *Don't catheterize routinely for other abdominal procedures*. Change your (outer) gloves: these can then be re-sterilized.

(j) Draping. Wait until the patient is anaesthetized. Aim to leave the operation site alone exposed and all other parts covered. Place the first towel across the lower end of the operation site. Place another across its nearer edge. Apply a towel clip at their intersection, under the folds of the drapes. Place another towel across the opposite edge of the site, and finally one across its upper edge. Clip them at their intersections. If the towels are in danger of falling off, secure them with a stitch. *Don't clip the skin with clips as this may cause skin necrosis.* For an abdominal operation, cover the whole abdomen with an abdominal sheet with a narrow quadrangular hole in its centre.
Remember to complete the draping at the beginning of the operation if you need more than one operation site, *e.g.* for skin grafting. Make sure the perineum is securely covered, and that drapes round limbs are secured snugly with clips or bandages. You can cover a hand or foot by putting on an extra large sterile glove and inverting it over the extremity.
If the patient is awake (*e.g.* with spinal anaesthesia), put drapes across two drip stands to separate the head from the operative field. If important areas near the surgeon become contaminated, remove them and cover the patient with fresh sterile towels.

(k) Suction tubing and diathermy. Secure these to the drapes securely with towel clips, so they don't fall off during the operation.

(l) Swabs and packs. Use 10cm gauze squares on sponge-holding forceps ('swabs on sticks'). You will also need abdominal packs. *Make sure these are counted* and checked at the end of each operation, and then disposed of quickly in the sluice.

(m) Cleaning the theatre. Remove clutter. Wash the floor and clean the table and accessories after each operation. Clean the theatre thoroughly after each day's list, and completely every week. Fumigate after a septic procedure with formalin.

(n) Cleaning instruments. Use an old nail-brush. Open hinged instruments fully, scrub them, and take special care to clean their jaws and serrations. *Beware of sharps!*

DIFFICULTIES WITH ASEPTIC METHODS
If you have no gloves or very few gloves, scrub up and then rinse your hands and arms in alcoholic chlorhexidine (2.5). The alcohol will dehydrate your skin. You can reduce this by adding 1% glycerol to the solution. Unfortunately, although antiseptics may help to protect the patient, they are not effective in protecting you from HIV (5.3) so use a 'no-touch' technique, using instruments between you and the patient. Limit your operating to emergencies.

N.B. **If you tear or contaminate a glove during an operation**, remove it. Grasp its cuff from the outside, and pull it down over your palm. Alternatively, if it is not soiled, put on another sterile glove on top over it, in the same way as described above.

If you have no drapes or gowns or very few of them, use plastic sheets and aprons and soak them in an antiseptic solution (2.5).

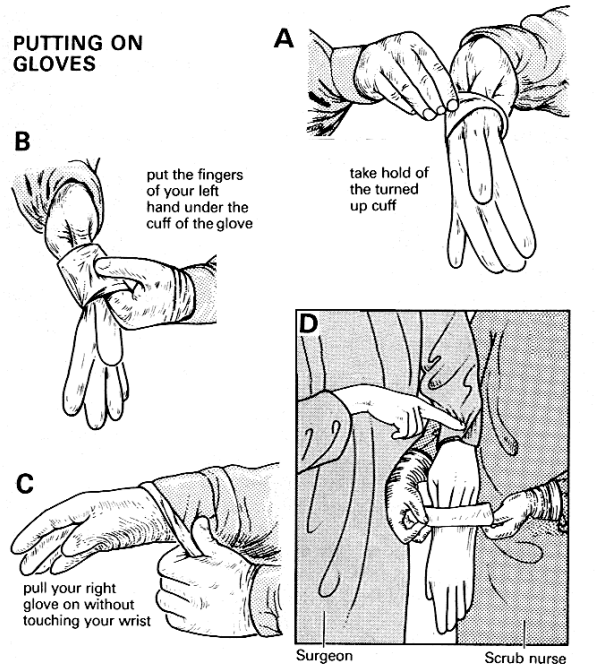


Fig. 2-6 PUTTING ON GLOVES.
A, take hold of the inside of the glove with your right hand, and put your left hand into it. B, put the fingers of your left hand under the cuff of the glove. C, pull your right glove on without touching your wrist. D, the first person to glove up (usually the scrub nurse) now gloves the second person (usually the surgeon), by holding out the gloves for him like this.

WOUND SEPSIS AND THE ART OF SURGERY

"In summary, I believe that regard for tissue is the foremost of our priorities. Let us strive to become first class surgeons, and let us train considerate disciplined theatre staff. Let us have plenty of soap and water, or some not too corrosive detergent. We do need sterilizers and autoclaves. We need well ventilated rooms which are light and easy to clean, and where the number of additional items is kept low. We should don theatre attire, should indeed change frequently, and should certainly change our masks. Gloves are important though not indispensable. Use sharp knives, few instruments and keep things neat and clean. *Don't bury undue amounts of biologically irritating material in the tissues*. Beware of haematomas and lymph collections. Use suction drains frequently. Use delayed primary closure where this is indicated. In the wounds you make yourself, bring the skin edges together carefully so that the wound is sealed in a few hours. Hydrate your patient, and *don't oversedate him.* Avoid stasis by elevation and movement. Use dressings sparingly, and observe the wound. If you find a haematoma and evacuate it speedily you will prevent sepsis." *Loefler IJP, Wound sepsis and the art of surgery. Proc Assoc Surg E Africa 1979;2:172-180.*

2.4 Autoclaving

Sterilization literally means destroying the fertility of organisms; in the hospital context it describes the elimination of all forms of contaminating organisms, including bacterial spores. Nitrogen dioxide (NO_2) is best; otherwise use heat, either dry heat in an oven, or steam under pressure in an autoclave. Processes (usually chemical) which don't destroy spores are termed 'disinfection'. Some of the most important agents to be removed by disinfection are HIV, HBV and HCV (hepatitis B and C virus). All the disinfectants mentioned (2.5) will do this if used as directed. If no alternative is available, hypochlorite is suitable for most purposes (5.4).

The basis of aseptic surgery is to kill all micro-organisms on all instruments and dressings, preferably by exposure to steam under pressure. If this is impractical, immersion in boiling water for 10mins at sea level will kill all viruses and all vegetative bacteria, but not spores, particularly those of tetanus and gas gangrene. A boiling water 'sterilizer' is therefore badly named. At a height of 3,000m above sea level, water boils at 90ºC and is much less effective.

Steam is simply the gaseous form of water; if it is to sterilize effectively, which means killing all spores:
(1) It must be at an appropriate temperature (which implies an appropriate pressure).
(2) It must be saturated with water.
(3) *It must not be mixed with air*, so it must displace all the air in the chamber of the autoclave.
(4) It must reach all parts of the load.

If it contains droplets of water, it will soak into porous materials. If, on the other hand, it is superheated and therefore too dry, it will be less effective as a sterilizing agent. If air is mixed with steam:
(1) The temperature of the mixture at a given pressure will be lower,
(2) It will penetrate less well into porous materials,
(3) The air may separate as a lower, cooler layer in the bottom of the chamber, so that the contents are not sterilized. If no air is discharged, the bottom of the chamber may be much cooler than the top.

As soon as the chamber of an autoclave is full of steam at the desired temperature and pressure, it must be held there for a critical time, the holding time. The standard holding time is 15mins, at 121ºC, but you will need to vary it as described below. This temperature is reached at a pressure of about 1kg/cm² (15psi). An easy minimum figure to remember is '1kg/cm² for 15mins' ('15lbs for 15mins'). If your autoclave is rated to 1·3kg/cm², you can shorten the sterilizing time to 10mins.

Here we only discuss the simpler forms of autoclave; high vacuum autoclaves are beyond the scope of this manual. Single walled autoclaves are strong metal chambers with water in the bottom, similar to large pressure cookers. They have several disadvantages:
(1) The air in the chamber is removed by steam rising from the bottom. This is inefficient, so that an undesirable quantity of air remains.
(2) They don't have thermometers at the bottom of the chamber, so you never know what the temperature there is.
(3) The load remains moist after sterilization, which can be dangerous, because bacteria can more easily enter through moist wrappings.
Double walled autoclaves can be vertical, but are much better horizontal. They should either have an effective pre-vacuum or a pulsing system (neither described here), or rely entirely on gravity to displace the air. A partial pre-vacuum at the start of the sterilizing cycle (which used to be the practice in some older autoclaves) causes turbulence when air is admitted, so that the gravity displacement of air cannot take place satisfactorily.

Steam is generated in, or admitted to, a jacket round the chamber, rather than in the chamber itself. This jacket keeps the walls of the chamber hot, which prevents condensation and helps to dry the load. Steam enters the chamber through a pipe at the top and displaces the air it contains. Air, condensate, and excess steam escape through a pipe at the bottom. This pipe has a thermometer in it to record the temperature in the bottom of the autoclave.

In some autoclaves a water pump, which works on the same principle as an ordinary laboratory water pump, sucks out some of the steam afterwards (post-vacuum). There is also a means of admitting sterile air to break the vacuum at the end of the cycle.

The drain at the bottom of the chamber should have a 'near-to-steam trap', which will allow the discharge of condensate and air, and will close automatically when they have been discharged, and the trap meets live steam, thus avoiding the need to close valve 13 (2-7) manually, which could spoil sterilization.

The thermometer records the temperature in the chamber drain, which is the coolest part of the autoclave. When this reaches the operating temperature, the timing of sterilization can begin.

More sophisticated autoclaves have better pumps, a recording thermometer, a thermocouple to measure the temperature of the load, and an automatic control system.

AUTOCLAVE, horizontal, downward displacement with near-to-steam trap in the chamber drain, post vacuum, six spare gaskets, three spare bellows for the steam trap, and a triple set of other spares. If you have a steam supply, this is the autoclave you need. Horizontal autoclaves are easier to use, but are more expensive. You will need a standby, in case the electricity fails, so you should have an autoclave that can be heated by kerosene or gas somewhere in the hospital (see below).

AUTOCLAVE, vertical, downward displacement, 350 mm, 2½ drum, electric, 6kW, state voltage, manual operation, with six spare elements, six spare gaskets, and a triple set of other spares as necessary. (A variation powered by gas is available.)

AUTOCLAVE, vertical, 'pressure cooker', 47L, UNICEF. This is a large autoclave which can be heated on a stove and has a machined lid so that it needs no gaskets. It is large enough for 5l of intravenous solution, or one laparotomy pack. It is a useful standby. It has an air exhaust tube which leads from the exhaust port to the bottom of the sterilizer. If you use it, you can start timing as soon as steam comes from the exhaust.

TUBES, Browne's, for testing autoclaves, Type 1 (black spot), for use with ordinary steam sterilizers below 126^{0}C. These change colour on the basis of time and temperature, and are reliable, provided that there is not a long drying cycle, when prolonged heat in a jacketed sterilizer could change their colour.

Or, *CARDS, autoclave testing, ATI 'Steam-clox'.* This brand of tape changes colour on the basis of moisture and temperature, to indicate that something has been autoclaved. Most other brands of autoclave tape are only suitable for high pre-vacuum autoclaves, not for the downward displacement ones described here. Another alternative is 'Diack® Control', a pellet in a glass tube which melts at 121 or 126^{0}C.

DRUMS, deep, 340x230mm. This is the standard size of drum.

DRUMS, shallow, 340x120mm. These are half-size drums. You may have difficulty getting drums because they are no longer used in the developed world. If you are short of drums, sterilize your equipment in packs, covered by two layers of towelling and preferably an outer layer of paper. If you are sterilizing without paper, use all equipment warm straight from the autoclave.

DRESSING BOXES, stainless steel, with hinged lid and perforated sliding shutters at front and back, 250x200x150mm. Use these for sterilizing gloves and dressings.

TRAYS, dressing, without lids, stainless steel, 275x320x50mm. Use these to prepare sterile sets for the wards. Boil a tray and the instruments, lay a sterile towel on the tray, put the instruments on it and fold it over them. Better, autoclave the tray.

Inadequate sterilization is an important cause of wound sepsis in poorly maintained theatres.

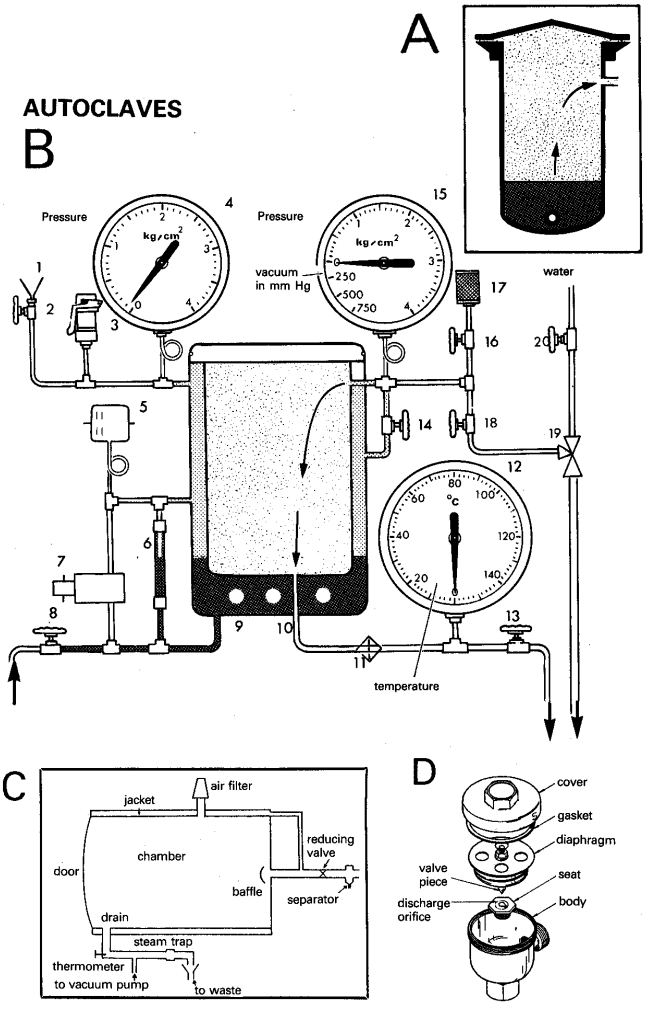


Fig. 2-7 AUTOCLAVES.

A, simple autoclave is a strong metal chamber with water in the bottom, like a large pressure cooker.

B, jacketed vertical gravity displacement autoclave. This is filled through a tundish (open funnel) (1) and a filling valve (2). On the same pipe there is a safety valve (3) and a pressure gauge (4) to measure the pressure in the jacket. A pressure switch (5) controls the pressure in the jacket and an indicator (6) monitors its water level. A float switch (7) cuts off the power if the water level is too low, and a drain tap (8) lets water out of the jacket. Several heating elements (9) heat it. The chamber is drained through a pipe (10) and a strainer (11). A thermometer (12) and a valve (13) are fitted to the drain pipe (the valve should be an automatic near-to-steam trap, preceded by a non-return valve, to prevent dirty air and some water being sucked up during the vacuum). Steam from the jacket is admitted to the chamber through valve (14). Pressure and vacuum in the chamber are measured by a gauge (15). Air is admitted to the chamber through a valve (16) and an air filter (17). Air and steam are discharged from the chamber through valve (18) by means of the water-operated ejector pump (19) operated by tap (20).

C, vertical gravity displacement autoclave. Steam is admitted fairly high up the sterilizer. The drain with the thermometer is as near the chamber as possible. There is a near-to-steam trap separated from the drains by a tundish, which prevents dirty water being sucked back up the waste pipe into the autoclave during a vacuum phase.

D, 'near-to-steam trap' (valve) in the waste line remains open, until steam following the air heats the bellows under the diaphragm and closes the trap automatically. *C,D kindly contributed by Ronald Fallon.*

STERILIZER, boiling water, electric: (a) 'Bowl sterilizer', 450x350x380mm, with counterbalanced lid, 6 kW, with six spare elements, state voltage. (b) Instrument sterilizer, 350x160x120mm, 1·2kW, with 6 spare elements, state voltage. One of these is for trays and bowls, and the other for instruments. Keep them both in the preparation room. *Never try to sterilize anything contaminated with faeces with boiling water in a sterilizer* – it does not destroy spores.
FORCEPS (2) sterilizer, Cheatle's, 267mm,
FORCEPS (2) sterilizer, Cheatle's, extra large, 279mm, complete with can of appropriate size for antiseptic fluid. These are useful for bowls and utensils, and will also pick up small objects.
FORCEPS, bowl sterilizing, Harrison's double jawed, complete with can of appropriate size for antiseptic fluid. Autoclave these and Cheatle's forceps and their cans after each day's use, then fill them with fresh antiseptic fluid.

Many hospitals don't have piped steam supplies. If so, use a vertical autoclave. Your electricity supply may be unreliable; think about using an alternative such as gas.

There are many pitfalls. Start by inspecting your equipment and taking an interest in it. Read the maker's instructions carefully, and make sure that:
(1) it has been properly fitted and tested. For example, if a water ejector pump is fitted, it is likely to need a water pressure of 1·5kg/cm².
(2) all the staff who use it understand how it works, and how to use it effectively. They must realize the importance of packing the drums loosely, the need to discharge the air, and the correct holding time.

(a) Sterilizing with moist heat

(1) **Boiling water**
Make sure that every article for sterilization is cleaned thoroughly to remove dried blood, pus or secretions before it is sterilized. Remove instruments from boiling water with long-handled Cheatle's forceps which have been in saponated cresol ('Lysol') up to their handles. If you are not wearing sterile gloves, make sure you let the instruments dry. If you use them wet, bacteria from your hands may flow down from your fingers in drops of water.

(2) **Packing any autoclave**
Sterilization is impaired by anything which hinders the removal of air, so arrange the contents loosely; a drum which can only be closed with difficulty is grossly overpacked. Place the contents so that air can readily be displaced downwards: the principles are the same in horizontal and vertical autoclaves. This means packing the items vertically rather than horizontally. To avoid air pockets, interleave sheets of mackintosh or jaconet with some permeable fabric, so that no two surfaces of the non-permeable material are in contact.

(3) A **simple autoclave** (or pressure cooker) (2-7A)
Make sure there is enough water in the bottom of the autoclave. Insert the drums to be sterilized, and turn on the heater. See that the discharge tap is open, and then screw down the lid. As the water boils the steam will rise and carry away the air in the autoclave.

CAUTION! Let the air and the steam escape freely until there is no more air in the autoclave; this usually takes about 10mins. To test this, lead a rubber tube from the discharge tap into a bucket of water.

When air no longer bubbles to the surface, there is no more air. After some trials, you will learn how long to allow for this to happen.

Close the discharge tap. Let the temperature rise until it reaches 121ºC. The safety valve will open and allow steam to escape. It should come out with a pure hissing sound rather than gurgling indicating the presence of air.

Now start to measure the holding period and continue this for 15mins. Then, turn off the heater and allow the autoclave to cool, until the pressure gauge records zero pressure. *Don't open the autoclave whilst the pressure is still high:* you might be badly burnt! Then open the discharge tap and allow air to enter the autoclave. Remove the load.

CAUTION! If anything in the load has paper or cloth wrappings, *don't allow them to touch anything unsterile*, until they have dried, because microbes can penetrate wet paper.

(4) **Jacketed autoclave** (2-7B)
Keep the jacket full of steam at 121ºC throughout the working day. Drain the chamber to remove any water that may gather in it. Load the heated chamber, close the lid, and *open valve* (13).

Sterilizing. Open valve (14). When the temperature on thermometer (12) has reached the sterilizing temperature (usually 121ºC), the holding time can start. Close valve (13). If it is letting much steam through, the temperature will not reach 121ºC, until it is closed. So, close it as soon as no further air and condensate come out of the chamber. If you still don't get the temperature you need (usually 121ºC), open valve (13) for a minute or two and try again (a near-to-steam trap does this automatically). When the temperature has been reached, start timing.

CAUTION! Don't infer the temperature from the reading of the pressure gauge. This may give you an inaccurate indication of its temperature and is a common cause of sterilization failure.

For drying (post-vacuum), open valve (20), then valve (18). Leave them open for 15-20mins. Close valve (18) then valve (20). Break the vacuum by opening valve (16).

(5) **Testing autoclaves**
If you are using Browne's tubes, put a tube in the centre of the load, with, if possible, one on the outside to show that the autoclave has indeed been switched on!

If you don't have Browne's tubes, put some dry earth in an envelope, autoclave this and then culture it in a bottle or tube of nutrient broth. Spores may be slow to grow, so incubate it for a week. If even this is impossible, put an egg in the middle of a drum to see if it is hard-boiled!

(6) **Particular procedures for autoclaving**
The following figures are guidelines only and vary with the type of autoclave and the size of the load. They apply to a sterilizing temperature of 121ºC.
Empty glassware and unwrapped instruments. Sterilizing time 15mins, drying 10mins.

Wrapped instruments, rubber gloves, tubes and catheters, and sutures being re-autoclaved. A common regime is 0·7kg/cm² (10psi) for 20mins.

Fabrics and dressings. Sterilizing time 20mins, drying time 15mins.

Liquids in flasks and bottles.

Sterilize bottles according to size and time as follows:

mls	100	300	500	1000	3000
mins	20	30	35	40	50

Switch off the heat and let the autoclave cool down. *Don't open it until the pressure is zero*, as the bottles may burst.

(7) **Preventive maintenance**

Follow the maker's instructions carefully. *Don't miss out on this for reasons of false economy or 'permanent' need!*

Make sure you make periodic checks by taking swabs for microscopy and culture. This is vital: <10% of hospital sterilizers work properly in LMICs.

(8) **Difficulties with downward displacement autoclaves**

If the temperature falls <121ºC, while the pressure remains at 1 kg/cm² (15psi), the outlet from the chamber may be blocked, and the chamber full of air. Check it daily.

If you work at high altitudes, for each 300m (1000 feet) you are above sea level, increase the time you immerse things in boiling water by 5mins, and increase the pressure of your autoclave by 0·03 kg/cm² (½psi). Water above 80ºC will kill all vegetative organisms and viruses; boiling water is still effective at 4,000m (13,200 feet).

If dressings are wet after autoclaving, the steam is probably wet, due to: (1) inadequate lagging of the steam supply pipe, or (2) inadequate tapping of condensate.

If you have reason to suspect imperfect sterilization, run the tests above. Also check that:

(1) The drums are packed properly.
(2) The correct temperature and sterilizing times are used.
(3) The chamber drain is not blocked.
(4) The drums are not being re-contaminated after sterilization.

(b) Sterilizing with dry heat

Use this for laboratory items, knives, and drills which don't tolerate steam well. You need a higher temperature (160ºC) for 1h. Although heat is the best way of killing micro-organisms, *it is not appropriate for delicate instruments, rubber or a person's skin*. *Heat also destroys a cutting edge*, so store your scissors in a chemical solution which will destroy bacteria. Classically, these chemicals are either antiseptics, which are safe to use on the surfaces of the body, or disinfectants, which are not. In practice, the distinction is not precise, but the only substances which you cannot apply to the body are: (1) saponated cresol ('Lysol'), (2) formalin, and (3) glutaraldehyde. There is an optimum antiseptic for each purpose, so try to use the right one.

Disinfectants have serious limitations and only work when the object they are disinfecting is clean: they are ineffective in the presence of blood or pus. So wash scissors and fine instruments carefully before you store them in an antiseptic solution.

If possible, drains and other heavily contaminated pieces of equipment should be boiled or autoclaved after washing and before being immersed in these solutions. Afterwards, wash them well in sterile water before you use them.

N.B. Catheters and tubes etc. deteriorate in antiseptic solutions and are better autoclaved before use.

Avoid cetrimide: it is mainly a detergent; chlorhexidine is better.

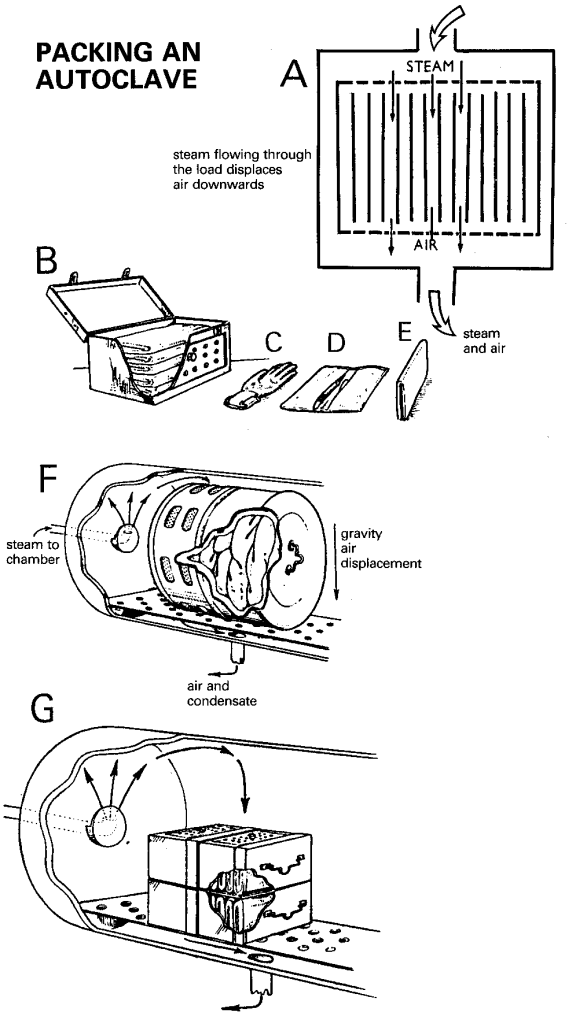


Fig. 2-8 PACKING AN AUTOCLAVE.
A, orientate a load to facilitate the escape of air in a gravity displacement sterilizer. Steam enters from the top, flows downwards through the load and displaces the air in it. B, pack a glove container properly. C, folded glove lined with gauze. D, a pair of gloves packed in a fabric envelope. E, fabric envelope on edge to show its correct position during sterilization. F, pack the drum correctly with open ports positioned to allow air to be displaced by gravity. G, turn glove containers in an autoclave on edge so that steam can displace air through them.
After Sterilisation by steam under increased pressure; a report to the Medical Research Council by the Working Party on Pressure-Steam Sterilisers. Lancet 1959;7070:425-35, with kind permission.

2.5 Disinfectants and antiseptics

(a) Skin. Any alcoholic solution will do. Alcoholic iodine is best: use it routinely, except in children, on the scrotum, and in allergic patients. Chlorhexidine 0·5% in spirit is a less satisfactory alternative. Apply it to the skin after removing all traces of soap.

(b) Wounds. There is no substitute for a scrubbing brush, plenty of water from a jug, and a thorough surgical toilet. Chlorhexidine is useful for cleaning the skin round a wound.

(c) Instruments, suture materials, and drains
The following agents are effective against HIV and HBV, in addition to the classical pathogens (5.4).
(1) 0·55% orthophthalaldehyde.
(2) 2% alkaline buffered glutaraldehyde.
(3) 8% formalin in 70% spirit or as a tablet.
(4) A 0·5% solution of chlorhexidine in 70% spirit with 0·5% sodium nitrite. (*This is in terms of the active agent.*)
(5) Plain 70% spirit.
The first two, 'Cidex OPA' and 'Cidex', are the best; glutaraldehyde needs to be activated before use but orthophthalaldehyde does not.
10mins is the absolute minimum time in these solutions, provided instruments are scrupulously clean, 24h is safer. Ideally, nothing should be considered 'sterilized' until it has been immersed for 24h. Wash all equipment well before using it.
CAUTION!
(1) Except for glutaraldehyde (which can be used for 14-28 days depending on the brand), you must prepare these solutions freshly every week, and keep them covered to prevent the alcohol evaporating.
(2) *A 'wipe' is not nearly as good as a soak!*
N.B. Formalin tablets can be vaporized in special chambers and used to sterilize endoscopes over 12h. It is irritant to the eyes and nose, and toxic to the tissues. It is useful for fumigating the theatre after a septic procedure.

(d) Furniture, doors, windows and other fixtures
5% phenol (carbolic acid) is a satisfactory cleaning agent; you can use a 10% solution for very soiled surfaces.

2.6 Antiseptic surgery

This used to be standard practice before aseptic methods made it more or less obsolete. But it may still be useful when power supplies have failed or your autoclave breaks, or an important operation has to be done in some remote place. *It has been said that a first-class surgeon can operate in any theatre in any clothes in any situation.* However, even if you are not an expert, *don't deny someone life-saving surgery if your autoclave has stopped functioning!*

Aim to sterilize everything coming into contact with the wound by soaking it for a sufficient time in an antiseptic solution.

Unfortunately:
(1) An antiseptic solution leaves everything wet.
(2) Sterilization is slow so that you may only be able to do one operation at a time.
(3) Wide areas of the body are exposed to the antiseptic, which causes much exudation from the wound.

Even so, antiseptic surgery is simple, and makes many kinds of operation possible. If necessary, you can combine antiseptic and aseptic methods, and sterilize smaller instruments in a pressure cooker. Chlorhexidine is the most practical antiseptic, but is far from perfect.

ANTISEPTIC SURGERY UNDER ADVERSE CONDITIONS. "The only means of access to our hospital at present is by walking over the mountains for a week. All supplies have to be carried in by porters who take two weeks for the journey. For the first 2½yrs, we worked in a traditional Nepali house with a thatched roof and a floor made of mud and cow dung. In it we did over 100 operations by the antiseptic method, without serious mishap. Later, limited space became available, so that although we enjoyed the advantages of tap water, a concrete floor, a clean ceiling, and adequate window ventilation, we still had to operate on a light outpatient type of table and in the same room in which the outpatients received all their medicines, injections, dressings, incisions, and dental extractions. We almost always used epidural or local anaesthesia". *Dick JF, Surgery under adverse conditions, Lancet 1966;7469:900.*

(a) Antiseptic solutions
Use chlorhexidine 5% concentrate to make two solutions:
(1) A weak solution of 1/2000 of the active agent in water. Use this for soaking towels, etc.
(2) A strong solution for instruments, as described (2.5). Make up small quantities of solutions frequently, make them up hot, and clean out the containers well between batches.

(b) Sterilizing equipment and drapes
Soak everything which will come into contact with the wound in one of these solutions for at least 30mins. Soak sutures and gloves in this solution overnight. Use monofilament (4.6) for ligatures and sutures, and the minimum number of simple instruments.
The most appropriate drape, for a tubal ligation, for example, may be a single solution soaked plastic sheet long enough, and wide enough, to cover the whole patient, with a hole in the middle through which to operate. If you have 2 such drapes, one can be in use while the other is being soaked in a flat container of solution.
CAUTION! Don't use syringes and needles soaked in antiseptic to give a subarachnoid or epidural anaesthetic.

(c) While operating, treat the patient's skin with the solution for at least 2mins before the operation.

Wash your hands as usual and put on the wet gloves. Wring out the soaked drapes as dry as you can, and apply them as near as possible to the operation site. Clean the patient's skin with the same solution.

If there is a danger that he might get cold, cover him with a dry blanket in a plastic sheet, and put this between the skin and the wet towels above and below the operation site, where it will not get in the way.

Swab the trolley with the solution, or put the instruments on a solution-soaked towel. Keep 2 bowls near the operating table, one containing water and the other antiseptic solution.

When instruments have been used, wash them in water and keep them in the solution until you use them again. Shake off the excess solution before you use them. Handle the tissues as little as you can, and try to keep the solution out of the wound as much as possible.
Don't let cleaning solution get into the body cavities.

(d) After operating, rinse everything free of blood. Rinse the instruments, and put them away. If the wound is well sutured and is not expected to discharge, leave it open to the air.

2.7 Antibiotics in surgery

Antibiotics have 2 uses in surgery:
(1) To treat invasive sepsis.
(2) In certain circumstances only, and when used in a very particular way, as prophylaxis to prevent post-operative infection.

They are less important than:
(1) Careful aseptic theatre routines.
(2) A thorough wound toilet.
(3) Delayed primary closure.
(4) Making sure there are no foreign bodies, dead tissue, excessive blood clots, or faeces in the wound.

In preventing sepsis, *antibiotics give you no licence to neglect the classical rules of good surgery*, especially if the patient is diabetic, very old, has HIV and is very ill, and so is less able to overcome any bacteria that cause infection.

Antibiotics will represent a very large part of your pharmacy's budget, so use them wisely *and not indiscriminately.* Generally speaking, antibiotics are prescribed far too often, far too long, and with far too little thought.

So:
(1) Handle the tissues gently; take care to avoid spillage and contamination of the wound.
(2) *Don't leave large pieces of dead tissue in the wound*, such as huge, massively ligated pedicles, or with excessive use of diathermy.
(3) *Don't put tissues or skin under tension*.
(4) Make sure there is secure haemostasis.
(5) Divert faeces if they risk contaminating a wound, by temporary colostomy.

Differentiate between *prophylactic* use of antibiotics (2.9) and the treatment of *invasive sepsis* (*e.g.* cellulitis, septicaemia).

For prophylaxis use a *single dose* of antibiotic: this is indicated in 'clean, contaminated' (category 2) cases such as hysterectomy, Caesarean section, appendicectomy, and cholecystectomy.

N.B. Clean uncontaminated wounds (category 1) don't benefit from antibiotic prophylaxis. Even with immunocompromised patients you should not change this principle.

That said, how can you use antibiotics for invasive sepsis to the best advantage, when your laboratory staff cannot culture bacteria, or at least not reliably? You can learn much, however, from a simple Gram stain. Nonetheless, encourage the laboratory to examine blood cultures, which are not difficult technically, and, when these are positive, to isolate the organism responsible for septicaemia in pure culture.

If you are fortunate, you will be able to plan a logical antibiotic policy for your district, and keep some antibiotics for hospital use only, in the hope that the arrival of antibiotic-resistant strains from elsewhere in the world will be delayed as long as possible. In such an ideal situation you might decide, for example, that the clinics should use only penicillin and tetracycline, with perhaps a little ampicillin or trimethoprim; keep streptomycin for tuberculosis only. This will enable you to use chloramphenicol with metronidazole as your main surgical antibiotics, especially when the gut and the genital tract are involved. For other occasions you can use gentamicin, or a cefalosporin.

Unfortunately, you are more likely to work in a situation of antibiotic chaos, in which any antibiotic is obtainable over the counter without prescription, and where multiply resistant strains, particularly those resistant to chloramphenicol, are common. *Be sure to find out what are the sensitivities and so the antibiotics of choice for your area*. You should get a good idea of which antibiotic, out of those generally available, to use for which situation. You may have donations of expensive newer antibiotics: *don't waste them through ignorance of their benefits!*

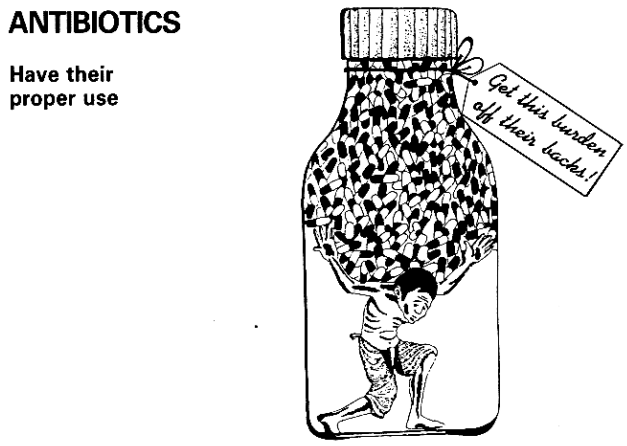


Fig. 2-9 ANTIBIOTICS MUST GET TO THE PATIENTS AND THE DISEASES WHERE THEY CAN DO MOST GOOD.
A poster from Oxfam's 'Rational Health Campaign' to show the enormous burden many communities bear in misused antibiotics that are bought in the market-place, or are prescribed by doctors on the wrong indications for the wrong patients. *Kindly contributed by Oxfam.*

2.8 Particular antibiotics

Some antibiotics are particularly important in district hospital surgery, either because they are life-saving, or because they are good value for money. *Don't, however, overuse them, particularly when there is no clear indication to do so!*

(a) Penicillins

Benzylpenicillin (penicillin G) is cheap and safe. For *streptococci and meningococci*, it is the antibiotic of choice. There is little point in giving very high doses. If penicillin fails to cure a patient, this will probably be because the β-lactamase of penicillin-resistant bacteria is destroying it, not because you are not giving enough. For an adult, 1·2g (2MU) qds is the standard dose for a severe infection, such as spreading hand sepsis, cellulitis round an infected wound, gas gangrene (6.24) and tetanus. It is also effective against anthrax, *borrelia*, diphtheria, gonorrhoea, and leptospirosis. However, if drugs are scarce, 0·6g given to 4 people is likely to do more good than 2·4MU given to 1 person. In infants, and in patients with cardiac or renal disease, the sodium or potassium in the penicillin can cause undesirable side-effects, so be aware of this.

Benzathine penicillin, or **ultracillin** (1·4G), is used in venereal disease (syphilis, yaws, bejel, pinta and chancroid) and anthrax, but not acute surgical infections. Its use is in prophylaxis in rheumatic fever, and after splenectomy.

Procainbenzylpenicillin (3G) may be used as a once daily dosage instead of benzylpenicillin, particularly in children.

Flucloxacillin or **cloxacillin** (500mg qds) are not inactivated by penicillinases and so are very useful against most *staphylococci* which are now generally resistant to benzyl- or phenoxymethyl-penicillin (penicillin V).

Ampicillin (250-500mg qds) and **amoxicillin** (250mg tds) are inactivated by penicillinases and so ineffective against *staphylococci* and common Gram-negative organisms such as *E. coli.*; they are useful against chest infections and otitis media caused by *Haem. influenzae* and *Streptococcus*, as well as endocarditis prophylaxis, but less so against urinary infections. The combination with clavulanic acid, **co-amoxiclav**, is effective against β-lactamase producing bacteria, and so has a broader spectrum. Amoxicillin is better absorbed orally than ampicillin.
(Use **ticarcillin** and **piperacillin** against *Pseudomonas septicaemia.*)

(b) Macrolides

Erythromycin (500 mg qds) is the standard alternative where there is penicillin allergy. It is the drug of choice for *mycoplasma* pneumonia, Legionnaire's disease, and chlamydial infections. It has a useful secondary effect of stimulating gastric emptying.

The others – **azithromycin**, **clarithromycin**, **roxithromycin –** have slightly better activity against Gram-ve organisms, but are expensive.

Metronidazole (400mg tds) is effective against anaerobes (which far exceed aerobes in the gut, and are the cause of foul faecal odour), especially *Bacteroides fragilis*, and *protozoa*. It is the drug of choice for amoebiasis, balanitis, giardiasis, Guinea worm infection, tetanus, and trichomonal vaginitis. Resistance to it is unknown. Alcohol should not be taken with it but otherwise it has few side-effects. Use it, blindly if necessary, for all patients who are severely ill with an infection that might be caused by anaerobes, and particularly for patients with intra-abdominal sepsis. Intravenous metronidazole (500mg tds) is expensive, but you can achieve adequate blood levels by using suppositories, or as oral tablets inserted rectally. Like this, it is only $^{1}/_{10}$ the price. Metronidazole is one of the drugs that no surgeon should be without. **Ornidazole** and **tinidazole** are similar.

Chloramphenicol (500-1000mg qds) is cheap, and has a broad spectrum of activity against aerobic Gram-ve bacilli and Gram+ve cocci. Also, if you do not have metronidazole for anaerobic infections, chloramphenicol is next best. It has good *in vitro* activity against anaerobes from most parts of the world. It also enters the eye (28.3). *Its life-saving properties outweigh the very small risk of aplastic anaemia*. It is the drug of choice in bubonic plague. *You cannot administer it IM.* Chloramphenicol with metronidazole is an excellent combination for established or expected peritonitis (10.1). However, resistance will be common if the drug is much used in the community. **Thiamphenicol** is similar. They enhance anticoagulants, anticonvulsants and the sulphonylurea hypoglycaemics (glibenclamide etc.).

(c) Cefalosporins

There are 4 'generations' of these drugs with increasing spectrum *and* cost:
1st: **Cefradine** (250-500mg qds),
cefazolin (500mg qds), **cefalexin** (250mg qds),
cefadroxil (500mg bd)
2nd: **Cefaclor** (250mg tds), **cefprozil** (500mg od),
cefuroxime (750mg tds),
cefamandole (500mg qds)
(less inactivated by β-lactamases than 1st generation, so cover some Gram+ve bacteria)
3rd: **Cefotaxime** (1g bd), **ceftazidime** (1g tds),
ceftriaxone (1g od), **cefsulodin** (1g bd)
(broader spectrum, but less good against Gram+ve bacteria than 2nd generation)
4th: **Cefoxitin** (active against bowel flora).
They are useful to treat severe Gram-ve infection, and with metronidazole as prophylaxis in bowel surgery.
Remember that 10% of penicillin-sensitive patients are also allergic to cefalosporins, especially if they have had an immediate reaction to one or the other.
Beware! Alas, more and more Gram-ve organisms are becoming resistant to cefalosporins!

(d) Aminoglycosides

Gentamicin (80mg tds, or 240mg od) is a very valuable broad-spectrum antibiotic, used IV or IM, often effective against *Pseudomonas*. For the 'blind' treatment of a serious infection, especially one due to intestinal bacteria, use gentamicin and ampicillin or penicillin with metronidazole. Gentamicin is toxic to the ears and kidneys if its use is prolonged; *don't use it at the same time as the diuretic furosemide.*

Other costlier similar aminoglycosides are **amikacin**, **kanamycin**, **netilmicin** and **tobramycin**; you can use **neomycin** orally but it is too toxic; use **spectinomycin** against gonorrhoea; reserve **streptomycin** for *tuberculosis* treatment; use **spiramycin** against *toxoplasmosis*.

(e) Sulphonamides

Trimethoprim (200mg bd) alone is preferable to cotrimoxazole, which is a combination of trimethoprim and sulfamethoxazole. The latter is rather toxic and not very effective. Sulphur sensitivity is common with HIV disease, and the resulting Stevens-Johnson syndrome is often fatal. Trimethoprim is also used for *pneumocystis*, *toxoplasma*, and *isospora.*

(f) Tetracyclines

Tetracyclines have broad spectrum activity, but bacterial resistance is a problem. They are the drug of choice in *chlamydia* infections (donovanosis, trachoma, salpingitis, urethritis, LGV), *rickettsia* (tick typhus), *treponema* (syphilis) and *brucella.* They also protect against malaria. They are deposited in growing bone and teeth, so *don't use them in children <12yrs, or pregnant and breastfeeding women*. Absorption of **doxycycline** (100mg bd), unlike **tetracycline** (250mg qds), is not decreased in effect by milk, antacids or calcium, iron and magnesium salts, and is safe in renal disease.

(g) Quinolones

Ciprofloxacin (500mg bd) is active against Gram-ve and +ve bacteria (but not usually *Strep pneumoniae* and *Enterococcus faecalis*) and is particularly active against *salmonella*, *shigella*, *campylobacter*, *Neisseria*, *pseudomonas*, and *chlamydia.*

Nalidixic acid (1g qds), **norfloxacin** (400mg bd), **ofloxacin**, **enoxacin**, **cinoxacin**, **pefloxacin**, and **sparfloxacin** are useful in urinary tract infections.
Don't use them in epileptics, for children, in pregnancy, and breastfeeding. They enhance the effect of anticoagulants.

(h) Others

Fusidic acid (500mg tds) should be specifically reserved for penicillin-resistant *staphylococcal* osteomyelitis; as a cream (2%), it is useful for impetigo, but should not be used for simple skin ulcers, because of the problem of resistance.

Nitrofurantoin (50m qds) is useful in uncomplicated urinary tract infection.
Mupirocin (2% cream) is also useful in impetigo and secondarily *staphylococcal* infected fungal skin infections.
Pivmecillinam is active against many Gram-negative bacteria, but not *Pseudomonas.*
Aztreonam (not active against Gram+ve), **imipenem** with **cilastin**, **meropenem**, and **moxalactam** are powerful broad-spectrum β-lactam antibiotics.
Clindamycin is useful against *staphylococci* and many anaerobes, but can produce fatal pseudomembranous colitis.
Vancomycin and **teicoplanin** are used against multi-resistant *staphylococci,* and *clostridium difficile*.

2.9 Methods for using antibiotics

Antibiotics for treating established infection call for little comment, and are described in many places in this manual. Antibiotics to prevent infection need to be used wisely, in ways in which their benefits outweigh their risks.

An operation site which was clean to start with can become contaminated with bacteria from:

(1) **Outside the patient**, in which case they will probably be *staphylococci*. Preventing such infection is the purpose of the ordinary aseptic routines, and prophylactic antibiotics are no substitute for them. Most surgical patients don't need antibiotic cover for sepsis of this kind. The only absolute indication for it is to cover the implantation of prostheses, which you are unlikely to do.

(2) **Inside the patient**, when you operate on the colon or the lower urinary tract, or on a woman's genital tract.
When you use antibiotics prophylactically, aim to provide a concentration in the blood that will kill any bacteria introduced into the wound at the time of the operation. To minimize the risk of peritonitis, it is important to protect against *enterobacteria* (mostly *E. coli*), as well as aerobic and anaerobic *streptococci*, *bacterioides*, and *clostridia*. A single broad-spectrum antibiotic with good tissue penetration and long half-life is ideal.
Use the antibiotics IV pre-operatively (especially with the premedication or the start of surgery), so that high concentrations are reached in the wound at the time of surgery. Starting them a day or more before the operation, or continuing them unnecessarily afterwards, promotes the selection of resistant organisms and the risk of side-effects, and *has been shown to confer no extra benefit*.

If you forgot to give the antibiotic before the operation, it is still worthwhile to do so before closing the skin, *but not afterwards. (That would be like washing your dirty hands after eating a meal!)*
There are several unacceptable methods:
(1) *Don't put topical antibiotics into a patient's wound.*
(2) *Don't use them in the hope of 'sterilizing the colon'.*
(3) *Don't use antibiotics for longer than a specified period* in the vain hope that infection or fever might finally be controlled.

As to the antibiotics to use, you will see from the list of indications below that, if chloramphenicol is not much used in the community, chloramphenicol with metronidazole is likely to be the most cost-effective combination. Otherwise, use cefradine (or some other cefalosporin) with metronidazole, which are much better than penicillin and streptomycin.

Always differentiate prophylaxis from treatment. Using your more expensive antibiotics in life-threatening sepsis makes more sense than wasting them in dubious prophylaxis. If you are treating septicaemia, aim to continue the antibiotic regime until the illness is under control (usually 5-7 days). Once a patient can take drugs orally, there is usually no longer any need to give them IV.

THE DOSE AND THE TIMING ARE CRITICAL: MAKE SURE THERE ARE ADEQUATE LEVELS AT THE TIME OF SURGERY

Perioperative prophylaxis

INDICATIONS

(1) Peritonitis (but antibiotic use here is likely to be therapeutic rather than prophylactic)
(2) Operations likely to contaminate the peritoneal cavity, especially with spillage from the colon, appendix, bile duct or stomach.
(3) Operations on the urinary tract when the urine is already contaminated, including bouginage, cystoscopy, and prostatectomy.
(4) Hysterectomy.
(5) Emergency Caesarean section.
(6) Intracranial explorations.
(7) Open fracture surgery, and amputations.
(8) Re-opening haematomas.
(9) Splenectomy.
(10) Dental or oral surgery with known heart valve disease.

CAUTION!

Gentamicin and other aminoglycosides may seriously prolong the action of long-acting (non-depolarizing) relaxants, and may prevent the establishment of spontaneous ventilation. Avoid them unless your anaesthetist is experienced.

N.B. Prophylactic antibiotics will probably not *cover the perioperative risk of respiratory infections*. Physiotherapy is far more likely to be effective, both pre- and post-operatively.

CONTRAINDICATIONS. Antibiotics are not needed for:

(1) Already well-localized infections.
(2) Clean category 1 operations (hernia repair, ovarian cystectomy, etc)
(3) Burns (initial treatment)
(4) Tracheostomy, intercostal drainage, simple lacerations.

If you are using a tourniquet, time the injection to provide the maximum concentration about the time that you release it, so that the clot which forms in the wound will be heavily loaded with drug.

ONLY A FEW HIGH-RISK PATIENTS NEED PROPHYLACTIC ANTIBIOTICS

"We may look back on the antibiotic era as a passing phase, an age in which a great natural resource was squandered."

2.10 When prevention fails: wound infection

If a wound discharges pus, this may mean that the aseptic routines described earlier in this chapter have broken down. Although this is not the only cause of a wound infection, it is the most unnecessary one.

Keep a record of your wound infections. They are most likely to occur if:

(1) You are operating for some infective condition, such as acute appendicitis.
(2) The operation is long and difficult.
(3) You leave dead tissues, foreign bodies, dirt, or clot, or an excessive number of sutures (especially non-absorbable) in the wound.
(4) You create dead tissue by operating clumsily.
(5) You do an unnecessary un-clean procedure at the same time as the clean surgery.
(6) You close a wound by immediate primary closure, when delayed primary closure would have been wiser.
(7) You leave IV cannulae, chest drains or other drains in longer than necessary.

SURGICAL SEPSIS

(1) A theatre had extractor fans installed, but the only inlets for fresh air were under the doors, so that dust from the corridor was drawn into the theatre continually. Only when three patients had died of tetanus was the flow of the fans reversed.

LESSON Keep dust out of the theatre.

(2) In a certain teaching hospital, there were two minor theatres in which many septic operations were done. On 2 mornings a week the same equipment was used for a list of circumcisions. One circumcised child acquired erysipelas which spread from the umbilicus to the toes and killed him.

LESSON Where possible, *don't do clean cases in a theatre which normally does septic ones*.

(3) An eminent professor electively resected an appendix at the same time as cholecystectomy. The patient developed an anaerobic wound infection and later a faecal fistula.

LESSON *Don't do unnecessary procedures which increase the risk of infection*.

(4) Hamilton Bailey, subsequently a distinguished surgeon, but then a registrar in the 1930s, was deputizing for the chief. Having done an elective list which began at 1.30p.m., he insisted on continuing with a non-stop flood of emergencies which continued rolling in all evening. At 3 a.m. the following morning, 'dead on the feet', he pricked himself when operating on a patient with streptococcal peritonitis. Bailey insisted that the finger be amputated, and survived. The patient died.

LESSON Accidents, including those which increase the risk of sepsis, hepatitis and HIV transmission, are particularly likely if you are overtired.

If >5% of your clean cases become infected, something has gone wrong. Prophylactic antibiotics are not the answer! *The chances are that the aseptic technique (2.3) is not being followed, or you are making the errors 3, 4, and 5 above*.

(a) The prevention of wound infections
AUTOCLAVING
(1) Check that your autoclave does reach 1 kg/cm^2 (2.4), that the air is being discharged, and that the holding time is being maintained.
(2) Check that the drums are not being overpacked, that they are labelled after autoclaving, and that the label includes the date.

THEATRE DISCIPLINE. Check that you and *all* your staff are following all the aseptic disciplines (2.3) carefully. If you set an example, your staff will follow. Check that:
(1) the theatre table and especially the plastic cover on its mattress, are being properly cleaned,
(2) there is no infected member of staff: check for nasal and skin carriers of *staphylococcus* especially if an outbreak of hospital infections occurs.
Examine yourself. Are you committing errors 3, 4, or 5 above?

(b) The treatment of wound infections
Sedate the patient with morphine, pethidine, diazepam or ketamine, if necessary. In infected sutured wounds the pus usually tracks the whole length of the subcutaneous tissues. So remove all sutures and convert the wound into an open gutter. If possible, send a swab for culture. Clean the wound; use hydrogen peroxide if it is smelly. Establish free drainage, especially in the depths of the wound, keep it open so that it can heal from the bottom, and pack the wound daily with antiseptic dressings. Either allow it to granulate or close it by secondary suture when it is 100% clean. If sepsis is troublesome, consider the use of pure ghee (the clear liquid skimmed off the top of slowly heated butter) and pure honey in a ratio 1:2, sugar, pawpaw, or even sterile maggots. Use low-suction vacuum dressings, connected to collapsed urine bags or similar, if you do not have suction devices available.

Antibiotics are only indicated if there is spreading infection (cellulitis) or septicaemia. There is no role for topical antibiotics. If you have many septic wounds to deal with, or not enough staff or dressing materials, leave the wounds open and exposed to the sun for as long as possible. Check that there is no indiscriminate or undisciplined use of antibiotics.

If there is oedema and a brownish discharge comes from the wound, and the patient is toxic and apathetic, suspect *gas gangrene* (6.24); **if there are spreading purplish discolouration and signs of subcutaneous necrosis**, suspect *necrotizing fasciitis* (6.23). In both cases, immediate extensive debridement is necessary to save life.

If a wound fails to heal, think of diabetes mellitus, HIV (5.6), anaemia, malnutrition, the presence of cancer or a foreign body.

If a sinus develops from a wound, suspect an infected buried non-absorbable suture knot (a *stitch sinus*); sterilize a crochet needle and use this to try to hook the knot out of the wound.

If you are successful, the wound will heal spontaneously. Otherwise, you will have to open around the sinus and extract the foreign material.

If a growth develops from the wound, this is a *pyogenic granuloma* (34.4): excise it and check for HIV disease.

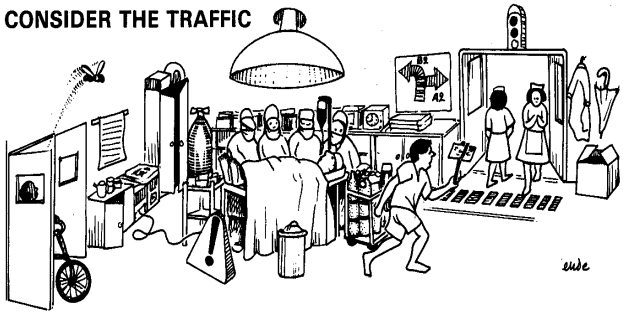


Fig. 2-10 CONSIDER THE TRAFFIC.
Wounds are less likely to become infected if the theatre is not used as a storeroom, and if there is a minimum of traffic in and out of it. So remove the teacups and cartons, the bicycle, the umbrella, and that coat! Close the doors! *Drawn by Nette de Glanville.*

2.11 Post-operative pain control

Your reputation will grow enormously if your patients don't suffer any discomfort after surgery; unfortunately much good pain management is hindered by myths, fear or ignorance. Unrelieved pain has significant effects on a patient's physiology as well as psychology. Pain scoring systems are very useful in establishing an objective measurement of analgesia:

VERBAL: NONE-MILD-MODERATE-SEVERE-EXTREME

NUMERICAL: INTENSITY 0 (no pain) – 10 (worst pain)

VISUAL: INTENSITY LINE ☻ (no pain) – † (want to die)

N.B. The intensity of pain is what the patient says it is!

The visual system is most useful in children. Since many patients after major surgery cannot speak well, you should have these scoring charts on a board ready to show them.

Don't ignore the patient who complains of pain: it may be a sign of a serious complication.

The aim should be to *prevent* pain: a patient should wake up after surgery with no pain, and be encouraged to ask for analgesia as soon as pain develops.

Combinations of analgesic drugs and of routes of delivery give the best results. You can provide much pain relief by putting large volumes of low concentration long-acting local anaesthetic (bupivacaine) into the wound at the end of the operation; *don't inject it into the surrounding tissues if the wound is infected*: you can then just drip it into the wound and leave it for 1min to get absorbed.

The sad reality is that in present practice many patients wake up with pain, shout for help and are shouted at in turn, until eventually, they are given a large IM dose of opioid. They then go to sleep again. Later, when the analgesic effect wears off, the cycle repeats itself. This is not only unsatisfactory from the point of view of needless suffering but is often the cause of post-operative complications: atelectasis, deep vein thrombosis, vomiting, anorexia, constipation, dehydration, urinary retention, and it also prevents people from getting out of bed.

(a) For severe pain, morphine is preferable to pethidine, because it produces less respiratory depression, less nausea, and is less of a cerebral irritant. It also lasts longer. (Pethidine needs to be repeatedly given 3hrly to be effective.)

Because these are controlled drugs, nurses will often only give them at standard drug dosage times. Challenge your local regulations if these inhibit patients getting proper pain relief. Try to get solutions of oral morphine made locally. *This should not cost >1c.(US)/mg! Don't use injectable opioids SC or IM* but always IV, injecting slowly: this way relief will be immediate and the dose received will be less. Small, frequent IV opioids will prevent pain and it will be possible to switch to the oral or rectal route within 24h in most cases. Apart from being much more effective if given IV, either as boluses or better as an IV infusion, they are safer given this way as you thereby *must watch the patient's response.*

A calculated IV infusion of opioid is not dangerous! (If the IV infusion has accidentally run in fast with all of its added 10mg of morphine, simply omit the dose with the next litre of IV fluid.) Arrange the infusion in theatre with the cooperation of the anaesthetist.
In children, tilidine oral drops (x1 per year of age up to 10) are very useful indeed.

Ketamine gives good post-operative pain relief; its hallucinatory effects are diminished by giving diazepam *before* the operation, *i.e.* with the ketamine.

Remember that opioids occasionally cause hyperalgesia (especially if used for non-malignant causes); but beware of the patient with chronic pain who regularly refuses opioids (*he probably needs them*) and the patient with aberrant behaviour who demands them (*he does not need them*)!

(b) For moderate pain, the choice is paracetamol-with-codeine and/or a non-steroidal anti-inflammatory drug. The latter have considerable side-effects: peptic ulceration, renal impairment, and coagulation problems. They can be given rectally if a patient is not taking in orally. The evidence that they are any more effective than paracetamol-with-codeine is not convincing, but *it is always best to ask the patient which drug he finds best!*

If you know that an operation will give considerable pain, prescribe *regular* analgesia for the first 2-3 days, not 'PRN' (which stands for *pro re nata* = as required, but often in practice implies 'presumably rarely needed')!

(c) For mild pain, paracetamol is ideal. It can be given as a syrup for children or those who have difficulty swallowing.

Trans-cutaneous electrical nerve stimulation and neuro-acupuncture can give added relief if you have these facilities.

2.12 Records

Keep meticulous records of operations performed: train the theatre staff to fill in the book immediately and keep these records accurately. Bad records are almost as good as no records at all! You should have *all* the following information in the theatre book (which obviously should be fairly large, and preferably hard-backed):

DATE
OPERATION NUMBER
PATIENT'S NAME
PATIENT'S AGE/SEX
PATIENT'S HOSPITAL NUMBER
DIAGNOSIS
OPERATION PERFORMED
EMERGENCY/ELECTIVE
SURGEON
ASSISTANT(S)
ANAESTHETIST
ANAESTHETIC USED
SCRUB SISTER
TIME STARTED & TIME FINISHED
COMPLICATIONS
HISTOLOGY/PUS SWAB RESULT

Keep your book neat: if necessary, fill in details initially in pencil. Keep to the columns drawn in the book.

It is important to use the same nomenclature throughout, *e.g.* 12 Feb 2004 for the date (and then not use 12/02/04 or, worse, 02/12/04), and particularly consistency in abbreviations (*e.g.* I&D for incision and drainage, MUA for manipulation under anaesthetic etc). Try to keep names consistent, using the family name first in CAPITALS and then the first (and second) names. The more detail you can put, the better will be your records, and your ability to do research.

Under 'Diagnosis', be sure to put the correct diagnosis (which may differ from the pre-operative diagnosis).

Under 'Anaesthetic Used', you should put *at least* GA for general anaesthetic, Sedation, or LA for local anaesthetic. You could put Thio/O_2/N_2O if using thiopentone, oxygen and nitrous oxide, or Ket if using ketamine, but the more detail in the records, the more diligence is required in keeping them. Often there are *no records at all*, which is a disastrous and unacceptable state of affairs.

You should come back and check the theatre records, in case details are filled in incorrectly. Get your nurses to write details in pencil for you to correct, if necessary, later. Make a particular note of complications. This includes not only immediate problems (like bleeding or a death on the table), but also later ones such as wound infections.

If you direct laboratory results of histology and pus swabs to theatre so that they are recorded there in the book, they are much less likely to get lost and can be much more easily referred to.

Some details are optional, *e.g.* indication for operation, grade of operation (minor, intermediate, or major), and type of procedure (endoscopy, orthopaedic, ENT etc). The grade of operation is notoriously subjective; if you do record this, we suggest using the grades described in Appendix A of this manual.

You should keep a separate book for deliveries of babies, and decide whether you should enter operative deliveries with the other operations, or separately. *It doesn't matter as long as they are properly recorded!*

If you keep good records, you will be able to highlight problems when things go wrong. You can keep an audit on how much work you are doing, what your requirements are likely to be, and therefore your costs. You will have a valuable resource for research. This is very important. You will also derive satisfaction from a job well done, and leave a functioning system in place for your successor.

3 The control of bleeding

3.1 Assisting natural mechanisms

Most surgical intervention will result in some sort of bleeding. This can also happen from an injury. The body has excellent mechanisms for controlling bleeding, so that your task is mostly supportive. The main mechanisms are the cascade of enzymatic reactions which make the blood clot, and the ability of the muscular walls of the arteries to contract.

If you fail to control bleeding adequately a patient may die, so take note of the amount of blood he loses. The loss of a given volume of blood is more serious in a child (3-1) and much more serious in a baby, than it is in a fit adult, who can usually lose 1L without the need to replace it by blood. A loss of >20% of the blood volume is critical: a child has a total of 75ml/kg of blood.

The most generally useful way of controlling bleeding is pressure, but there are also special methods for particular parts of the body, such as the scalp and the dura, the bowel (11.3) and the liver.

Most importantly, *don't panic!* Have a plan of action, starting with the simplest methods, and if these don't work, progress to more complicated techniques. Stop to reassess the situation: *don't fumble around;* you will lose valuable time and achieve nothing! *Don't try to get definitive control of bleeding from the outset:* aim for temporary control initially. This should be quick, effective and not cause more damage to the patient.

These are the methods you can use:
(a) Pressure is the simplest and most valuable way to control bleeding. When you press on tissue, the walls of its vessels come together, and where their edges are cut, thrombus will start to form. When you release the pressure you will probably find that bleeding has stopped, or that only the arteries will continue to spurt at you, and these you can tie off. Press with a gauze pack. If pressure is to succeed, you must press for long enough: this is normally at least 5mins by the clock, which is one reason why every theatre should have a clock. If the tissue behind the bleeding area is firm, as when you press a bleeding scalp against the skull, pressure is even more effective. Likewise a finger in a groin wound, pressing against the hip joint, is extremely effective. For bleeding from the finger, *don't try anything else!*

Note that putting on more and more dressings (so increasing the applied area) dissipates the pressure (which is force per unit area), so their effectiveness is reduced. If a wound dressing is soaked, remove it, and apply pressure directly to the bleeding point.

A pressure dressing is only really effective on the head, hand or foot; otherwise on a limb it acts as a venous tourniquet, which may increase bleeding! On the chest it will interfere with respiration, and it is useless on the outside of the abdomen.

Inside the abdomen, remember that pinching the base of the mesentery between the fingers of one hand will occlude its blood supply, so you can buy time if there is significant haemorrhage from the bowel or mesentery itself.

You can also control bleeding from the liver by compressing the vessels in the free edge of the lesser omentum (the Pringle manoeuvre, 15.8), or from the uterus or lower abdomen by pressing the aorta against the spine (22.11). This is most effective if you go through the avascular area of the lesser omentum after pulling the stomach downwards. Alternatively, if the bleeding is higher up, you will need to open the space between both *crura* of the diaphragm to expose the abdominal part of the thoracic aorta. Clearly, if you can get a vascular clamp onto the aorta, this is better than your fingers, *but don't give up if you don't have vascular instruments!* Just press and wait.

(b) Packing. A variation of this method is to pack a wound and to remove the pack ≤24h later, as with hepatic bleeding, bleeding from the pelvis, or after a sequestrectomy (7.5). *Note that packing does not mean stuffing gauze indiscriminately into a cavity,* but laying it carefully and methodically to obliterate a space if the packing is done inside a cavity, or laying gauze outside a solid organ on both sides like a sandwich, and wedging this firmly. If the packs become soaked at the edges, remove them gently and pack more tightly.

(c) Ligature. A haemostat (artery forceps) can be used to grasp a bleeding vessel, particularly an artery which is spurting blood at you. If the vessel is a large one which you'll need to repair, use vascular clamps or gauze-covered forceps. *Be sure you can see the vessel*. You can then tie it. Get an assistant to hold the forceps and release it when your tie is secure. If you cannot see the bleeding point, use a suture on a large needle and pass this through a good firm amount of tissue adjacent, and pull the suture towards you. This may control the bleeding, at least partially. Pass the suture in a parallel direction below the first point and so tie it as a figure-of-8 (4.8). Sometimes this does not fully control bleeding, so take 2 more bites at right angles (the 'clover' suture).

(d) Inflating a balloon in an orifice is a very useful procedure, especially in bleeding from the neck, liver or uterus. Pressure in a confined space is very effective at stopping bleeding.

(e) Repairing a bleeding vessel, either by closing a laceration in its wall or by making an end-to-end anastomosis, will be necessary to control haemorrhage in a major vessel. This may save a limb.

You may be able to control massive bleeding from a large vessel by inserting a balloon catheter into its lumen, and inflating the balloon.

Alternatively, occasionally you can put a tube shunt between the widely separated ends of a large important artery, fixing these in place with tape.

Get proximal control by formally exposing the vessel high above the bleeding point. This will only be necessary on unusual and desperate occasions. On rare occasions you may have to tie off the artery despite the consequences of distal ischaemia.

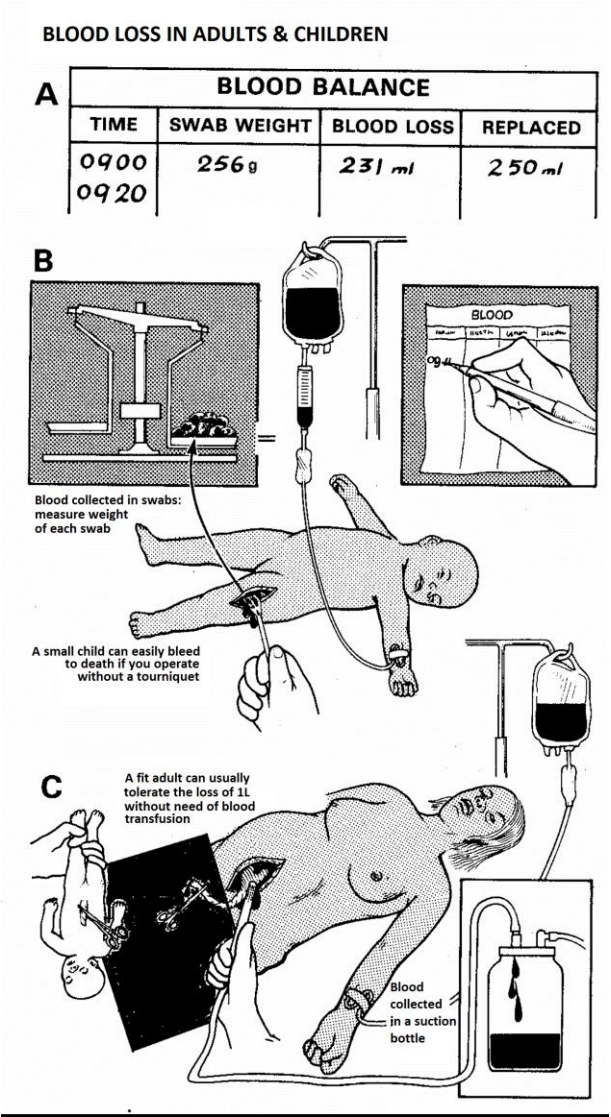


Fig. 3-1 BLOOD LOSS IN ADULTS AND CHILDREN.
A,B, when you operate on a child, make an accurate 'blood balance sheet'. In a major operation measure the blood lost by weighing the blood-soaked swabs on a balance. Replace blood lost with an equal volume of blood as soon as possible. This should be HIV-, HBV- and HCV-free. C, a fit adult, such as a mother having a Caesarean section, can tolerate a blood loss of up to 1L or even 1·5L, before you need to transfuse blood, rather than Ringer's lactate or saline. You can usually measure the blood lost in a suction bottle.

(f) Haemostatic gauze will eventually stop bleeding from the oozing cut surface of the liver, or the surface of the brain. Unlike ordinary gauze, it is slowly absorbed. It is expensive and rarely indicated. A substitute is to cut a piece of muscle, hammer it flat, and use this.

(g) Bone wax packed into the bleeding edge of the skull into the diploe, or into the marrow of a bone, will stop the bleeding if it is not too aggressive.

(h) Adrenaline, already added 1:100,000 to lidocaine solution or to saline, used to infiltrate the tissues, will minimize capillary and venous bleeding, *e.g.* during the repair of a vesico-vaginal fistula (21.18), in thyroidectomy (25.7) or cleft lip repair (31.7). You can also use a pack soaked with 1mg adrenaline in a bleeding nose (29.7), or on a bleeding tooth socket (31.3).
Never use adrenaline in the penis, or the distal parts of a limb such as a finger or toe, or in an IV forearm block, because it may constrict the vessels so much that the part becomes gangrenous.

(i) Hydrogen peroxide (6%, 20 vols) is useful not only to clean a wound infected with anaerobic organisms, but will also slow bleeding.

(j) Assisting blood clotting is important. When you have transfused >5 units of blood, the citrate in it will lower the calcium concentration in the blood and prevent it clotting. So *don't forget to add 10ml of 10% calcium gluconate IV after every 4th unit of blood*. When blood fails to clot, you can use fresh blood, but this may be impractical. You can store fibrinogen for such a purpose; fresh frozen plasma (FFP) is ideal but often pooled from several blood donors, and so its risks of HIV transmission are significantly greater than blood.

(k) Tranexamic acid (cyklokapron) 1g IV over 10mins and then 1g over 8h or as 20mg/kg tds is a useful adjunct, without these problems.

(l) Blood may fail to clot in the presence of liver disease, vitamin C deficiency, or if the patient has taken excess warfarin or its effect is potentiated by other medicines. In this case, use vitamin K 10mg IV if possible, as orally it takes 48h to be effective! Remember also that aspirin as well as garlic have an anticoagulant effect, and excessive use by patients may cause bleeding problems!

(m) Raising the bleeding part will lower the pressure in its veins, and so minimize bleeding. This is valuable if there is bleeding from a limb, or the venous sinuses of the brain (a rare and difficult emergency), when the level of the head in relation to the rest of the body is critically important. But there is a risk of air embolism if a rigid vascular channel, such as a sinus, is raised above the level of the heart.

(n) A proximal pneumatic tourniquet will control bleeding from the distal part of a limb, especially before or during an operation (3.4). For many operations this is essential, because it produces a bloodless field. Using a tourniquet in the trauma situation is useful to buy you time whilst you are organizing theatre. *Make sure you note how*

long the tourniquet is applied! A tourniquet round the cervix or uterus (22.11) can control uterine bleeding.

The common mistakes are:
(1) To panic when there is severe bleeding.
(2) Not to apply pressure when this is indicated, and not to apply it for long enough, or to apply it diffusely through more and more rolls of cotton wool and bandage.
(3) To grasp wildly with a haemostat in a pool of blood, to fail to grasp the bleeding vessel, and perhaps to injure some important structure.
(4) Not to apply the special methods for special sites.
(5) To cross-match blood too late.

A STORY ABOUT BLEEDING. A young trainee surgeon was excited to be able to assist the professor at an operation for a leaking aortic aneurysm. Predictably, there was quite a lot of bleeding seen when he released the big aortic clamps. In fact he hadn't seen so much blood in the abdomen outside of the trauma situation, where there was always frantic activity to stem the bleeding. When the professor had sutured in the graft, there was considerable oozing from the suture lines. He simply put in a big pack and asked the assistant to press gently, but firmly, till he returned, and went off to have a cup of tea! Petrified, the trainee hardly dared breathe, let alone move. When the professor came back 10mins later well refreshed, he re-scrubbed, and removed the pack; the operative field was perfectly dry.
LESSON When you control bleeding by pressure or with a pack, sufficient time (≥5mins by the clock) is all important.

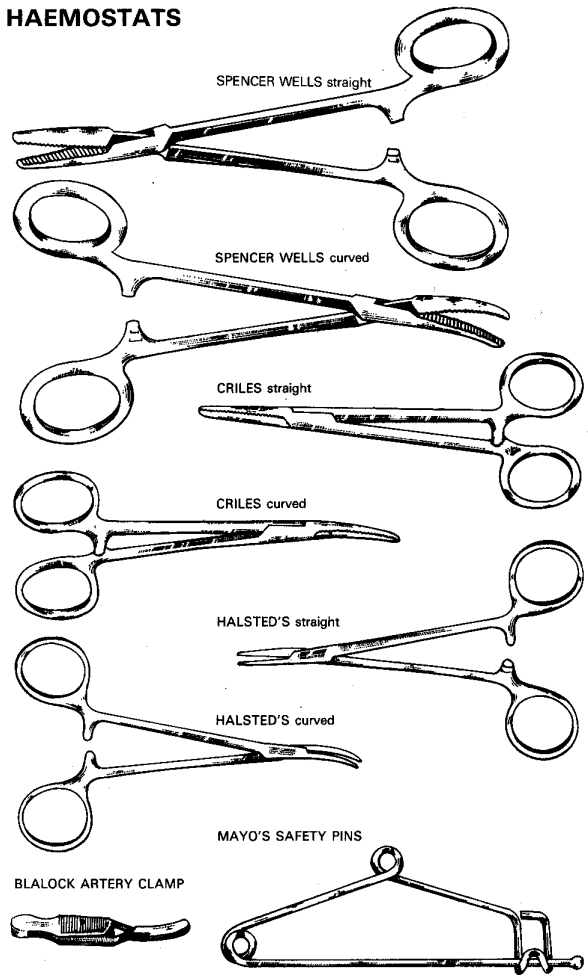


Fig. 3-2 HAEMOSTATS.
If you can see a bleeding vessel, you can usually grasp it with these locking forceps, which are one of the great inventions of surgery. *Don't use them as towel clips, because they will then no longer close finely enough for blood vessels*.

Spencer Wells are general purpose haemostats; Crile's are medium-sized and more robust than Halsted's, which are some of the finest and most delicate instruments and must be used with care. Blalock (bulldog) clamps are non-crushing clamps to stop blood spilling from a vessel whilst it is being repaired. Mayo's pins keep forceps together in bunches during sterilizing.

Spencer Wells, straight, box joint, (a) 200mm, (b) 150mm.
Spencer Wells, curved, box joint,
(a) 230mm, (b) 200mm, (c) 150mm, (d) 125mm, curved.
Crile's, straight, box joint, 140mm.
Crile's, curved, box joint, 140mm.
Halsted's, ultrafine, mosquito, haemostatic, straight, box joint, 120 mm.
Halsted's, ultrafine, mosquito, haemostatic, curved, box joint, 120mm.
Kocher's, straight, box joint, 200mm.
Kocher's, curved, box joint, 200mm.
Blalock artery clamps (various sizes).

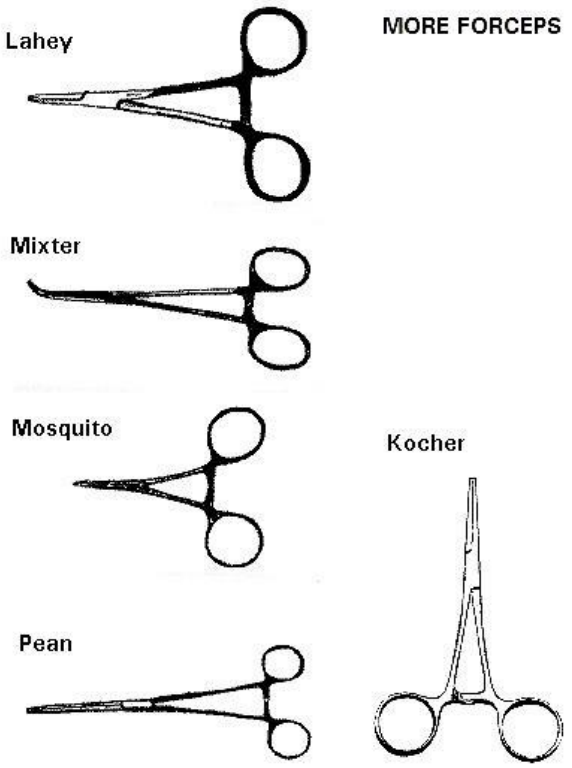


Fig. 3-3 MORE FORCEPS.
Lahey's are similar to Crile's; Mixter's have a more angled curve on their ends. The Mosquito forceps are fine and useful to hold threads. Pean's are long straight forceps. Kocher's are large haemostats with a tooth at the end of their jaws, for use on a wide vascular pedicle when an ordinary haemostat may slip.

3.2 Arterial bleeding

If you can see a bleeding vessel, you can grasp it with a haemostat (locking or artery forceps), which is one of the great inventions of surgery. Tie all larger vessels, either immediately or later. Small vessels, especially those in the skin, seldom need tying. When you remove a haemostat ≥5mins later, you will probably find that bleeding will have stopped. You can encourage it to stop by twisting the haemostat before you remove it, or if the bite of tissue is too large to twist, you can release the jaws and quickly pinch them together again a few times before you remove them.

Either of these methods will encourage the blood in the vessel to clot and will minimize bleeding, so that fewer vessels need tying.
Haemostats can be large or small, straight or curved, so that they rest over the edge of the wound. Haemostats do have some disadvantages. Each time you tie off a bleeding vessel you leave some crushed tissue and some suture material in the wound. If this is excessive, it can encourage delayed healing or infection later.
The tips of haemostats, especially small ones, must meet accurately, so good-quality instruments are important. *Never misuse haemostats as towel clips!* Box joints are worth the extra expense. Order them in sets of 6 (you can hardly have too many) because they will enable you to make up several sets (4.12).
It is best to cut skin boldly, which produces less bleeding, than tentatively and timidly, which produces a sawing type of action on the vessels.

(a) To tie an artery, use the following materials in this order of preference: long-acting absorbable, linen thread, cotton thread, or silk. *Don't use catgut for larger and more important vessels*: it slips off too easily and may be reabsorbed too quickly.
Grasp the bleeding artery with a haemostat. Either:
(1) Tie it with one firm reef knot.
(2) Tie it with a surgeon's knot (4.8) followed by 2 to 3 more throws.
(3) Transfix it, tie it with a reef knot, then pass one ligature through it with a needle, and tie it with another reef knot. This is the method for critically important vessels, such as those of the renal pedicle. For even more security, tie it proximal to a branch, and then cut it distal to this.

If it is a critically important vessel, ask yourself if what you've done is enough. If not, do it again: put a 2nd tie in a separate groove.

If there is a long length of vessel distal to your tie, shorten it, so as not to leave too much dead tissue in the wound, but *don't shorten it too much!*

If other methods of controlling severe arterial bleeding have failed, you may, very occasionally, have to expose and tie a major vessel, such as the external carotid or the subclavian artery. Use linen, cotton thread, or silk; *don't divide the vessel after you have tied it*, as it may recannulate.

(b) To control bleeding from a large pedicle, such as that of the spleen or uterus, *don't try to use a single ligature*. Control of the vessels will be safer if you take one or more bites of the pedicle and tie them separately.

(c) To control a difficult bleeding artery, try to get into the correct tissue plane. First find the artery by feeling for pulsation. Push the points of a fine haemostat into the connective tissue around it and separate them to open up a plane (3-4B). Gradually develop this plane until you can see the artery you are looking for. In this way you will avoid tying some important nerve in the ligature.

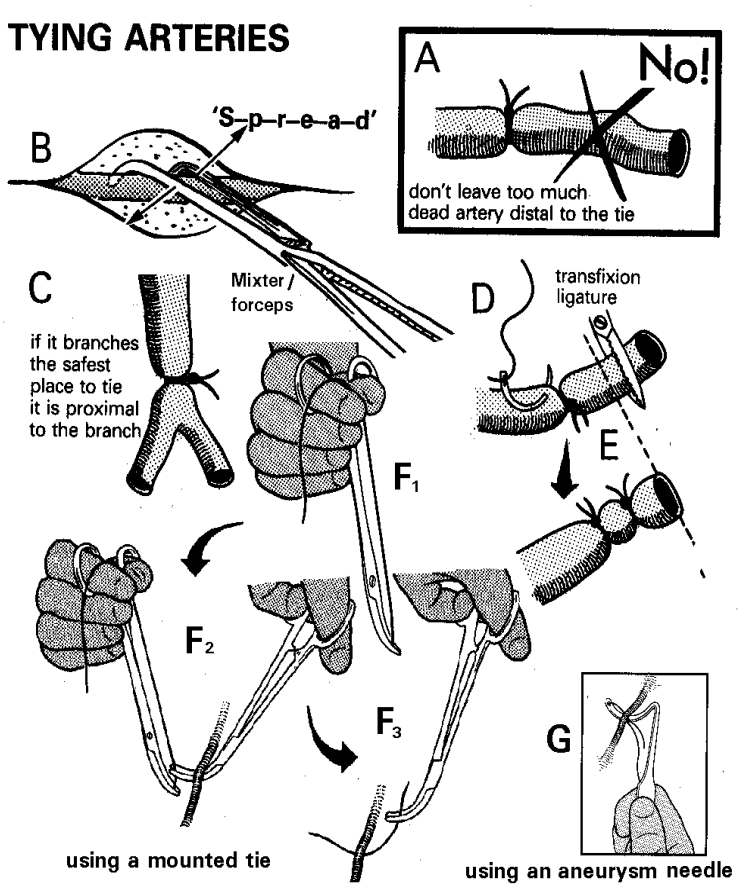


Fig. 3-4 TYING ARTERIES.
A, *don't leave too long an end*; this will leave unnecessary dead tissue in the wound. B, to free a vessel buried in tissue, insert Mixter forceps and spread the tissues. C, if possible, put the ligature proximal to a branch. D, tie the artery and insert a transfixion ligature; the needle is going through the vessel and its distal end is about to be cut off. E, completed ligature. F1, hold a length of suture material in a curved haemostat. F2, pass another curved haemostat under the vessel to grasp the suture material. F3, pull the suture material under the vessel. G, using an aneurysm needle.

TO GET A LIGATURE ROUND AN ARTERY, either use an aneurysm needle, or pass a curved haemostat under it, and ask your assistant to pass into your other hand a curved haemostat with a ligature 'bowstrung' across it (3-4F). This is useful in 'deep' surgery. You may be able to use ligaclips (4.10).

3.3 Diathermy

Heat causes coagulation of blood in vessels; this has been known for centuries. In order for an electric current to provide sufficient heat on a small area but without causing muscle spasm and cardiac dysrhythmia, diathermy uses radiofrequency currents of 0·5-1·5MHz.

(a) In monopolar diathermy, there is a high current density ensured at the point of contact with the active electrode at the diathermy probe tip but the current is then dissipated in a large volume of tissue through a large surface area 'indifferent' electrode, usually a plate placed under the buttock (3-5A). You must make sure this contact is good and uniform, otherwise a burn may result.

Make sure the wire connections in the instrument are sound, because poor contacts will increase the heat and so cause burns. Usually you will pick up a blood vessel with dissecting forceps, and touch the forceps with the diathermy tip. As metal is a good conductor of current, little heat is generated in its passage through the forceps.
Make sure though that your gloves have no holes, otherwise you may experience an electric shock and burn when the metal forceps come in contact with your own skin!

(b) In bipolar diathermy, the current passes between two point electrodes placed across the vessel to be coagulated. In this way a very high current density, and so much heat, is produced over a very small volume of tissue, with virtually no heat generated elsewhere (3-5B). Bipolar diathermy only works with low currents, and is therefore most suitable for small blood vessels, and fine surgical procedures.

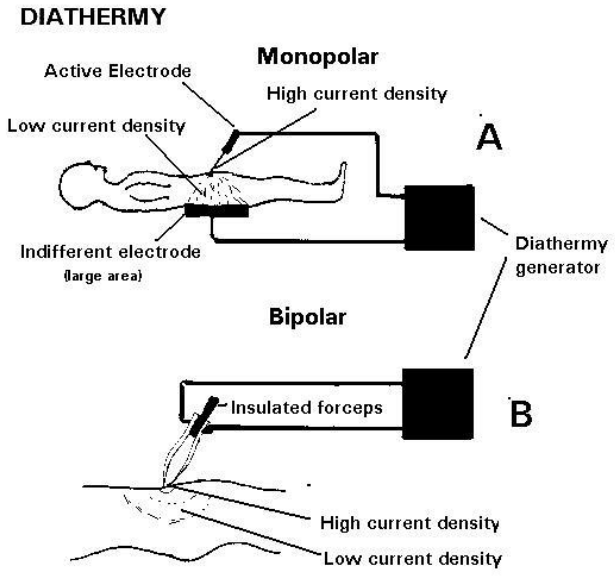


Fig. 3-5 DIATHERMY.
A, monopolar diathermy produces high current density at the active electrode dissipated through the body tissues through the 'indifferent' electrode with resulting low current density. B, bipolar diathermy produces high current density across insulated diathermy forceps tips.
After Sear JW, Rosewarne F. Anaesthesia for Surgeons, Oxford Textbook of Surgery, OUP 1994 p.83 Fig.2,3.

Radiofrequency generators produce different waveforms to give a coagulating (repetitive bursts of current cycles) or a cutting (continuous current) pattern; these are blended together for endoscopic resection (*e.g.* in prostatectomy).

DANGERS OF DIATHERMY
If you touch the skin or vessels very close to the skin edge with the diathermy electrode, you will produce a skin burn. If small, it is best to excise this, especially if it is on the edge of a wound. Otherwise, treat it like any other skin burn wound.

Don't use diathermy on the penis: you may cause thrombosis in the *corpora* unless you use bipolar diathermy.

Don't use diathermy on the groin if the scrotum is not in contact with the rest of the body: you might cause coagulation in the testicular vessels, especially if you lift the scrotum up in your hand.

Don't use diathermy in an amputation for an ischaemic leg (35.3): you will increase tissue necrosis.

Don't use diathermy on large blood vessels: tie them instead.

If the patient has a cardiac pacemaker, the diathermy current may affect this; so place the indifferent electrode far away, or use bipolar diathermy.

Don't use diathermy in the presence of inflammable anaesthetic agents, *e.g.* ether, and take care, if you use spirit-based skin cleansing preparations, that the fluid does not pool: serious burns may result.

Don't use diathermy on an obstructed bowel: it may detonate if methane gas has accumulated inside!

Reduce the current of the diathermy inside the mouth in operations under GA, because nitrous oxide as well as oxygen supports combustion, and its concentration is always higher there than elsewhere.

3.4 Bloodless limb operations

One of the great advantages of operating on a limb is that you can use a tourniquet to prevent bleeding. This will save blood and enable you to see the tissues more clearly. You can use any of these:

(a) A special pneumatic tourniquet which resembles the cuff of a sphygmomanometer. The pressure at which a tourniquet is applied is important; this is more easily controlled pneumatically, so a pneumatic tourniquet is much the best. Also you can, if necessary, let it down rapidly during an operation to perfuse the tissues, or to find arteries that need tying. The Conn improved pneumatic tourniquet with dial, complete in a case, in adult and child sizes is one of the most useful surgical appliances, and is almost essential; *alas, few hospitals have them*.

(b) An Esmarch bandage is a strip of red rubber. It is satisfactory, provided:
(1) You spread it out carefully over an encircling cotton wool pad.
(2) You don't put it on too tight, especially on a thin limb. (You can make one from an inner tube of a motorcycle tyre (3-6): the tube from an ordinary car tyre is too thick.)

(c) A reliable sphygmomanometer. You may not have a special pneumatic tourniquet, so this is probably what you will have to use.

A tourniquet will prevent blood entering a limb, but it will not remove blood which was already there when you applied it. Remove this blood in 2 ways:
(1) Raise the limb for ≥1min to help the blood to drain before applying the tourniquet. This is the only safe thing to do if there is sepsis. It will leave a little blood in the vessels, which can be an advantage, because you can more easily see where they are.
(2) Wind an Esmarch bandage round the limb from its distal to its proximal end to squeeze out the blood. Then apply a pneumatic tourniquet (or a sphygmomanometer) round the base of the limb to stop blood entering it. Finally, remove the Esmarch bandage. This will provide an almost totally bloodless field, *but is only safe if there is no sepsis*, which would then be spread proximally.

N.B. A tourniquet has disadvantages:
(1) **If you apply too much pressure for too long over too narrow an area**, you may injure the nerves to the limb, and cause a paresis; this is usually only temporary, but it may be permanent. A transient radial nerve palsy is common, even if you apply a tourniquet correctly.
(2) **If you forget to take a tourniquet off, so that it is left on for ≥6h**, Volkmann's ischaemic contracture, myoglobinaemia, or gangrene may follow. This happens more easily if there is arterial disease.
(3) **If a tourniquet is too loose**, it may obstruct only the veins, and increase bleeding.
So apply a tourniquet carefully; record the time when you applied it, and *don't leave it on too long*.

N.B. Never use a Samway's tourniquet. (This is a rubber tube with a hook at one end: it too easily injures the tissues beneath it.)

IF YOU APPLIED A TOURNIQUET, IT IS YOUR RESPONSIBILITY TO REMOVE IT

TOURNIQUETS
INDICATIONS
(1) Wound toilet in an injured limb, particularly if this has to be followed by repair of the vessels, nerves, and tendons.
(2) Any hand operation, other than a very small one. Hand injuries, and hand sepsis.
(3) The exploration and drainage of bones and joints, when this is anatomically possible, as in the lower humerus, the elbow and parts distal, or the lower femur, the knee, and parts distal.

CONTRAINDICATIONS
(1) The SS and CS varieties of sickle cell disease, *but not AS heterozygotes*.
(2) Ischaemia due to arterial disease.

ANAESTHESIA. A tourniquet is painful and a conscious patient will not usually tolerate one for >5mins. You will therefore need either GA or regional anaesthesia in most cases.

HANK (42yrs) was to have a bunion removed. The junior resident was asked to apply an Esmarch tourniquet. He had never applied one before, so he just wound the whole bandage round the patient's unpadded leg. 10 days later at the follow-up clinic the patient had a numb foot.
LESSON Learn how to apply a tourniquet before you apply one.

SITES FOR APPLYING A TOURNIQUET
There are only 4 of these:
(1) The middle of the upper arm (3-6D).
(2) The finger (3-6F). Use part of a rubber glove. This is only safe for a short procedure, such as draining a pulp infection.
(3) The upper thigh, a hand's breadth below the groin in an adult (3-6E). At this point the femoral artery lies close to the femur and is easily compressed.
(4) The cervix (23.7).

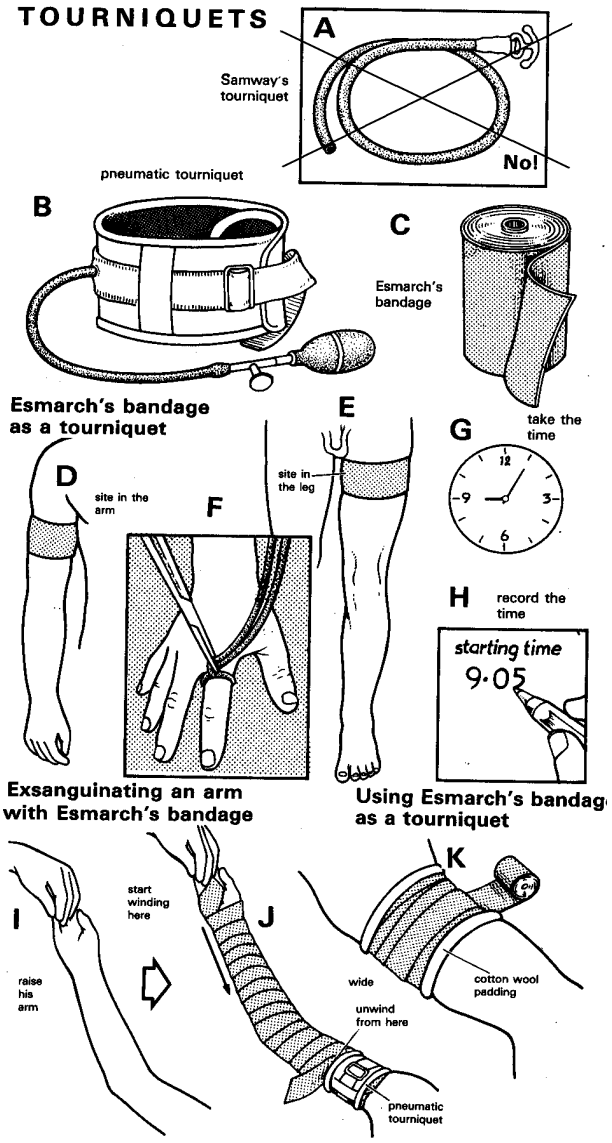


Fig. 3-6 TOURNIQUETS.
A, *don't use Samway's tourniquet,* as you may damage the tissues. B, a pneumatic tourniquet is much the best. C, the Esmarch bandage is a roll of red rubber. D, site to apply it in the arm. E, site in the leg. F, use a rubber catheter as a finger tourniquet. G & H, when you apply a tourniquet, take the time and record it. I, if you want to exsanguinate the arm, raise it and then apply Esmarch bandage, starting at the hand. J, inflate the pneumatic tourniquet, then unwind the bandage, starting proximally in the limb. K, you can use an Esmarch bandage as a tourniquet.

N.B. Don't exsanguinate a septic limb or where there is malignancy distally with an Esmarch bandage; you can use a simple tourniquet, though.

CAUTION!
(1) *A tourniquet on the forearm or on the lower leg is dangerous*, because you may damage the radial nerve at the ulna, or the common peroneal nerve at the neck of the fibula.
(2) Tie a tourniquet to the operating table, to prevent anyone forgetting it, because the patient cannot later be lifted off the table without removing it. *A tourniquet hidden under drapes can easily be forgotten.*

THE SAFE TIMES for an adult of average build are: the arm 1½h, the leg 2h. Shorten these times by 60% in a thin adult or in a child <8yrs. Apply a tourniquet to a finger for a few minutes only. The responsibility for keeping within these times lies with the anaesthetist, who should remind the surgeon every 15mins how long a tourniquet has been applied, and write on a board in the theatre when it was applied.

ELEVATE THE LIMB for a few minutes before you apply any kind of tourniquet. If you are going to apply an Esmarch bandage, now is the time to apply it.

REMEMBER TO USE ANALGESICS if you keep a tourniquet on for more than 30mins: it is painful!

PNEUMATIC TOURNIQUET. Place a folded towel, or a thin layer of cotton wool, around the limb at the site where the tourniquet is to be applied. Wrap this snugly round the limb: *it must not be loose*. Pump it up to the appropriate reading for 'arm', or 'leg', on the scale. For a child, use a lower pressure as indicated on the scale. Drape it out of the way of the operation, but keep the dial where you can read it. If the bag becomes contaminated, autoclave it (2.4).

USING A SPHYGMOMANOMETER AS A TOURNIQUET
On the leg apply the cuff over the femoral artery. On the arm apply it as if you were taking the blood pressure (BP), or if necessary higher up the arm. Bandage it in place with a firm unyielding bandage, and fix this with adhesive strapping. Inflate the cuff until the distal pulses just disappear. Remember the pressure, and let the cuff down again. When you want to use the cuff, blow it up to 80-100mm Hg above the pressure which just stops the pulses. This is about 200mm for the arm in an adult and 180mm in a child. For an adult leg blow it up to 250mm. Ask an assistant to keep the cuff at this pressure, and to inflate it as necessary if the pressure drops.
CAUTION! Don't inflate any cuff to >80-100mm above the pressure that will just obliterate the pulse.

USING AN ESMARCH BANDAGE
Raise the limb and squeeze blood out of it. Tape a folded towel or a thin layer of cotton wool in position over the limb. Apply the Esmarch bandage over c.12cm. Put on the first 2 layers of the bandage without pulling.
Next, do a trial run to find how many turns are necessary to obliterate the pulse. Pull out the bandage to about ¾ of its potential expansion length with each wind. Count how many winds you need to obliterate the pulse.
When you do apply it, apply 2 more winds than are necessary to obliterate the pulse. When you have finished, it should feel moderately firm, but *not rock-hard*.
CAUTION!
(1) *Don't apply a tourniquet over too narrow a band of muscles.*
(2) *Don't ever wind on more than five turns after you have obliterated the pulse*.
N.B. Every turn may add 100mm Hg more pressure.

TOURNIQUET TIME:
1½h IN THE ARM and 2h IN THE LEG;
60% LESS FOR THIN ADULTS AND CHILDREN

EXSANGUINATING A LIMB
INDICATIONS
Any operation in which you want a completely bloodless field, particularly orthopaedic.

CONTRAINDICATIONS
(1) Sepsis.
(2) Amputations for malignancy. It may spread both of these.

AT THE END OF THE OPERATION
There are 2 ways of controlling bleeding after you have applied a tourniquet:
(1) Release it just before you close the wound. Use this method when you do a fine operation on the hand, for example. It will reduce the blood clot in the tissues, and the stiffness and fibrosis that this might cause. Release the tourniquet, raise the limb, apply large swabs to the wound, and press on them firmly for 5 mins. Normally, bleeding will stop, though you should expect a measure of post-operative bleeding.
(2) Release it at the end of the operation after you have closed the wound. Use this method after operations in which clot in the tissues will be less important, as when you do a sequestrectomy (7.5). Tie any major vessels when you meet them during an operation. When the operation is complete, suture the wound, apply a dressing, and let down the tourniquet. Remove the pressure dressing 48h later. Usually, this is all that is necessary.
Observe the circulation in the limb at least hourly; the capillary reflex is important, so pinch the nail beds. Always check that a tourniquet is removed post-operatively: this must be part of the time-out procedure (1.8).

3.5 Post-operative bleeding

After you have closed an operative wound, it may start bleeding:
(1) During the first 48h (reactionary haemorrhage) because a clot in a vessel has been displaced, or a ligature has slipped.
(2) 8-14 days later (secondary haemorrhage) when the wound has become infected and eroded a vessel, usually quite a small one, sometimes a larger one.

One of the purposes of monitoring a patient immediately after an operation is to watch for reactionary haemorrhage, so make sure your staff observe carefully for early signs of blood loss, and understand what to do.

If a wound bleeds, try firm local pressure and packing. If it bleeds briskly, you may have injured an artery, such as the inferior epigastric. Minor bleeding is probably coming from the subcutaneous tissues, and is unlikely to be serious.

If local pressure fails to control bleeding, *don't apply more and more dressings;* take the patient back to theatre, open and, if necessary, enlarge the wound.
Irrigate it thoroughly with warm water. You can usually do this under LA. Remove the sutures and tie (3.2) or coagulate any bleeding vessels that you can see: you may need to put a running suture to control such bleeding. Liga clips may be useful (4.10).
If necessary, remove a pressure bandage or split a cast lengthways and open it at least 2cm. If you need to immobilize an open fracture, loosely apply a well-padded cast. You may need to re-apply the tourniquet, *but don't forget to remove it!*

If you have had to re-open a haematoma, add a single dose of prophylactic antibiotic (2.9).

N.B. Particular operations, *viz.* draining a peritonsillar abscess (6.12), removing a sequestrum (7.5), any laparotomy (11.2,10), draining an empyema of the gallbladder (15.4), cholecystectomy (15.8), liver biopsy (15.11), laparotomy for pancreatic abscess (15.15), splenectomy (15.17), block dissection of the groin (17.8), laparotomy for ectopic gestation (20.6), Caesarean section (21.10), D&C (23.4), myomectomy (23.7), hysterectomy (23.15), mastectomy (24.5), thyroidectomy (25.7), prostatectomy (27.20), eversion of hydrocoele (27.24), tonsillectomy (29.12), dental extraction (31.3) or varicose vein ablation (35.1), all have their own specific hazards.

If there are signs of circulatory failure post-operatively, with a fast pulse, pallor, perhaps with abdominal distension, confusion or even coma, this may be the result of:
(1) Blood lost at the operation not being replaced, especially if there was hypovolaemia before bleeding began.
(2) Fluid lost into the sequestrated bowel not being replaced.
(3) Anaesthesia too deep and depressed respiration, leading to hypoxaemia and hypotension.
(4) Overdosage of opioids, such as morphine or pethidine.
(5) Use of a high subarachnoid (spinal) anaesthetic.
(6) Septicaemia.

If bright red blood comes from a drain or incision, there is profuse arterial bleeding. Restore the circulating volume with 2L Ringer's lactate fast. Transfuse blood if the systolic blood pressure remains <90mm Hg. *Don't wait till the blood pressure is normal! Stop the haemorrhage!*

If you aspirate large quantities of fresh blood from a nasogastric tube after upper gastro-intestinal surgery, there is probably bleeding from a suture line. This is unlikely to stop spontaneously. You will probably need to re-open the abdomen to deal with the problem.

If there is bleeding some days after a laparotomy, the blood may be coming from a stress ulcer, or from a pre-existing duodenal ulcer unrelated to the previous pathology. This may threaten life. Treat this as described (13.4).

If blood is not clotting properly, check the clotting time. Take 5ml into a dry glass tube; invert it every 30 seconds, keeping it at body temperature, and time when it clots. If this takes >8mins, there is a clotting defect. Administer 10mg vitamin K IV. Use whole blood or packed cells and FFP to replace the blood loss, to try to replace the clotting factors.

Disseminated Intravascular Coagulation (DIC) may develop, especially with retained products of conception. If blood clots in ≤2·5min, it is hypercoagulable: thereafter if the clot lyses in 30mins, fibrin degradation has occurred. Use whole blood, FFP and fibrinogen 4-8g if available, to correct the clotting disorder.

3.6 Complications of blood transfusion

Blood is a dangerous substance and transfusion can cause severe, lasting problems, even death. Your laboratory ought to be able to cross-match any blood you transfuse; in a dire emergency, it is best to use the same group as the patient, then A(Rhesus)+ if the patient is A-ve or vice versa. But otherwise you should use O-ve blood, but you can probably get away with O+ve, except in women of child-bearing age. Even then there may be significant incompatibility problems.

The most important of the complications is transmission of hepatitis and HIV disease, and therefore your laboratory must be able to check for these also. There remains the danger of the 'window' period for HIV and so you should always think about auto-transfusion, even in the presence of mild sepsis (5.3).

These are the commoner infections that can be transmitted by blood transfusion:
1. Hepatitis A, B, C, D.
2. HIV disease.
3. Malaria.
4. Staphylococcal (or other bacterial skin) sepsis.
5. Atypical mononucleosis (glandular fever).
6. Brucellosis.
7. Cytomegalovirus.
8. Syphilis.
9. Yersinia.
10. Trypanosomiasis.

Apart from these, there may be ABO or Rhesus (Rh) incompatibility as well as 26 other types of cross-reactions, as well as plasma reactions and problems related to the blood being still frozen or overheated.

A graft-versus-host disease can rarely occur, producing an ARDS-type lung injury.

Stored blood may have [K^+] of 40-70mM, so multiple transfusion may produce a dangerous hyperkalaemia, and the citrate used to preserve its liquidity may produce a worsening acidosis. However, often more importantly, as stored blood loses its clotting factors after 24h, coagulation becomes disrupted. Further, the citrate soaks up calcium, and this further aggravates bleeding. Haemorrhage may even be exacerbated by a consumptive coagulopathy producing DIC.

If your laboratory produces packed red cells (because other blood products are filtered off), there are no platelets in the blood. Using whole blood avoids this problem and holds the clotting factors necessary for haemostasis. Stored blood is just not as good!

However, there may be a greater risk of multiple pulmonary emboli (the acute respiratory distress syndrome), and an antigenic response producing release of vasoactive substances and complement as well as depressing the reticulo-endothelial system. Blood transfusion definitely decreases immunity, and the risks of recurrence after cancer surgery may increase by c. 10%.

Finally, if you transfuse an anaemic patient, especially if his anaemia is chronic and compensated, you can so increase the blood volume that you tip him into acute heart failure. This is particularly important in children.

(a) Protocol

Check the blood units individually for compatibility (name, hospital and batch numbers, group) and expiry date. Make sure an IV line is patent and flushed with saline. Warm the blood (*don't heat it up!*). Check the blood unit to be used again, and make sure it is signed for. Attach the blood unit to a blood-giving IV infusion set (with a filter). Observe the patient ½hrly for pyrexial or other reactions, and chart infusion volumes.

N.B. Don't stop a transfusion because of a minor pyrexia especially if the patient is septic anyway.

Administer 10ml (2.2mmol) 10% calcium gluconate IV with every 4th unit of blood transfused.

N.B. 10ml of 10% calcium chloride IV provides 6.8mmol.

Administer 20mg furosemide IV with each unit of blood if cardiac failure is feared.

Stop the transfusion if there is a serious reaction; administer 100mg hydrocortisone IV, and preserve the blood unit for laboratory analysis later.

(b) Life-threatening emergencies

Severe haemorrhage often occurs in unforeseen circumstances. It is best to be prepared rather than sorry after the event. Try to keep at least 2 units of O-ve blood continuously available in your hospital, because there will be no problem transfusing this in 95% of your patients.

If you cannot get O-ve, O+ve will be satisfactory for 85-95% of cases, so if a patient is *in extremis, don't fear the risk of 5-15%!*

Fresh blood is often better than stored blood; try to have reliable persons (tested regularly HIV-ve) in your community available to assist in an emergency with blood transfusion. Remember to try to correct clotting disorders, if present.

N.B. Fresh frozen plasma can be stored for a long time as opposed to blood and should therefore be available via the national/regional blood bank. However, as one unit is collected from more than one donor, the risk of HIV, hepatitis transmission etc. is that much greater.

4 Basic methods & instruments

4.1 Appropriate surgical technology: the equipment you need

You may step into a beautifully organized theatre, or you may have to create it from scratch. To help you in this task we have listed everything you might need to do the procedures we describe, down to the last needle and cake of soap. To minimize the tediousness of long lists, we have described the equipment in the text. We have included everything which you could reasonably obtain but may not have at the moment. For example, many district hospitals don't have skin-grafting knives, pneumatic tourniquets, simple bone drills, Kirschner wire (K-wire), or manometers for measuring the central venous pressure, but you could reasonably try to get them, so we have included them. Some of the special methods we describe don't need any extra equipment: *e.g.* the plastic bag method for laparostomy (11.10). Learn to recognize the instruments you use and to know them by their names. Remember the instruments may have different names in different countries! When you first arrive at a hospital, check the theatre equipment and find out what is missing!

When you order equipment that is not listed here, try to make sure that:
(1) It will work reliably (good quality) without needing to be returned to the makers to be mended.
(2) It will work well in your hands and is electrically compatible.
(3) You can afford both its initial and its running costs.
(4) Spares are available.
(5) You can easily learn how to use it and teach other people to do the same.
(6) It can be repaired locally if need be.
Think about whether it needs to be portable, and so how robust it needs to be. *Don't get persuaded by wily salesmen into buying things you don't really need!*

If you want to be well supplied, encourage and motivate your storeman. Look at what there is and how he has organized things. *Don't forget to visit your central medical stores*; you may find things you need, which the storeman there cannot identify, and you can make good use of. The equipment we list is the equipment he should stock.

You will certainly have to improvise. If you don't have the standard stainless steel instruments, *don't hesitate to use ordinary steel ones*, if you can buy, adapt, or make them. You will need to wipe these carefully with an oily rag after each operation. For example, you can use an ordinary steel carpenter's drill instead of a bone drill, and a sterile pair of ordinary pliers may be the best way to remove a plate. You can save much on IV fluids by infusing water rectally: an adult patient will readily tolerate and absorb 500ml over 6h. If you have no K-wires, you may be able to use sharpened bicycle spokes.

Don't store instruments of ordinary steel sterilized in packs or drums: the interior of these is damp and they will rust rapidly.

Stores and equipment

SUPPLY CYCLES. If your supply period for a consumable item is 'x' months, try to keep 3 times the quantity you consume during this period in stock, so that one indent can go astray without causing disaster.

When you order equipment, try to include the catalogue number. Where possible, write to the supplier and ask for a 'proforma invoice' giving the exact details and costs, etc. This will make ordering much easier.

The theatre. Theatre furniture and lighting, gowns, gloves and drapes (2.1, 2.3), drains and tubing (4.9). Miscellaneous smaller items of theatre equipment (4.12).

Preventing sepsis. Sterilizing equipment (2.4), antiseptics and disinfectants (2.5).

Preventing bleeding. Haemostats and arterial clamps (3.1), tourniquets (3.4).

Cutting and holding tissues. Scalpels and dissectors (4.2), scissors (4.3), forceps (4.4), retractors (4.5), suture materials (4.6), needles and their holders (4.7).

Instruments for bones (7.5), bowel (11.3), obstetrics (19.2), proctology (26.1), urology (27.1), eyes (28.1), ENT (29.1), tracheostomy (29.15), dentistry (31.1, 31.3), chest aspiration (36.1). *N.B. Names may be different in non-Anglophone countries!*

4.2 Scalpels and dissectors

A sharp scalpel cuts tissue with less trauma than any other instrument. There are 2 ways of holding one:
(1) If you need force to make a big bold cut, grasp it with your index finger along the back (4-1).
(2) If you want to cut more gently, hold it like a pen.

The size of a blade does not change the way you use it, but its shape does. A small blade allows you to make precise turns. Some blades have very specialized uses. Use the stab point of a #11 blade to open an abscess. Use #12 blades for removing sutures. Use a #15 blade for small incisions, and a #10 for larger ones; a #20-24 is best for a laparotomy incision. The smaller blades (sizes 10-19) fit onto the #5 Bard Parker handle, and the larger ones (sizes 20-36) onto the #4 handle. A guarded scalpel is useful for special situations, like tonsillar (6.7) and retropharyngeal abscesses (6.8). A fixed scalpel is especially useful for symphysiotomy (21.7).

If you find the scalpel difficult to use at first, use sharp scissors, but as your experience grows you will find the scalpel easier and safer.

Beware of cutting yourself or an assistant when using sharp instruments!

Make sure you have secured the blade tightly on the handle, because if you lose it in a body cavity it is difficult and hazardous to find it again!

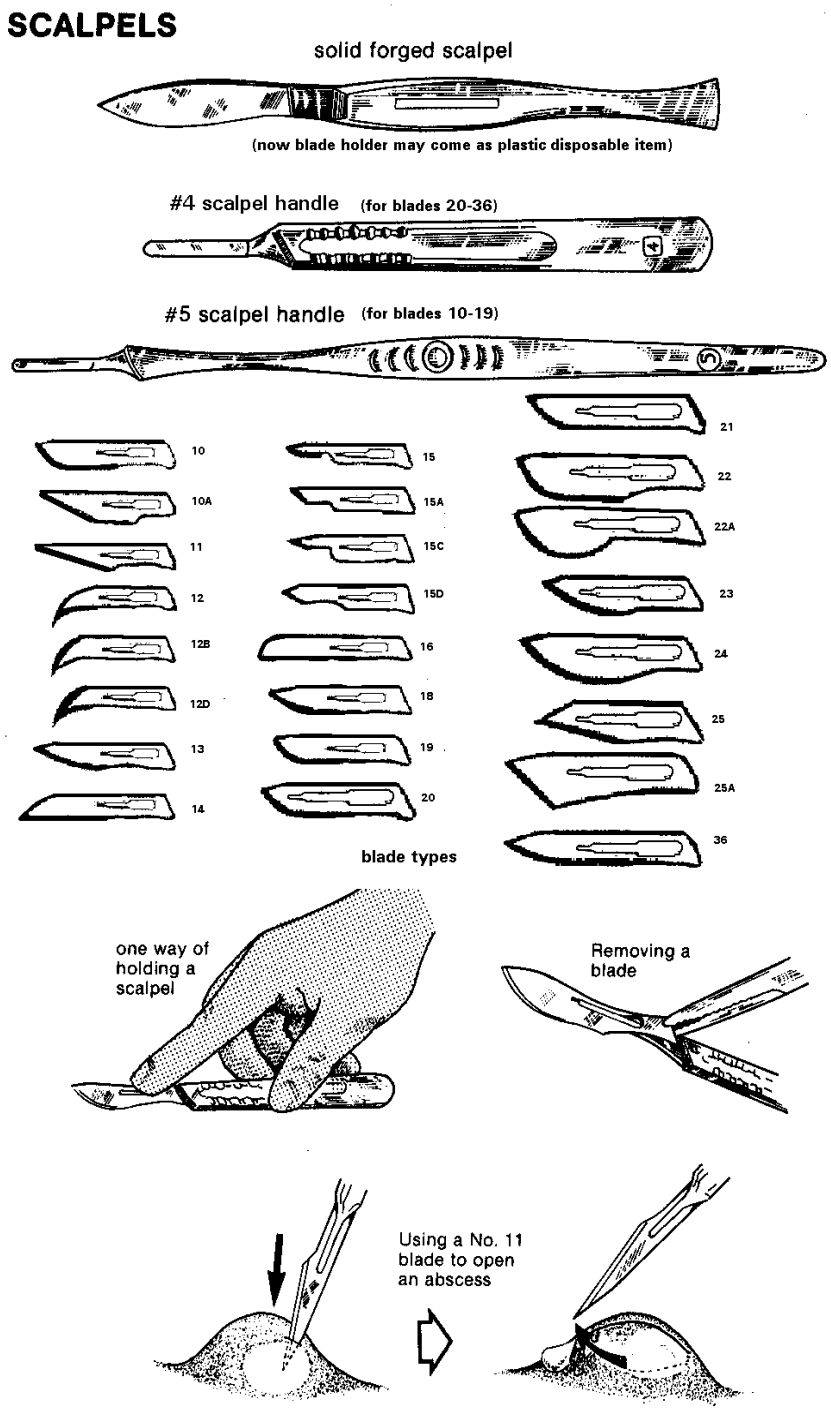


Fig. 4-1 SCALPELS AND HOW TO USE THEM.
The advantage of a solid forged scalpel is that you can resharpen it. It is useful for symphysiotomy (21.7)**. Take care when you remove a blade: *always use an instrument, hold the blade with the sharp side away from you and never use your bare fingers or plastic forceps!* Dispose of sharps in special containers** (5.3)**.**

SCALPEL, solid forged, size #1, 30mm, and size #5, 40mm. If your disposable blades are exhausted, you can use a solid scalpel and re-sharpen it (4-3), whereas you cannot re-sharpen a disposable blade.
HANDLE, scalpel, Bard Parker, #4. Get good-quality handles, because poor ones may not fit the blades.
HANDLE, scalpel, Swann Morton, #5.
BLADES, scalpel, disposable, Bard Parker or Swann Morton type, stainless steel.
OILSTONE, hard Arkansas pattern, 150x70x30mm.
Use this to sharpen scalpels and scissors. A very blunt instrument needs a carborundum stone first.
DISSECTOR, MacDonald. A blunt dissector is often safer than a scalpel. This is a blunt general-purpose dissector, with 1 straight flat end and 1 round curved end, neither of which are likely to injure anything.

4.3 Scissors

The tips of a pair of surgical dissecting scissors are usually rounded; scissors in which both tips are pointed are only used for very fine dissection. Look after your scissors carefully. Use straight scissors near the surface and curved ones deeper inside. Hold them with your index finger resting on the joint. Use only the extreme tips for cutting.

You can also use scissors for blunt dissection by pushing their blades into tissues and then opening them. This will open the tissues along their natural planes, and push important structures, such as nerves and blood vessels, out of the way. This is the 'push and spread' technique (4-9B). If there is something nearby which it would be dangerous to cut, blunt dissection is always safer. But remember that *even blunt dissection can injure veins, and that venous bleeding can be very difficult to control.*

Remember:
(1) *Don't use sharp-tipped scissors in dangerous places, or cut what you cannot see*.
(2) *Don't use scissors which are longer than the haemostats you have,* or you may find yourself cutting a vessel which you cannot reach to clamp.
(3) Mayo's, McIndoe's, and Metzenbaum's scissors are intended for cutting tissues, so *don't use them for anything else*. Use other scissors for cutting sutures and dressings.
(4) Carefully keep and pack very fine scissors, *e.g.* ophthalmic instruments, separately.
(5) Beware, when passing scissors, to do so holding the closed tips, so that your assistant or scrub nurse can take them by the handle. Alternatively, place them on a special tray.

Note that it is not necessary or even desirable to have all these sorts of different scissors.

Make sure you look after your sharp instruments carefully. Buy good-quality scissors, and *don't autoclave them mixed together with the other instruments*. The very best ones have tungsten carbide inserts, which make their cutting edges last much longer. These are 4 times more expensive, but justify their extra cost.

IN DANGEROUS PLACES BLUNT DISSECTION IS SAFER THAN SHARP DISSECTION WITH SCISSORS

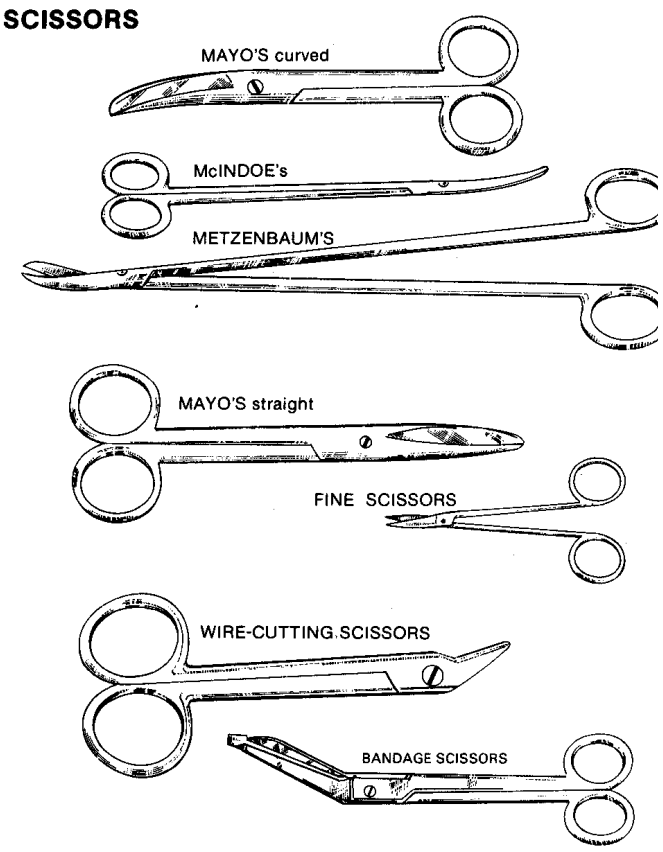


Fig. 4-2 SCISSORS.
Mayo's, McIndoe's, and Metzenbaum's scissors are intended for cutting tissues, so *don't use them for anything else*. Use other scissors for cutting sutures and dressings.

SCISSORS, operating, Mayo, straight, bevelled, 200mm.
SCISSORS, operating, Mayo, curved, bevelled blades, 170mm.
These tissue scissors are curved in the plane of the blades.
SCISSORS, operating, McIndoe's, curved, with rounded tapering blades, 180mm. These elegant tapering tissue scissors are curved perpendicular to the plane of the blades.
SCISSORS, operating, Metzenbaum, curved 275mm. These have long handles and quite narrow blades. Use them for dissecting at the bottom of a deep wound.
SCISSORS, Aufrecht's, light, curved, 140mm. This pair of scissors is for the set of instruments for hand surgery.
SCISSORS, straight with fine sharp points, Glasgow pattern, 100mm, stainless steel. Use these very fine scissors for cutting down on veins.
SCISSORS, suture cutting, 'assistant's scissors', rounded ends. Keep these in spirit with the other scissors. Your assistant needs a pair; so does the scrub nurse.
SCISSORS, suture wire cutting, 130mm. If you cut suture wire with ordinary scissors, it will ruin them.
SCISSORS, bandage, angular, Lister, 180mm. These have a blunt knob at the end of one blade which goes under the bandage to protect the patient. Insert them away from the wound; if they become soiled or wet, clean and sterilize them before you use them on someone else.

CARING FOR YOUR EQUIPMENT

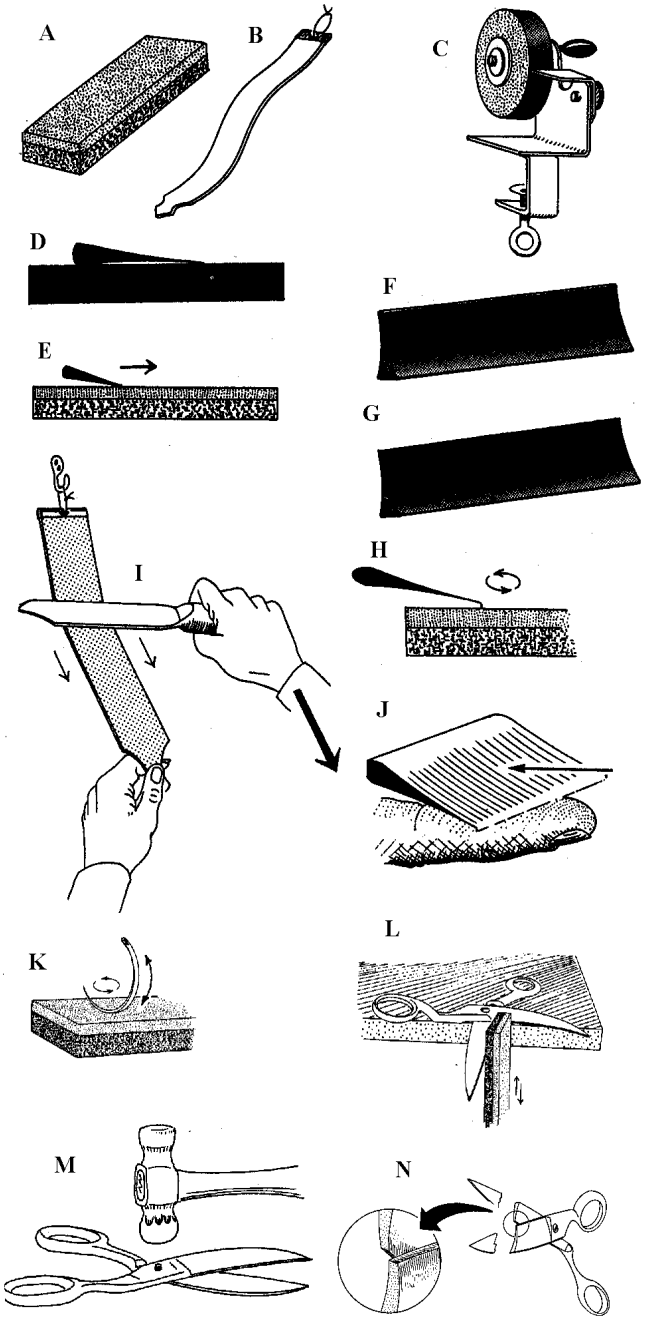


Fig. 4-3 CARING FOR YOUR EQUIPMENT.
A,C, grindstones. B, strop. D, if your razor is hollow-ground, lay it flat, so that both edges rest on the stone and push it forwards. E, if it is ground on the flat, lift its heel slightly and push it forwards. F, light reflecting from the *blunt* edge of a razor. G, no reflecting light from a sharp razor. H, removing a burr. I, stropping a knife by pulling it towards you. J, feeling if there is a burr on a blade by drawing it backwards across your finger *(make sure you do this when the blade is sterilized)*. K, sharpen a cutting needle by rotating it in 2 planes on a stone. L, sharpen a pair of scissors against a grindstone. M, tighten the rivet of a pair of scissors with a light hammer. N, the cutting edges of scissors should look like this.

4.4 Forceps

(a) Dissecting (thumb) forceps can be short for working close to the surface, or longer for working more deeply. They can be plain, or toothed with an odd number of teeth on one jaw, and an even number on the other, either 1 into 2 teeth, or 3 teeth into 4, etc. Toothed forceps hold tissue so firmly that only a little pressure is necessary, but they can easily puncture a hollow viscus or a blood vessel. Strong, plain, straight forceps without teeth are even more useful for blunt dissection than they are for holding tissues.

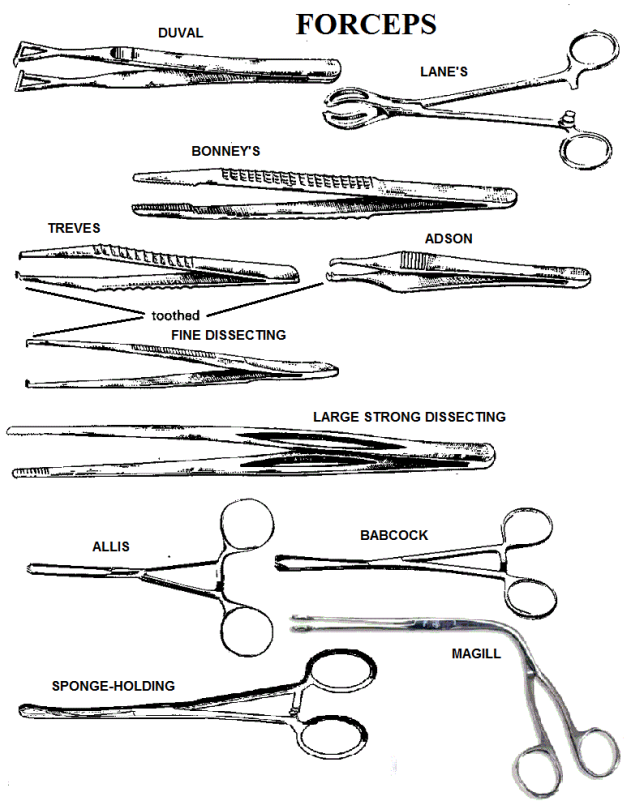


Fig. 4-4 FORCEPS.
Dissecting forceps are also called thumb forceps, and can be plain or toothed. Use forceps according to the size of tissue you are handling: Lane's tissue forceps have teeth and are useful for holding a large piece of tissue you are going to excise; Bonney's for the abdominal wall; Treves or Adson's for skin; Duval's have a groove to hold tissue with minimal trauma to it; Babcock's have bars on each blade that come together gently without damaging the tissues, and so are useful for holding bowel or ureter which you don't want to damage; Sponge-holding forceps for gauze swabs, or to retract the gallbladder (15.8). Magill's are specifically for guiding an endotracheal tube, but also for extracting foreign bodies from the throat (30.1).

(b) Tissue (locking) forceps have a ratchet which keeps them closed. Some have teeth (Allis) and some have none (Babcock's). The blades of Allis forceps meet together, and inevitably injure the tissues a little, whereas Babcock's have bowed jaws with a gap between them. This makes them gentler but less secure. When you use Allis forceps for retracting a skin flap, apply them to the subcutaneous tissue or fascia, and not to the skin itself, which may be injured. Kocher's forceps are stronger, have a toothed end, and are even more traumatic; they are for clamping fascia or wide vascular pedicles, so that the vessels don't slip out (3.1).

FORCEPS, dissecting, thumb, blunt, non-toothed, Bonney's, 180mm. These are strong dissecting forceps without teeth.
FORCEPS, dissecting, thumb, toothed, Treves', 1x2 teeth, 130mm. These are the standard toothed dissecting forceps.
FORCEPS, dissecting, thumb, fine, Adson's, (a) plain, (b) 1x2 teeth, 120mm. These have broad handles and fine points and are particularly useful for the eye.
FORCEPS, dissecting, thumb, Duval's, 150mm, with non-traumatic teeth on triangular jaws. These are thumb forceps for general use.
FORCEPS, dissecting, thumb, toothed, 180mm. These are long fine dissecting forceps.
FORCEPS, dissecting, thumb, Maingot's, 280mm. These are large toothed forceps with fenestrated sides that are easy to hold.
FORCEPS, dissecting, McIndoe's, plain, 150mm. These are for the hand set.
FORCEPS, dissecting, ophthalmic, Silcock's, 100mm. This is a fine pair of forceps for operating on the eye or the hand.
FORCEPS, tissue, locking, Allis, box joint, 150mm, 5x6 teeth.
FORCEPS, tissue, locking, Babcock's, box joint, 160mm. These have a bar on each blade that comes together gently without damaging the tissues. Use them to hold bowel.
FORCEPS, tissue, Lane's, 15cm. These have curved jaws, teeth and a ratchet.
FORCEPS, sinus, Lister, box joint, 150mm. You can use these for many other purposes besides exploring sinuses. Use them for packing the nose, or putting a drain into an abscess cavity.
FORCEPS, cholecystectomy, curved jaws with longitudinal serrations, Lahey's, box joint, 200mm. These forceps are useful for other purposes besides dissecting out the cystic duct. If you put them into the tissues and separate them, you can use their rounded ends to define arteries, veins and ducts.
FORCEPS, intestinal, Dennis Browne, 180mm. Use these to pick up the bowel during an abdominal operation, or a hernia repair.
FORCEPS, Moynihan, box joint, 220mm. Use this massive pair of crushing forceps for wide vascular pedicles, such as those which contain the uterine vessels at hysterectomy.
FORCEPS, Desjardin's, screw joint. Use these for removing stones from the bile duct.
FORCEPS (clamps), hysterectomy, curved, box joint, 1 into 2 teeth, 23cm, Hunter or Maingot. Hysterectomy is difficult without several long curved clamps for big vessels, preferably with longitudinal serrations and teeth at their tips.
FORCEPS, Magill's. Use these in endotracheal intubation and for removing foreign bodies in the throat (30.1).

N.B. It not necessary nor even desirable to have all these sorts of different forceps.

4.5 Retractors and hooks

You will need a retractor to hold tissues out of the way of where you want to operate. There are 2 kinds. One has to be held by an assistant, the other holds itself.

Self-retaining retractors should never stretch a wound and cause ischaemia: make a bigger incision if the field is inadequate! Strong retraction causes trauma, especially to the edges of the wound. So avoid it by approaching deep areas through larger incisions. Avoid sharp, pointed retractors.

Any blacksmith should be able to make you the simpler retractors from ordinary steel.

If you need an assistant to hold a retractor for a considerable length of time, engage him in the operation lest his concentration wander at a critical moment!

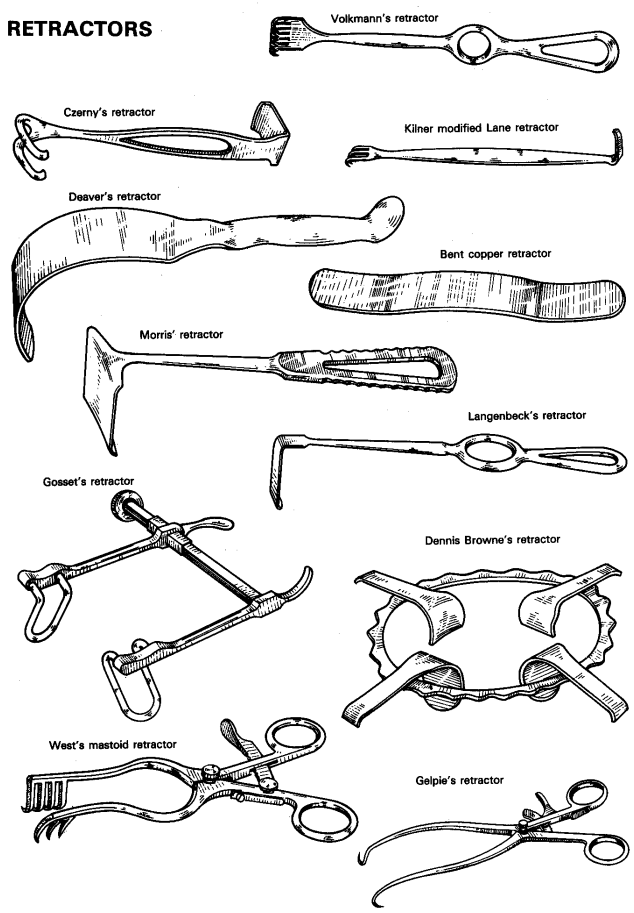


Fig. 4-5 RETRACTORS.
You cannot operate on a patient if the surrounding tissues get in your way. These retractors will help to give you a clear field. Some have to be held, others hold themselves.

RETRACTOR, Volkmann's rake, sharp, 4 prong, 220mm. These have sharp teeth like a cat's paw. Take care that they don't injure anything important.
RETRACTOR, Langenbeck, 13x44mm. These are fairly small narrow deep retractors.
RETRACTOR, Czerny, double-ended. These have a flat blade at one end and 2 deep prongs at the other. They are thus more versatile than Langenbeck's retractors.
RETRACTOR, Lane's modified by Kilner, double-ended, 150mm. This is a light general-purpose retractor with short shallow hooks at one end and a tongue at the other.
RETRACTOR, Gelpie, 170mm. A pair of these are very useful as general-purpose retractors.
RETRACTOR, Morris, double-ended. This is a double-ended abdominal retractor. Some surgeons prefer single-ended ones which are easier to hold.
RETRACTOR, Deaver's, plain handles, set of five sizes. These inexpensive general-purpose abdominal retractors nest together, and so are easy to store.
RETRACTOR, malleable copper, set of 4 sizes. These are strips of copper that you can bend into any shape to suit your needs.
RETRACTOR, Meydering, 178mm. These are for hand surgery and are used as a pair.
RETRACTOR, self-retaining, West's, straight, sharp-pronged. This is a small self-retaining general-purpose retractor.
RETRACTOR, abdominal, self-retaining, 2-blade, adult, Gosset's. The 3 blades of this large abdominal retractor can be arranged so that they support one another, and don't have to be held.
RETRACTOR, universal, Dennis-Browne, with (a) 1 frame 300x240mm, (b) 3 hook-on retractors 50x65mm, (c) ditto 80x90mm, (d) ditto 98x50xmm, (e) ditto 105x35mm. This is a useful but expensive retractor. It has a notched ring and hooked prongs.
HOOKS, tendon, Harlow-Wood, 114mm. These are for the hand set (4.12).

4.6 Suture materials

If you bring soft tissues together and hold them there for 5-10 days (depending on their blood supply), they will join. Most surgery depends on this. The easiest way to hold tissue is to suture it. You can use:
(1) Absorbable sutures which are absorbed by the tissues so that you need not remove them.
(2) Non-absorbable ones which you leave indefinitely if they are deep, or remove if they are on the skin.

(a) Absorbable sutures used to be solely plain catgut (from the submucosa of the bowel of sheep, not cats!) which usually holds its strength for about 10 days. Catgut can be treated with chromic acid which slows its absorption by phagocytosis and makes it keep its strength for 20 days, but this remains unpredictable. Sepsis speeds the dissolution of catgut, especially plain catgut, so that it may dissolve in 2-3 days. Catgut is soft and holds knots well, but not so well as a non-absorbable multifilament, such as linen or cotton. If a suture material does not hold knots too well, its knots need longer ends (>5mm). While catgut is being absorbed, it makes a good culture medium and may promote sepsis. So *don't use more than is necessary, don't leave the ends of ligatures unnecessarily long* and *avoid thick #2 or #3 catgut.* Plain catgut does not hold its strength for very long, so never use it for tying larger vessels or suturing the bowel. One problem with catgut is that it may be of poor quality and does not preserve for long, and so gives way early and perhaps disastrously. This is another reason for using monofilament where you can.

If necessary, you can use almost any suture material almost anywhere, especially on the skin. But, always use absorbable for:
(1) The urinary and the biliary tracts, because non-absorbable sutures can act as the focus around which a stone can form.
(2) The mucosa of the stomach, where a non-absorbable suture may be the site of an ulcer later.
(3) The mucosa of the uterus (less important).
(4) Sutures close under the skin, where non-absorbable sutures may work their way to the surface.
(5) The scrotal skin, where sutures easily 'disappear' and cause intense itching.

There are long-lasting absorbable sutures which are more reliable, but they are expensive. It might be useful to have limited stocks for special purposes, *e.g.* bowel anastomosis, where suture breakdown is a disaster and the extra cost readily justified. Various synthetic materials are used, with different absorption times:

Polyglycolic Acid	('Dexon', 'Polysorb')	30-60 days
Polyglactin	('Vicryl', 'Visyn')	60-90 days
Lactomer 9-1	('Clinisorb')	60-90 days
Poliglecaprone 25	('Monocryl')	90-120 days
Glycomer 631	('Biosyn')	90-120 days
Polydioxanone	('PDS')	120-180 days

N.B. Vicryl rapide has a much shorter absorption (10-15 days) and Vicryl plus (preserved in triclosan antiseptic) longer.

(b) Non-absorbable sutures can be polyamide ('Nylon'), polypropylene ('Prolene', 'Surgilene', 'Surgipro'), polyethylene ('Courlene'), polyester ('Dacron', 'Mersilene', 'Surgidac'), which may be coated with polybutilate ('Ethibond') or silicone ('Ticron'), linen, cotton, silk, or stainless steel wire. The first three come as a single (mono)filament, or as multiple filaments which are braided or twisted together. Monofilament is the most useful general-purpose suture. Although non-absorbable sutures remain as permanent foreign bodies, monofilament nylon, polyethylene, and steel are less likely to promote infection than catgut, or multifilament cotton, linen, or silk.
Unfortunately, a single thicker filament makes less reliable knots than many finer ones braided or twisted together, except for steel wire, which is always used as a single filament, and which knots superbly but is difficult to work with. So, always tie monofilament with a surgeon's knot (4.8). Silk, linen or cotton knot well, and you can cut these sutures 2mm from the knot.
Apart from the indications for absorbable sutures given above, you can use monofilament for almost anything, but silk, cotton, or linen threads are better than monofilament for tying larger vessels. However, *never hold monofilament with artery forceps or a needle holder* because you will seriously weaken it at that point. Braided silk may cause troublesome stitch abscesses. *Don't use it immediately under the skin*, because it may work its way through to the surface, long after healing is complete. If it does become infected, you may have to remove it piece by piece. Even monofilament can come to the surface, so keep it well buried, and use absorbable close under the skin.

(c) The strength of sutures is measured in 2 systems. In the old system the finest ones are measured in 'zeros' and the thicker ones are numbered. From finest to thickest, the sequence is, with doubling of diameters each time, 6/0, 5/0, 4/0, 3/0, 2/0, 0, 1, 2, 3, 4. Although attempts are being made to replace the old system with a metric one from 0-8, most surgeons still use the old one.
Use the thinnest sutures you can: they need only be as strong as the tissues they are holding together. You can do most operations with sutures between 3/0 and 1. Only very occasionally will you need sutures which are thicker or thinner than this, except for fine work such as nerve or tendon repairs, and for eye and plastic surgery. If you do need a thicker suture, you can double up a thinner one.

(d) The cost of sutures can significantly increase the cost of an operation. In the industrial world they are now sold in individual disposable packs, which are expensive to make and waste much suture material each time a pack is opened. (If the outer wrapping is opened in error, the suture is still sterile and *should not be discarded!*) The suture is combined with an atraumatic needle and this means that sutures for one operation may cost US$20. But if you buy monofilament in rolls, and use ordinary needles, the suture materials for a single operation cost almost nothing. Monofilament suture material in packets is 20,000% more expensive than in reels, and with needles swaged on is 30,000% more!

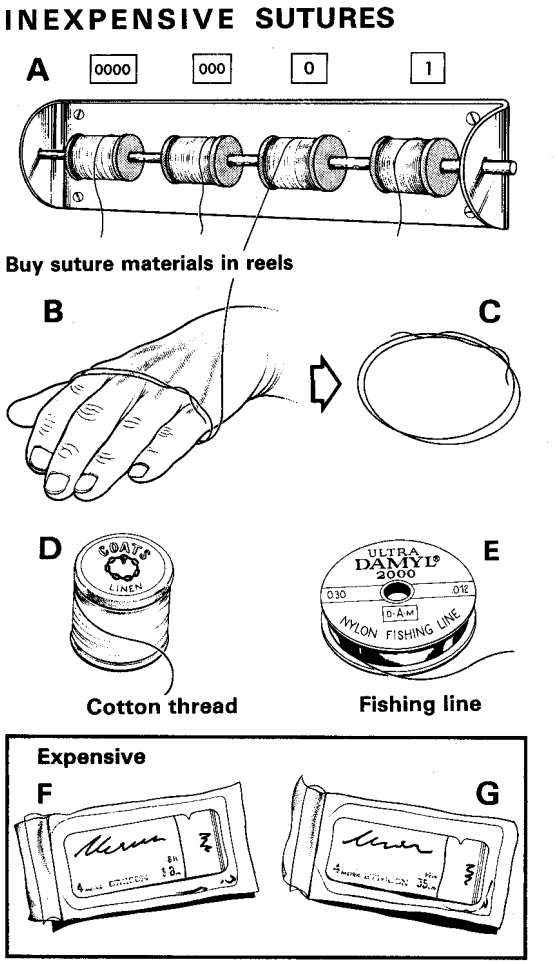


Fig. 4-6 BUY MONOFILAMENT IN REELS.
Hang them from a wall bracket (A), cut lengths of suture material about a metre long and twist them into loose coils (B, C), or wind them round the empty spools used for disposable sutures. D, E, you can also use cotton or linen thread, or colourless fishing line. Match this against surgical monofilament nylon strength for strength. A good strength for abdominal sutures is 12-20lb breaking strength. F, G, if funds are scarce, avoid the expensive proprietary sutures.

Dr JAMES MUKOLAGE was horrified to find in the village a woman with an abdominal wound from which bowel was protruding. He was only recently qualified and had not operated on one of these cases before. He had few facilities, but he managed to find some local anaesthetic solution and some linen thread in the shops. A few instruments from the local health centre were boiled up; he washed the wound thoroughly, and anaesthetized the tissues round it with lidocaine. Fortunately, her bowel had only a minor cut in it which was easily repaired. When he had returned her bowel to her abdomen he was able to close it with linen thread. She survived.
LESSON Improvisation can save lives.

MONOFILAMENT IS THE MOST USEFUL GENERAL-PURPOSE SUTURE MATERIAL

Never let the lack of suture materials be the reason for not doing an urgent operation. Ordinary nylon fishing line, which is exactly the same material as that used for surgical sutures, can be used. Thread this through a hollow sterile needle of correct size, snap off the plastic Luer lock, and crimp the metal of the needle tight onto the thread to secure it. Bend the needle to whatever shape you want and use it with a needle holder.
Here are comparative suture sizes related to fishing wire breaking strengths:

Non-absorbable suture size, U.S.P. (and metric)	*Diameter limits (mm)*	*Fishing line size by breaking strength (in pounds) and approximate diameter*	*Uses*	*Improvised syringe needle gauge and inner diameter (mm)*
6-0 (0.7)	0.070 - 0.099	N/A	Face, blood vessels	30g (0.140) *or* 31g (0.114)
5-0 (1)	0.100 - 0.149	1 lb (0.12-0.14 mm)	Face, neck, blood vessels	28g (0.191) *or* 29g (0.165)
4-0 (1.5)	0.150 - 0.199	2-4 lb (0.15-0.20 mm)	Neck, hands, limbs, tendons, blood vessels	26g (0.241) *or* 27g (0.216)
3-0 (2)	0.200 - 0.249	6 lb (0.22-0.26 mm)	Limbs, trunk, bowel, blood vessels	23g (0.318); 24g (0.292) *or* 25g (0.267)
2-0 (3)	0.300 - 0.339	8-10 lb (0.30-0.33 mm)	Trunk, fascia, viscera, blood vessels	22g (0.394)
0 (3.5)	0.350 - 0.399	12-14 lb (0.32-0.39 mm)	Abdominal wall closure, fascia, muscle, drain and line sites, bone	20g (0.584) *or* 22g (0.394)
1 (4)	0.400 - 0.499	15-20 lb (0.40-0.48 mm)		20g (0.584)
2 (5)	0.500 - 0.599	25-30 lb (0.50-0.58 mm)		18g (0.838)
3, 4 (6)	0.600 - 0.699	N/A		18g (0.838)
5 (7)	0.700 - 0.799	50 lb (0.70-0.77 mm)		18g (0.838)

N.B. The thickness of fishing line is not necessarily proportional to its breaking strength!

Or, if necessary, you can use ordinary linen or cotton thread almost anywhere, especially as ties. You can likewise buy this cheaply on a reel, and re-sterilize it.

Use 4/0 monofilament as your basic suture material for fine skin sutures.

SUTURES, catgut, plain, 3/0, in boxes of 12. Plain catgut is soft. Use it for suturing the mouth, tongue, and lip.
SUTURES, absorbable, strengths 3/0, 2/0, 0, 1 and 2.
SUTURES, absorbable long-lasting, atraumatic, (a) 2/0 on half circle 30mm needles. (b) 2/0 on 5/8 circle 30mm needles. (c) 4/0 on 16mm curved needle. These sutures have needles swaged on to them. Use them for the bowel, the gallbladder, and the stomach, held in a needle holder. The smaller needles (c) are for children.
SUTURES, prolene, atraumatic, (a) 4/0 on 16mm half circle, round-bodied needles, (b) 8/0 on 3mm 3/8 circle atraumatic needles.
SUTURES, linen, #1. Use linen for tying vessels. It holds knots well and is stronger than cotton.
SUTURES, nylon or virgin silk, 8/0. These are for suturing the cornea.
WIRE, monofilament, soft stainless steel, (a) 5/0, (b) 0.35mm, (c) 1.0mm. Surgical wire must be soft and malleable because springy wire is difficult to work with. Autoclave the whole reel.
(a) Fine 5/0 wire is cheap, and is excellent for the skin, if you can use it efficiently.
(b) 0.35mm wire is for wiring the teeth and for hemicerclage.
(c) Tension 1.0mm wire in a stirrup and use it for exerting traction.
These wires and the equipment to use them are essential. One of the advantages of wire is that, unlike more massive pieces of metal, it does not promote infection, so that you can if necessary put it though infected tissues. You can wire tissues in the presence of sepsis; for example, when you repair a burst abdomen (11.14).
Fasten wire by passing its ends through any convenient tube, such as that from a ball pen, and then grasping the ends. Finally, cut the twisted ends of the wire short. This will prevent it from coiling up in an inconvenient way.
WALL BRACKET, stainless steel, to hold rolls of monofilament (4-6). Fix this to the wall, and pull lengths of monofilament from it. If you cannot get one of these brackets, make it.
REELS, stainless steel, egg-shaped ('eggs'), for holding suture material. Wind monofilament into these, autoclave them and cut off the length of suture material you require.
CRIMPING PLIERS, for bending needles.

4.7 Needles and their holders

Needles can be round-bodied, taper-pointed, or they can have cutting edges. They can be thin or thick, large or small; straight, J-shaped, or curved into ¼, ⅜, ½, or ⅝ of a circle. Curved needles are for working in confined spaces.

Use a ⅜ circle needle in a shallow space, and a ⅝ needle in a deep one.

The narrower and deeper the space, the smaller and more curved the needle has to be. If necessary, you can try to bend a half-curved needle into a ⅝ circle. To economize on commercially produced sutures, you can use fishing wire (4.6).

A needle can have an eye, or the suture material can be fixed to it to form an atraumatic needle. These are expensive, but they make smaller, neater holes, because the suture material is not doubled through the extra thickness of the eye. Use atraumatic needles to suture bowel, the urinary tract, blood vessels, nerves, the cornea and the face, especially the eyelids. These commercially available sutures are much easier to use. It is worthwhile trying to get them through donations. Though they have a commercial expiry date, their reliability lasts at least 12-24 months past this date.

Always use a cutting needle for the skin, either a straight, half-curved or a large curved one held in your hand, or a smaller curved one held in a needle holder. Use a cutting needle for tough fascia. Mayo's needle is a hybrid: it has a trocar point and a curved round shank. Use it for big wide vascular pedicles and tough tissues, such as ligaments. Use round-bodied or taperpoint needles for most other tissues, because of the danger of needle stick injuries. Re-sharpen cutting needles on a stone (4-3).

You will want a needle holder to hold small needles and suture in a confined space. Use a holder with a short handle near the surface, and a long one deeper inside. Use big needles in big holders, and small needles in small holders. A large needle can break a fine needle holder such as Derf's, so treat it with care. Needle holders can have plain jaws, or tungsten carbide inserts which prevent the hard steel of the needles wearing them away. These cost twice as much, but last more than twice as long. Quality counts in needle holders, so get good ones. Hold the needle at the middle of its curvature at the very tip of the needle holder, and follow the curvature of the needle when you draw the suture through the tissues.

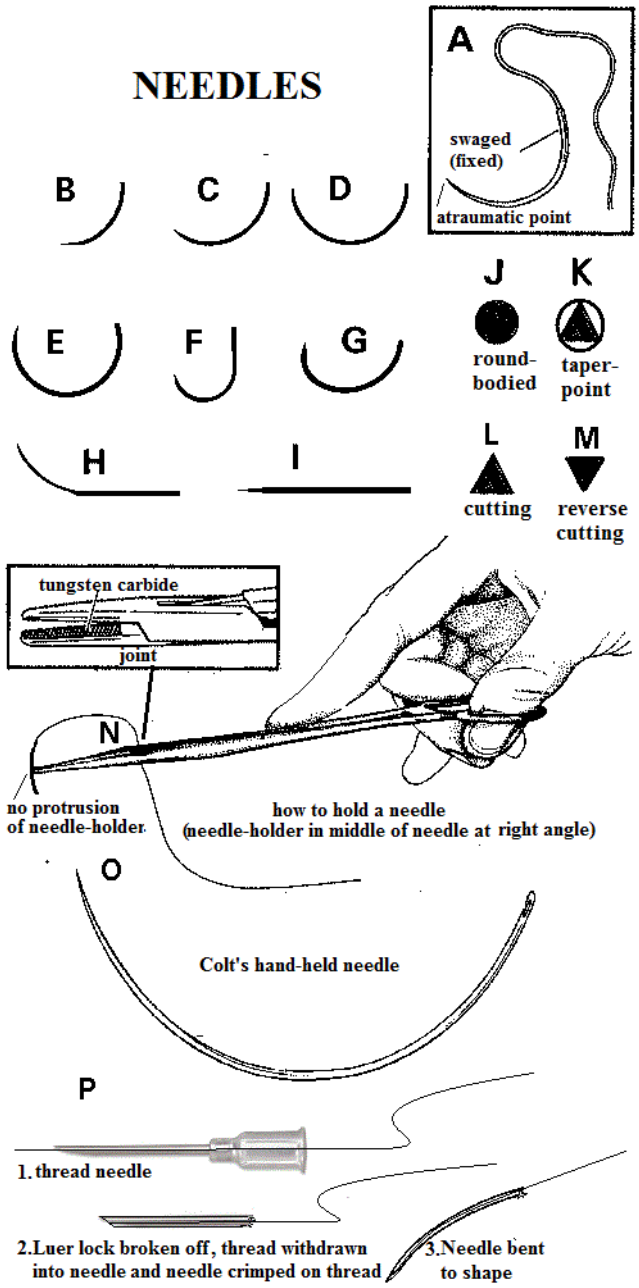


Fig. 4-7 NEEDLES.
A, atraumatic suture with a needle swaged on to it especially for suturing bowel, vessels etc. B, ¼ circle. C, $^3/_8$ circle needles. D, ½ circle needle for suturing soft tissues such as the broad ligament. E, $^5/_8$ circle needle for suturing deep in a wound. F, J-shaped needle for access into a small deep wound. G, compound curved needle. H, half-curved needle. I, straight needle, especially for subcuticular skin closure.
Needles can be round-bodied (J), taperpoint (K), cutting (L), or reverse cutting (M).
N, the best needle holders have tungsten carbide tips. Hold a needle where its cutting edge joins the shaft, with no part of the jaws protruding. O, Colt's large hand-held curved needle. P, economize on sutures by using fishing line (4.6), thread this through an injection needle, break off the plastic Luer lock, withdraw the thread into the needle, and crimp it tightly onto the thread. Bend the needle to the shape you want.
Partly after Remis R, Improvisation of medical equipment in developing countries, Trop Doct 1983; 13(2):89.

NEEDLES, suture, Keith, triangular straight 64mm. This is the standard straight, handheld needle for stitching skin. It is easy to sharpen and 1 needle may last you a year.
NEEDLES, suture, $^3/_8$ circle, curved, triangular point, size 4, 12 and 18. These are the standard curved needles. Hold the largest ones in your hand and the smaller ones in a holder.
NEEDLES, suture, $^1/_2$ circle curved, triangular, size 2, 8, 14 and 20. Use these strong, triangular cutting needles for the scalp.
NEEDLES, suture, round-bodied, $^3/_8$ circle curved, size 4, 10 and 18. Use these for suturing soft tissue such as the peritoneum and broad ligament.
NEEDLES, Moynihan, $^5/_8$ circle curved, round-bodied, fine, size 1, 4 and 6.
NEEDLES, Mayo, intestinal, round-bodied, half circle curved with sharp perforating ends, 23mm, size 20. Use this small curved needle in a holder.
NEEDLES, suture, round-bodied, ½ circle curved, size 1, 4, 10, 15 and 20. Hold these in a holder and use them in the depths of a wound.
NEEDLES, suture, Moynihan, Lance point, $^5/_8$ circle, 115mm. Use these large curved needles for sewing up the abdomen (11.8).
NEEDLES, suture, curved, tension, Colt, 102mm. This is a very large curved needle used for tension sutures into the abdomen (11.8).
NEEDLES, straight triangular, cutting, 35mm. Hold these in your hand and use them for suturing tendons.
NEEDLES, suture, Jameson Evans, triangular, curved, 10mm. These small curved needles have flattened shafts, triangular points and lateral eyes. Use them for delicate sutures, such as repairing the eyelids.
NEEDLES, suture, Dennis Brown, round pointed, $^5/_8$ circle, 16mm. Hold these small curved needles in a needle holder, when you are working at the bottom of a narrow deep hole, such as the bottom of a burr hole.
NEEDLES, suture, $^1/_2$ circle, catgut, Mayo, size 1 and 3. These are strong needles for tough tissues. They have short cutting edges, so you can use them to repair an artery.
NEEDLE, Deschamps, angled to the right. This is the only needle (not illustrated) in this list which you can use to thread wire, to close the abdomen (11.8), or to wire the patella.
NEEDLE HOLDER, Boseman, 210mm, ratchet and box joint, tungsten carbide jaws. This is the standard needle holder for medium and large needles.
NEEDLE HOLDER, Mayo's, with ratchet and box joint, tungsten carbide jaws, 185mm.
NEEDLE HOLDER, Mayo Dunhill, 160mm, ratchet and box joint, tungsten carbide jaws.
NEEDLE HOLDER, Mayo's with narrow serrated jaws, box joint, tungsten carbide jaws and ratchet, 185mm.
NEEDLE HOLDER, Derf, box joint and rachet, tungsten carbide jaws, 115mm. This is an expensive fine needle holder for tiny needles.

4.8 Suture methods

(a) Suturing wounds (GRADE 1.1)

There are 2 kinds of wound to suture:
(1) Those caused by trauma.
(2) Those which you make yourself when you operate. You can suture both in much the same way. Here, we are mostly concerned with the skin; the special sutures for other structures are described elsewhere: arteries (3.2), and bowel (11.3).
Remember, when you suture wounds, you are simply approximating tissue and skin edges. It is not your sutures which promote healing, but the body's own repair mechanisms. So, *don't tie your sutures too tight;* this causes ischaemia and ultimately tissue death, not healing. Place your sutures accurately and neatly to produce a scar as near invisible as possible. *Put the patient in as comfortable a position as possible so he does not fidget while you suture!*

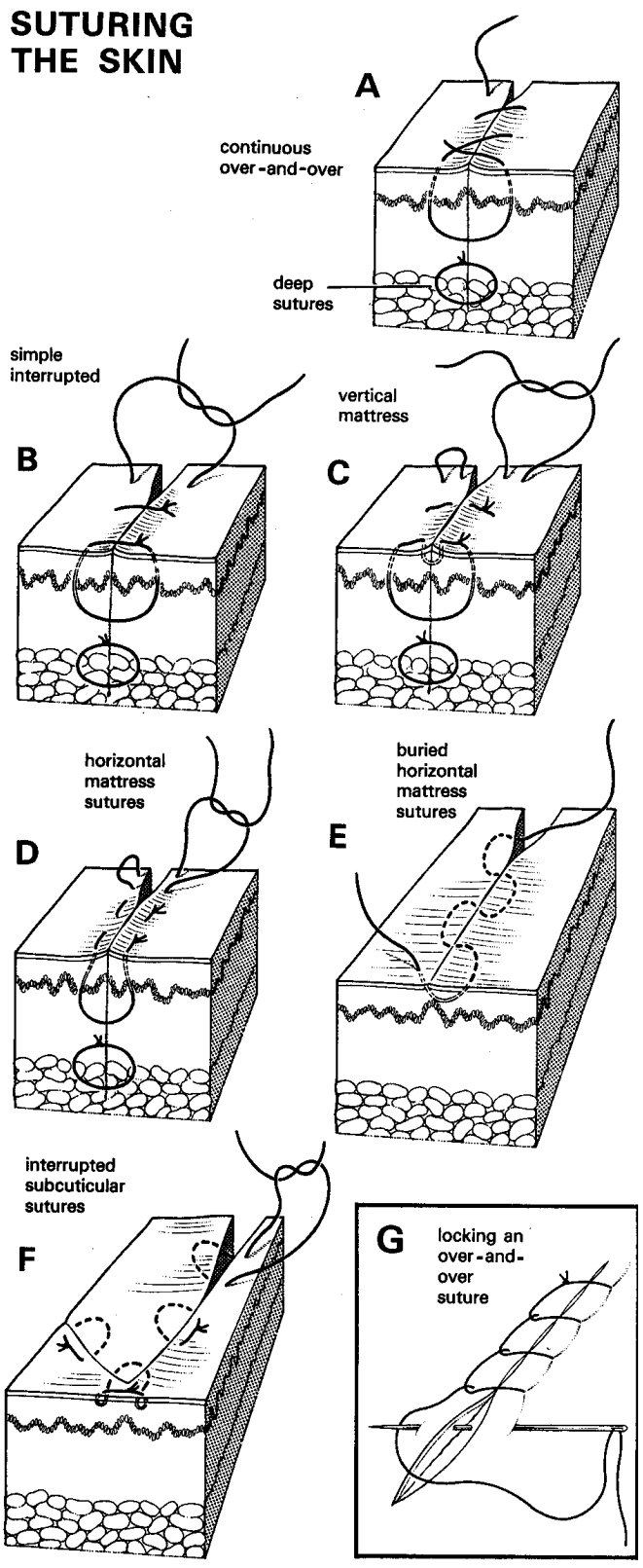


Fig. 4-8 SUTURE METHODS FOR THE SKIN.
A, continuous over-and-over suture. B, simple interrupted suture. C, vertical mattress suture. D, horizontal mattress suture. E, buried horizontal mattress suture. F, interrupted subcuticular suture. G, continuous over-and-over suture which is being locked.
After Grabb MD, Smith JW, Plastic Surgery, Little Brown 3rd ed 1979 Figs 1-8, 1-9, with kind permission.

(1) **'Over-and-over' sutures** are the most commonly used, and can be continuous (4-8A) or interrupted (4-8B). Each interrupted suture needs its own knot; each knot can act as a nidus for infection; and each takes time to tie. So continuous sutures are quicker, but they are also less reliable, because, if the knot on a continuous suture unties, or the suture breaks, or it cuts out, the whole wound may open up. The loss of a single interrupted suture, however, usually matters little. A beginner usually finds interrupted sutures easier. If you wish, you can lock a continuous skin suture to make it more secure; you can lock every stitch (4-8G), or every few stitches.
(2) **Vertical mattress sutures** (4-8C) take a superficial bite to bring the skin edges together, and a deeper one to close the deeper tissues; so they are useful for deeper wounds, but they leave scars: they are usually interrupted.
(3) **Horizontal mattress sutures** may be interrupted (4-8D) or continuous, superficial or buried (4-8E), and are merely alternatives to 'over-and-over' sutures without any special merit, except that they are better at everting the skin edges. *Don't bunch together the skin edges tightly*: gentle approximation is all you require.
(4) **A subcuticular (or intradermal) suture** brings the skin edges together accurately, and is particularly useful in plastic surgery. By not puncturing the skin, it probably leads to less wound infections. It can be interrupted (4-8F) or continuous (4-8G). If it is continuous, anchor both ends using a knot internally, or leave the end long. (*Abandon the use of threaded beads because of the danger of needle-stick injury.*)
(5) **The simple mattress suture** (4-9G) is different from the 'figure of 8' suture (4-9H). Use this to stop bleeding from soft bulky tissue when there is no obvious vessel to tie. This can occur, for example, when you have closed the uterus after Caesarean section with the usual 2 layers of sutures and the wound is still bleeding at one end.
(6) **Skin staples** (4.11) are quick and easy to insert to approximate skin edges, and leave little scarring, but need a special clip-remover to get them out easily.

(b) Knots and sutures

(1) **Suturing**. Hold a straight needle in your hand. Hold a curved one in a holder about half of its length from its end, with no part of the needle holder protruding beyond the needle.
You will also have to hold the tissue you are sewing. Hold a hollow viscus, such as stomach or bowel, with plain forceps; hold skin or fascia with toothed ones. If the needle is curved, move the holder through an arc, so as to follow its curve.
In the skin, insert the needle a regular distance from the edge of the wound, and place sutures regularly. Include an equal amount of skin on each side of the wound.
Set knots down so that they lie square, and *don't tie them too tight*: just tight enough to bring the skin edges together. The skin will swell during the following day, and if the knots are already tight, they will become even tighter and impair the circulation, leading to necrosis.

CAUTION!

(1) *Don't insert the needle at different depths*, because the edges of the wound will overlap.

(2) *Don't leave dead spaces*, or they will fill with fluid which may become infected.

(3) Suture towards you.

(4) When you suture 2 tissues together, one of them may be mobile and the other fixed (because you are holding it). Suture from the mobile tissue towards the fixed.

(5) Continue in the curve of the needle.

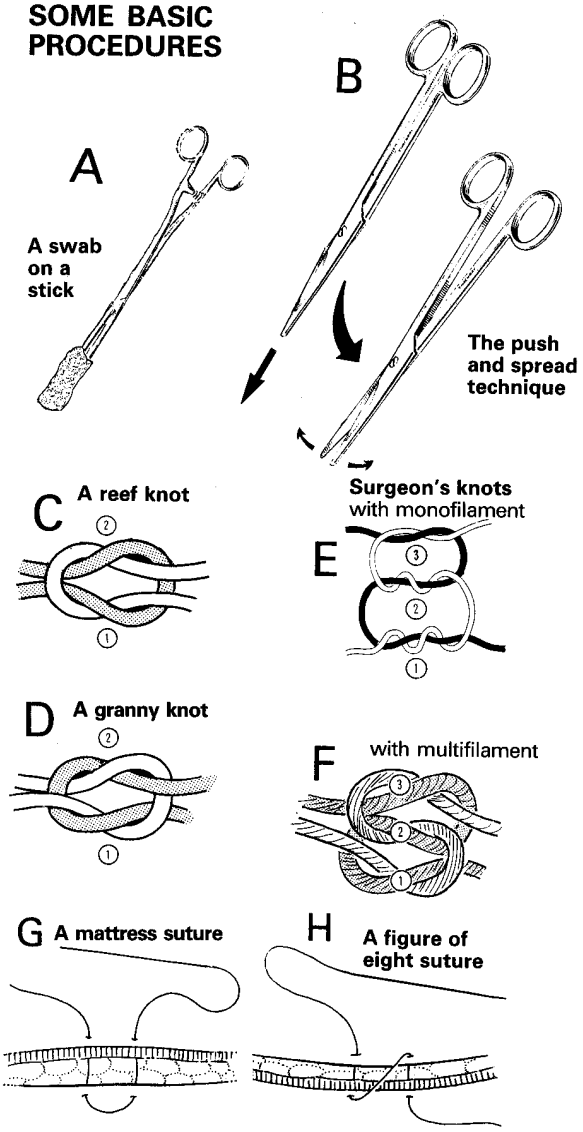


Fig. 4-9 SOME OF THE BASICS.
A, sponge holder grasping a swab ('a swab on a stick') can be a useful instrument for dissecting delicate structures, as when separating the peritoneum from the vagus nerves (13-13). B, as well as cutting with scissors you can push them into the tissues and then gently open them to spread structures apart. This is the 'push and spread technique'. Be gentle! It is useful for tissue planes, but forceful spreading can injure thin walled structures, such as veins. C, a 'reef' or 'square' knot. D, a 'granny knot' which does not hold so well. E, a surgeon's knot for monofilament has three hitches (or 'throws') with 2 turns (or more) on the first 2 hitches and 1 turn on the 3rd. F, a surgeon's knot with multifilament is less likely to slip and need only have a single turn on each of the three hitches. Note that each hitch should ideally make a reef knot with the previous one. G, a mattress suture. H, a 'figure of 8' suture, which is like a mattress suture, except that the needle is inserted in the same direction both times. *Don't use this on the finger.*

(2) **Knots**. Tie reef (square) knots, *not 'granny knots'*. These are both made from 2 half hitches; in a reef knot they go in opposite directions, in a granny knot they go in the same direction. Pull equally on both ends, pull horizontally, and watch the knot go down. If one end is tense and the other loose, you will get a slip or sliding knot.

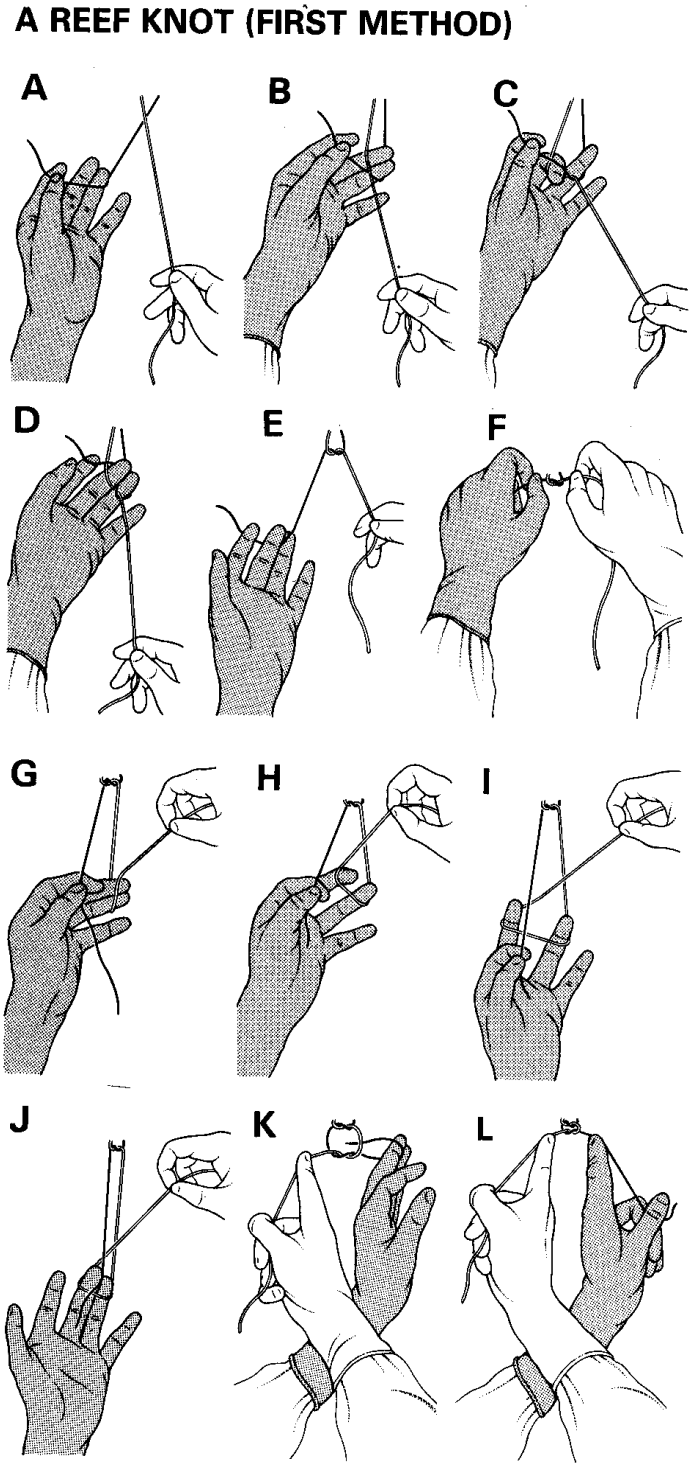


Fig. 4-10 TYING A REEF KNOT: 1ST METHOD.
The standard method without using instruments. Difficult steps are C, and D, in which you grasp one of the ends between your middle and ring fingers, and I, and J, where you do the same again.

A surgeon's knot is merely a reef knot with a 3rd half hitch in the same direction as the 1st one. This 3rd half hitch makes the knot less likely to undo. Some surgeons tie 3 hitches in all suture materials.

Some suture materials undo more easily than others. Non-absorbable multifilament makes the safest knots. Knots of braided suture seldom undo, but knots of monofilament undo much more easily. So either use a surgeon's knot or at least 4 hitches when you tie monofilament. For important knots put ≥2 turns on the 1st and 2nd hitches. With multifilament a single turn is enough on each hitch.

Practise these knots with string or your shoelaces, until you can do them quickly, and do them blind. Learn the various ways of doing them in the following order.

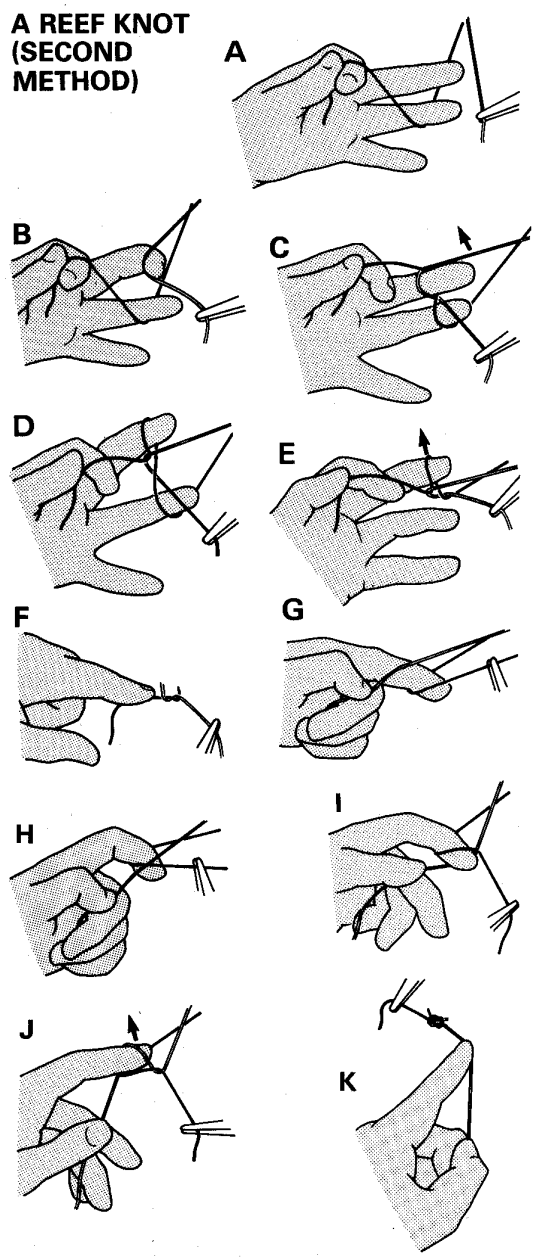


Fig. 4-11 TYING A REEF KNOT: 2ND METHOD.
This method is similar to 4-10 except that you are using forceps in your right hand. Use it if you are working in a hole.

Reef knots can be tied in several ways. The 1st method (4-10) is the surest way of tying a knot and is the one to use if you want to exert continuous pressure while you tie. In the 2nd method (4-11), use forceps in your right hand. The 3rd (4-12) is an 'instrument tie' and is useful if one end of a suture is short, or if the knot is in a deep cavity. The short end can be quite short. First, make a loop with the instrument in front of the long end. Grasp the short end and pull it through this loop. Then pull the first half hitch tight in the plane of the knot. To make the second half hitch, start with the instrument behind the long end.

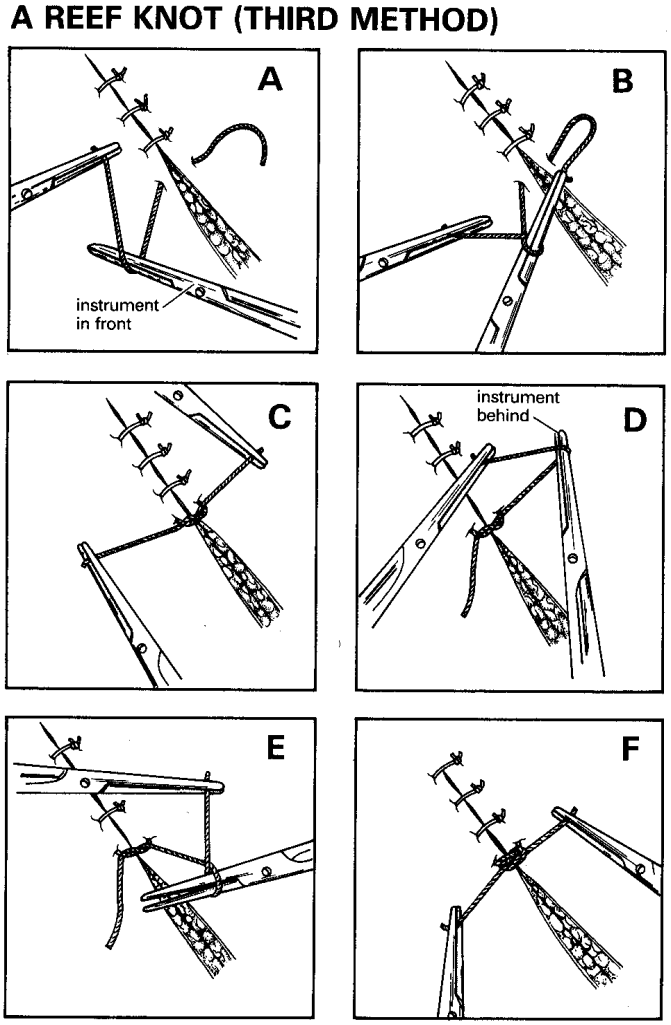


Fig. 4-12 TYING A REEF KNOT: 3RD METHOD.
If there is not enough room for your fingers, use forceps in each hand. This is an 'instrument tie'. Notice that for the 1st half hitch the instrument is in front of the long end (A), and for the 2nd one it is behind (D). In this way you will tie a reef knot, *not a 'granny' knot.*

TO CUT A SUTURE, almost close the scissors, slip their open ends over the suture material, and move them gently down towards the knot. Twist the tip to give you the length of tail you want, then cut. Cut the tails of interrupted skin sutures short enough to prevent them tangling in the next suture. Leave buried catgut sutures with 5mm tails, others with 2mm tails. Cut buried sutures close beside the surgeon's knot.

CAUTION! Keep the tips of the scissors in view, and *don't cut unless you can see what you are cutting*.

An Aberdeen knot (4-13) is a useful method to secure a mass closure of the abdomen (11.8); hold the suture in a loop and pass successive loops of the suture through the 1st loop, c. 4-6 times, and then pass a single strand finally through the loop. Pull this taut, thus taking the slack off the loop, and creating the knot. You can then bury the suture end by taking a bite through adjacent tissue.

Fig. 4-13 AN ABERDEEN KNOT.
This is a very secure knot, especially useful for securing an abdominal closure.

REMOVING SUTURES. Leave them until the wound has healed adequately. Some sutures can be removed on the 2nd day, others not until the 14th. Remember the function of sutures is simply to approximate tissues, not to tie edges together! Here is a guide:

Skin sutures on the face and eyelids	2-3 days
The tongue	4 days
The scrotum	5 days
The scalp	6-7 days
The arm, hand and fingers	7 days
The abdomen: transverse incision	7-9 days
vertical incision	9-11 days
The skin of the back over the shoulders	11-12 days
The skin of the legs	14 days

N.B. Add 50% to these values for patients taking corticosteroids or cytotoxic medication.

When you remove a suture, try not to pull any part of the suture material which has been on the surface through the tissues, or you may contaminate the wound. Clean the skin, cut the suture where it dips under the skin with sterile scissors or a blade. Remember that after 3wks a wound has only 15% of the strength of normal skin, at 4 months 60%, and only full strength at 1yr.

4.9 Drainage tubes

Inserting a drainage tube may be the principal aim of surgery, as when you drain the pleural cavity (36.1), or it may merely be part of an operation, as in decompressing the stomach when the bowel is obstructed (12-4, 12-6). You can also use tubes to drain pus and exudate. The insertion of a tube for gastrostomy (13.9), jejunostomy (11.7), caecostomy (11.6), and cholecystostomy (15.4) are described elsewhere: first we describe the use of nasogastric tubes, which are of great value, even though they are a burden to nurses and an irritation to patients.

TUBE, nasogastric, plastic, Ryle's, with several side holes near the tip, Ch14, Ch16, Ch18. Transparent plastic tubes are better than rubber ones, because they are less irritant, they don't collapse, and you can see what is inside them. Most tubes have markings, the first at 45cm showing that the tip is about to enter the stomach, and the second that it is in the antrum.
TUBE, stomach, plastic, adult and child, assorted sizes, Ch8-22. These are critically important for making sure that a patient's stomach is empty before he is anaesthetized, and for washing it out if he has swallowed a corrosive (30.3). Adults need tubes of Ch16-22, children Ch10-14, and infants Ch8-10.

(a) Nasogastric (NG) tubes

INDICATIONS
(1) To remove fluid from the stomach before anaesthesia, so as to reduce the risk of inhalation.
N.B. The solid food from a recent meal will not come up a small nasogastric tube, so if you want to anaesthetize a patient safely who has recently eaten, or has intestinal obstruction, you will have to empty the stomach with a large nasogastric tube.
(2) To decompress the stomach during upper abdominal surgery or in cases of high intestinal obstruction.
(3) To keep the stomach empty after a laparotomy for acute intestinal obstruction and in cases of pancreatitis.
(4) To feed a patient.
(5) To monitor severe gastric bleeding.
For all these reasons, it is good practice to pass a tube whenever you do an emergency laparotomy. *It is, however, not necessary with simple cases of appendicitis, cholecystectomy, or elective bowel resection and most gynaecological procedures.*

CONTRAINDICATIONS
(1) If you suspect oesophageal varices.
(2) After corrosive injury of the oesophagus where the mucosa is friable and easily perforated.
(3) Where there is severe respiratory embarrassment (a gastrostomy (13.9) is better).
(4) A deformed or blocked nasal passage.
(5) If you suspect a basal skull or cribriform plate fracture: *the tube may penetrate into the brain!*

PASSING A NASOGASTRIC TUBE

Lubricate the tip of the tube with a water-soluble jelly. Sit the patient up and tell him what you are going to do. Choose the nostril which has the widest channel. If he is agitated, spray the nostril with lidocaine. Pass the tube horizontally through the nose. When the tube touches the posterior pharyngeal wall, he will gag, so give him a little water to sip, as you slowly advance the tube. The act of swallowing will open the *cricopharyngeus* and allow the tube to enter the oesophagus. Continue to advance it until its second ring reaches the nose; its tip should now be in the stomach.

If the tube is too flexible and curls up in the pharynx, put it in the freezer for 2mins and try again.

CAUTION! If you are only aspirating through the tube, you cannot do much harm, but *never start tube feeding until you are sure a tube is in the stomach*. You can easily pass a tube into the trachea of an elderly, debilitated, or unconscious patient and drown him with feed.

To make sure the tube is correctly placed in the stomach:

(1) Aspirate greenish-grey stomach secretions and test these with blue litmus paper, which should turn red.

(2) Inject a little air down the tube and listen over the stomach with a stethoscope for a gurgling sound.

(3) Listen to the end of the tube. The sound of moving air confirms that the tube is *not* in the stomach, but is in the trachea or bronchi.

When you are satisfied that the tube is in the right place, secure it with 2 narrow strips of tape, one on the side and the other on the bridge of the nose, extending downwards on to the tube. In this way you will avoid pressure necrosis of the *alae nasae*.

Connect the tube to a bedside drainage bottle or plastic bag, to let the stomach contents syphon out. Assist this by aspirating. Suck the contents out hourly, or more frequently if there is much aspirate, to prevent the tube blocking. If you cannot aspirate anything, try irrigating the tube with 5-10ml of water; its terminal holes may be plugged. *Never clamp the tube!*

If the tube fails to decompress the stomach:

(1) Its tip may still be in the oesophagus.

(2) It may be kinked or blocked.

(3) The stomach may be filled with large food particles.

(4) Excessive suction may have sucked food or mucosa into the holes in the tube and blocked it.

Occasional sips (not gulps) of water will help to ease the patient's misery. Keep a fluid balance chart, and as a general rule replace gastric aspirate by IV 0·9% saline or Ringer's lactate.

CAUTION! If you don't care for the mouth adequately, the parotid may become infected. So, arrange 4hrly mouth care as a routine after major surgery, especially if there is a nasogastric tube *in situ*.

REMOVING A TUBE

As a general rule, leave a tube in place until:

(1) There is no abdominal distension.

(2) There is no longer any nausea.

(3) The bowel is active normally, indicated by the passage of flatus. If there are only c.400ml gastric aspirate daily, this is the normal volume; if you aspirate ≥750ml, suspect ileus or bowel obstruction.

CAUTION! Don't remove a nasogastric tube if the patient is nauseated, or the abdomen is distended and he has passed no flatus, or has >500ml of gastric aspirate od. If he has any of these, he probably has paralytic ileus, obstruction (12.15), peritonitis (10.1), or an anastomosis that is too narrow. However, if the tube has migrated down into the duodenum, it will continue to produce large volumes: withdraw it then by 10cm and observe the effects.

DIFFICULTIES WITH NASOGASTRIC TUBES

A patient who is very weak, dehydrated or shocked may vomit through the act of passing a tube and inhale the vomit. If so, lie him on the side, with the head tilted down, and pass a large stomach tube (Ch30). If he vomits, he will now do so under controlled conditions. Afterwards, pass the nasogastric tube.

If pulmonary complications develop, these may partly be caused by the discomfort of the tube through:

(1) causing ineffective coughing, and

(2) drying out the mouth by making nose breathing difficult.

If the nasal cartilages necrose (rare), you applied tape unwisely. Pressure is usually caused by an acute angulation of the tube. A debridement of the dead tissue will be necessary.

If oesophageal erosions develop, you may have been using too hard a tube. Also, a large tube may allow regurgitation through the cardiac sphincter and cause an erosive oesophagitis.

(b) Other drains

Not all wounds need drains, and drains have their risks:

(1) Bacteria may enter from outside, especially if nursing care is poor. This risk is small if you use a closed drainage system.

(2) Bacteria may come from inside a patient and infect the tissues through which the drain passes, particularly the abdominal wall.

(3) A drain may erode a vessel or a suture line, especially if you leave it in for a week or longer.

(4) A drain may block.

(5) A drain reduces mobility and so delays convalescence.

(6) A drain may knot itself.

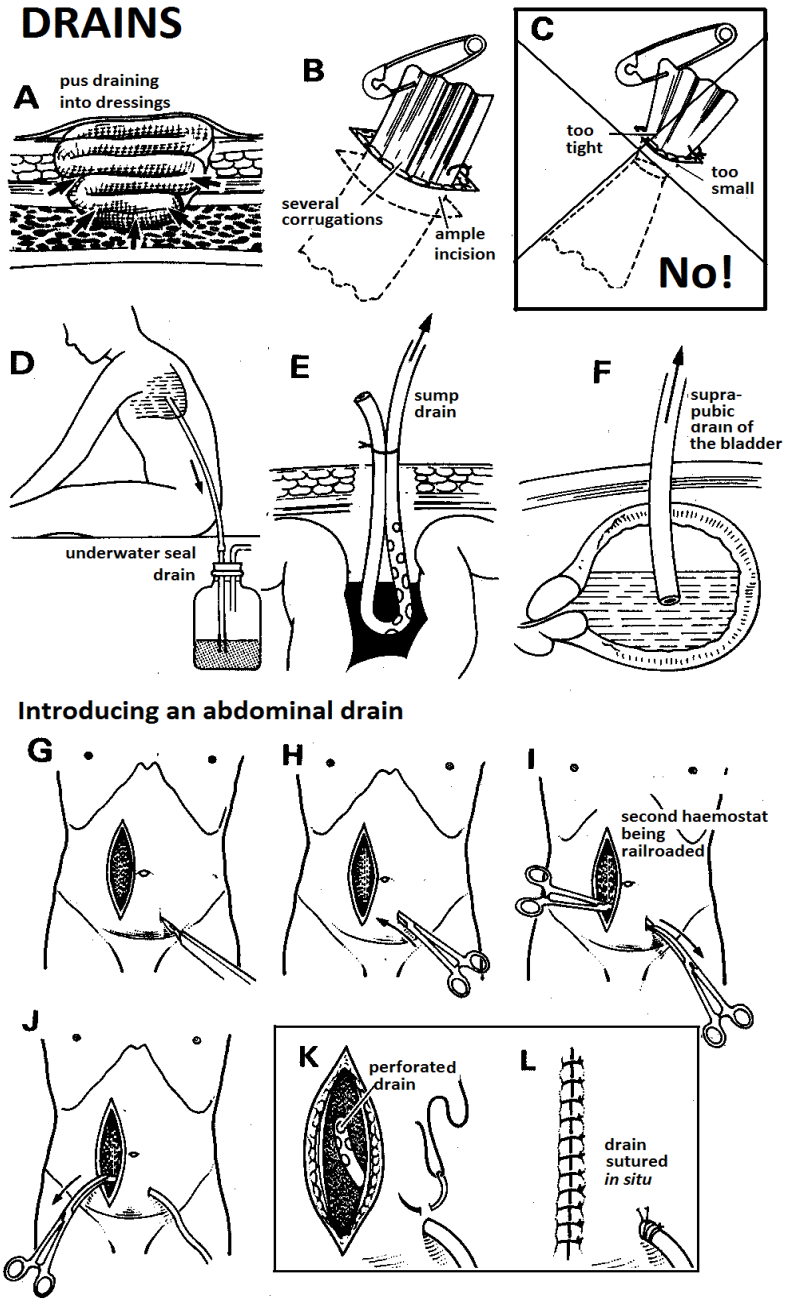
INDICATIONS

(1) To remove blood, serous oozing, or lymph from a loosely confined space (*e.g.* breast, scrotum, neck, wound): in this case a suction drain is most efficient.

(2) To drain urine, bile or pancreatic juice, which may leak from a suture line, or formally to drain the bladder (27.2,6,7,8), gallbladder (15.5), or pancreas.

(3) To drain the pleural cavity (9.1): here you need an underwater sealed drain or special system to prevent air being sucked into the pleural space.

(4) To complete the drainage of an abscess cavity: you can let the exudate flow down a tube, or you can let it seep away round the edge of a Penrose fine rubber drain.
(5) To permit the controlled escape of content from a possibly leaky suture line, for example when you are worried about an extra-peritoneal, *i.e.* oesophageal or rectal anastomosis.



N.B. There is no evidence to support the use of drains in the peritoneal cavity to 'control' secretions. They block or seal off within 24 and 48h anyway, unless kept irrigated. *Don't insert a drain unless there is a good reason to do so*. Therefore *don't drain all wounds routinely;* insert a drain when there is a proper indication to do so. *Drains may actually cause the fistula they are trying to 'control'.*

(1) **Penrose fine rubber drains** are useful for abscesses. Cut more than an adequate hole in the superficial tissues, cut a strip of rubber to fit loosely and push this into the depth of the wound (4-14B). *Don't make the hole for the drain so small that it is tight* (4-14C). Use a cutting needle to transfix it with a suture and anchor it to the skin, then tie the ends of the suture several times. When you shorten a drain, you may be able to leave a loop of suture material securing it. A safety pin will prevent it slipping inside the wound, *but will not prevent it slipping out*.

(2) **Tube drains** are useful in large wounds where you expect much exudate, or in areas of infection (4-14D). They are especially useful in the abdomen (4-14E). Have 2-3 sizes of drainage tubes ready sterilized with suitable adaptors. Use silicone rubber or polyethylene, rather than red or latex rubber, which is more irritant.

Try to use a tube drain with a tight seal which will lead the exudate safely into a bottle, rather than a piece of rubber which will lead it into dressings.
Try to place the drain at the bottom of the cavity to be drained, so that exudate can easily flow out downwards; make it follow a straight path.

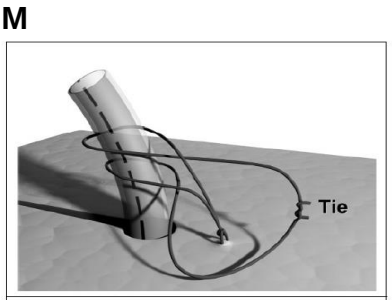


Fig. 4-14 DRAINS.
A, in a superficial wound pus can drain into the dressings. B, a corrugated drain should usually consist of several corrugations and fit loosely through an incision in the superficial tissues. C, *don't push a drain tightly through a small incision*. D, chest drain. E, sump drain. F, draining the bladder.
G-L, introducing a tube drain into the abdomen. G, making the incision. H, inserting the 1st haemostat. I, catching a 2nd haemostat by the 1st and pulling it through out of the abdomen. J, releasing the 1st haemostat and pulling a drainage tube inside with the 2nd. K, holes cut in the end of the drainage tube. L, drain sutured in place and the abdominal wall closed. M, method of fixing the drain so it is held tight.
After Maritz D, McLauchlan C. A novel way to secure a chest drain. Ann R Coll Surg Engl 2014; 96: 82.

Insert the drain through a separate stab incision, not through a sutured wound.
If a drain is in any danger of falling out, stitch it in as it passes through the skin (4-14L).
Keep dressings over the drain separate from the main wound, so the former does not contaminate the latter.
Don't try to drain the whole peritoneum in peritonitis: it is impossible anyway. Instead, wash out the peritoneal cavity (10.1).

Finally, be sure to explain to the ward staff why you have inserted a drain, how they are to manage it, and when they are to remove it.
Make sure, if there is more than one drain, that they are labelled appropriately with a permanent marking pen.

(3) **An abdominal tube drain** should be big enough and in the right place; try to fit a wide bore tube tightly in a small hole. Make a small incision in the skin. Use a 10mm (Ch30) tube, and cut side holes in the end. Make a small hole in the tissues and 'railroad' the drain in (4-14G-J), using a hand to protect the bowel. *Try to do this under direct vision!* Anchor the drain to the skin with a horizontal mattress suture, and place a knot along the free ends of the thread. Wrap the 2 threads tightly round the drain tube up to the level of the knot; then pass both free ends through the loop of the skin suture (4-14M). Now separate the ends and tie these round the tube in opposite directions, and knot them with a reef knot. Finally, tape the drain to the skin. Connect it to a sterile bottle. *N.B. You can use the same suture to close the skin after removing the drain.*

(4) **Suction drains** are ideal, especially the disposable plastic kind. More practical is the reusable 'Redivac' suction bottle type, which has disposable drainage tubes.

(5) **Sump drains** are useful if you have a suction pump and you want to drain fluid such as urine or pancreatic juice which is welling up from the depths of a wound.

THE TIME TO REMOVE A DRAIN varies with the fluid to be drained. Here are some guidelines:

Blood	48-72h
A suspect bowel anastomosis	5-7days
A septic cavity	5-7days usually
Bile, pancreatic fluid or urine	10days

.

Don't leave a drain in longer than is necessary, because you run the risk that it may erode a vessel. There is seldom any need to leave a drain >2wks at the most, except in a very large deep abscess. If you remove a drain too early, however, pus may build up and seek to discharge itself elsewhere.
If a drain is long, shorten it progressively over several days before you remove it. Shorten it by pulling it out, not by cutting it off. Place a safety pin through it and tape this to the patient's skin.

DRAIN, corrugated red rubber, sheets 1x50x300mm. Pus drains between the corrugations. Cut the sheets to make drains of various shapes and sizes. *Don't discard used sheet rubber drains:* wash them, boil them, and store them in antiseptic solution (2.6). For tiny drains, cut up old intravenous sets or gloves.
TUBING, red, rubber sterilizable, 2mm wall, (a) 10mm bore, (b) 15mm bore. This is multipurpose tubing, the 10mm size is for draining air and blood, the 15mm size is for pus. The firmness of the wall of a drainage tube is important. The tube from a chest drain should be firm enough to ensure an open pathway through the chest wall. The abdominal wall is less likely to pinch a drain closed, so a firm drainage tube is less important. If necessary, use a large bore catheter.
SUMP DRAIN, rubber or plastic. In an ordinary drain the holes through which fluid is sucked frequently block. A sump drain overcomes this difficulty by having 2 tubes, an outer one with many holes in it, and an inner one through which fluid is sucked. Fluid trickles into the outer tube and is then sucked away down the inner one. Ideally, suction down the inner tube needs to be applied with a low-pressure pump. There should also be a single hole in the inner tube close to the surface to prevent too high a pressure building up in the sump. There are many kinds, and you may be able to improvise one. A sump drain is particularly useful for draining large quantities of fluid from fistulae or a large localized abscess in the peritoneal cavity. Alternatively, use a folded catheter. Suck through one end and let air enter through the other (4-14E).

4.10 Wounds

(a) Dressings
If dressings are in short supply, wash the wound with water 2-4hrly and cover it with a dressing towel (1.11). Gauze will stick to raw wounds, and paraffin gauze is the standard alternative, but is expensive. You can make your own non-stick dressing with liquid paraffin, coconut or red palm oil.

(b) Leaving wounds open post-operatively, where you can, is a useful economy. Do this if a wound is not going to discharge. If it oozes a little, put a thin dressing of gauze or whatever you have on it for 24h. If you do use post-operative wound dressings, *don't routinely change them unless they are wet, soiled, or smelly, or you suspect a wound infection because of a fever.*

(c) Layers of gauze and cotton wool will collect the discharges from a wound which is too shallow to let you insert a rubber drain (4-14A). Change these dressings frequently. If necessary, place a sheet of plastic or waterproof paper between the outermost layer and the patient's clothes. Suction tubes in the wound will help (11-20).

4.11 Stapling devices

A large variety of mechanical devices is available, and you may have some to use; *don't let them gather dust because you think they are too sophisticated for you to use!*

(a) Skin staples
Skin staples are metal clips with fine sharp teeth at both ends; mount them on a special instrument and lift them off with a toothed dissecting forceps of the correct calibre. Bring skin edges for closure carefully together, apply some tension to straighten the wound and crimp the clip with the forceps to hold the edges together. Then repeat the process moving along the wound. Disposable clip applicators exist; these are placed across the opposed skin edges as above, and fired sequentially.

(b) Circular anastomosis gun
The first device to staple together bowel was invented by Russian technicians; the instrument has a safety catch to prevent inadvertent firing. It has a cartridge of 1 or 2 concentric rows of staples mounted on a rod, and an anvil at the end of the spindle.

Insert the gun into the lumen of the organ to be anastomosed, and tie it down with a snug purse-string suture around the rod over the cartridge; put the anvil inside the lumen of the other organ to be anastomosed and tie another purse string to hold that end snug. Fix the spindle of the anvil onto the rod of the gun, and screw them together by turning the end of the handle of the gun, till the desired point is reached (marked on the instrument). This traps the tissues to be stapled between anvil and cartridge. Release the safety catch, and with one firm movement, pull the trigger. This fires the staples across the tissue and simultaneously cuts a central portion of excess tissue away. Release the trigger, and wiggle the instrument out with a gentle twisting movement. Unscrew the instrument: you should find 2 *complete* doughnuts of tissue under the head of the anvil if it has worked properly. An incomplete ring will mean a defect exists, which you then need to close by hand. *This may be very difficult!*

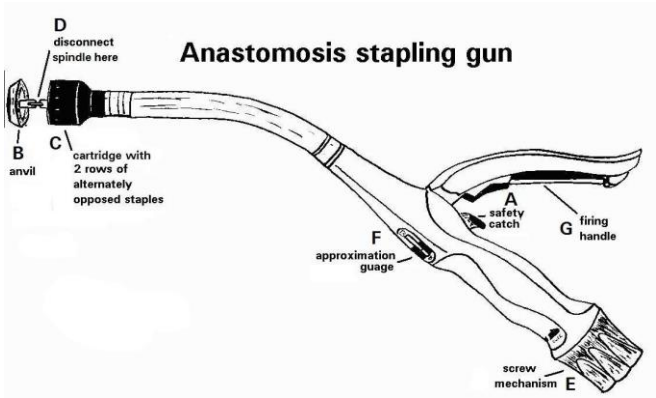


Fig. 4-15 ANASTOMOSIS GUN.
A, safety catch. B, anvil. C, cartridge with staples. D, spindle. E, screw for approximating anvil and cartridge. F, gauge to measure adequate approximation. G, firing handle.

Typical uses of the anastomosis gun are in colorectal anastomosis (12.10) and oesophageal transection (13.6)

LINEAR CUTTING AND STAPLING DEVICES

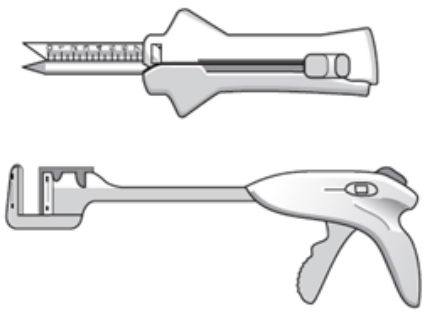


Fig. 4-16 LINEAR STAPLING DEVICES used to divide and close bowel.

(c) Linear resection/stapler devices
The linear stapler inserts 4 parallel rows of staples and cuts between the 2 middle rows; the instrument divides into 2 parts which can be locked together. Place the separate jaws of each half of the instrument inside the bowel lumina to be anastomosed, making sure that no excess tissue is inadvertently trapped between the jaws.

Slide the cutting handle down the full length of the 2 jaws, and separate them. This should create a perfect anastomosis; any defect must be closed by hand. You can then close the remaining open ends with another linear stapler.

Typical use of the linear stapler is in bowel anastomosis, *e.g.* in a right hemicolectomy (11.3) or in a gastrectomy (13.10).

(d) Haemostatic clips
'Ligaclips' are useful for closing blood vessels or ducts deep in the pelvis when ties by hand are awkward because of limited space; apply the clips double with a special instrument for better safety.

4.12 Miscellaneous equipment and materials

Some of the humblest equipment is also the most necessary. Here are many of the things which you should not be without.

TUBES, rectal, rubber, (a) child's size 8mm (Ch24); (b) adult's size 10mm (Ch30). You can also connect these to a large bore funnel and use them to give an enema. Introduce them carefully: you can easily perforate the sigmoid colon.
CONNECTORS, end-to-end, polypropylene, external diameter (a) 4mm, (b) 7mm, (c) 10mm, (d) 15mm, (e) 19mm. Use these to join short lengths of tubing together for suction or drainage etc.
CONNECTORS, plastic 3 way 'Y', assorted sizes.
CLIPS, towel, cross action, 90mm. These are the simplest towel clips.
CLIPS, towel, with ratchet, Backhaus. These are more expensive than the towel clips listed above, but they have several other uses, including holding the sucker tube, and the ribs in chest injuries.
FORCEPS, sponge holding, Rampley, straight, (a) 240mm, box joint. (b) 120mm. Use these for swabbing, and for "swab dissection'.
LOUPE, binocular, Bishop Harman, x2 magnification. Perch its 2 lenses on the very tip of your nose, or wear it over your spectacles. Curl its ear pieces so that it fits your face. This is invaluable for fine operations like repairing nerves or arteries, or 'cut-downs', or removing splinters. The disadvantage of a loupe is that it focuses close to your nose, so use short-handled instruments.
TROCAR AND CANNULA, straight, with nickel silver or stainless steel cannula and metal handle, (a) 4mm (Ch12). (b) 8mm (Ch24). (c) 12mm (Ch36). The small size is useful for tapping hydrocoeles, the middle one for suprapubic cystotomy, and the largest one for chest drainage.
CANNULA WITH SIDE ARM. Attach suction to the side arm and use it to aspirate the gall bladder etc. (15.3).
PROBES, malleable, with eye, nickel silver, 150mm, 3 sizes. Use this to probe perianal fistulae etc.
HERNIA DIRECTOR, Key's. Use this for opening the neck of a hernial sac.
DIRECTOR, probe-ended, Brodie, 165mm. Use this for exploring sinuses.
RING CUTTER. Try, before using this, to remove a ring with soap and string.
NEEDLES, aneurysm, Dupuytren, (a) needle curving right, (b) needle curving left. These are curved needles on the end of a handle. Use them for passing a ligature under something (3-4).
NEEDLES, aneurysm, small, with blunt point. Keep these in your 'cut down sets', and use them to pass ligatures under a vein.
CATHETER, metal female.
BRUSH, for cleaning instrument jaws. The jaws and joints of surgical instruments need brushing regularly. You can also use suede brushes with bronze bristles.
RAZOR, safety, for pre-operative preparation. Shaving a patient pre-operatively is not the essential ritual that it was once assumed to be. You can also adapt a safety razor for skin grafting. Because of the risk of cross-infection, especially of HIV, these should be disposable.
BUCKET, stainless steel, with handles.
KIDNEY DISHES, stainless steel, with half curled edges, 4 sizes 100-300mm.

GALLIPOTS, stainless steel or autoclavable plastic, set of 6 sizes 40-200mm. Use these for lotions, swabs etc.
JAR, stainless steel with dropover lid, 150x150mm. Use these for spirit swabs.
JUG, plastic, autoclavable, conical, 3l. Stainless steel jugs have become standard, but plastic ones are satisfactory.
BIN, soiled.
JELLY, hydroxymethylcellulose, sterile petroleum jelly. This is a sterile non-greasy jelly for catheters etc.
'BIPP', bismuth iodoform and paraffin paste. This is a mildly antiseptic self-sterilizing anaesthetic packing material. You can leave it in the nose for a week without significant infection, or much smell (29.6). If you don't have any, smear gauze or bandage with any non-adherent antiseptic ointment.
CARPENTER'S EQUIPMENT (a) Saw. (b) Twist drill. (c) Hammer, claw head. If you cannot get the surgical equivalents of these, you will find these very useful.
OTHER MATERIALS include gauze, cotton wool, bandages, adhesive tape, and laparotomy pads (1.11).

4.13 Instrument sets

For most operations you will need about 50 general-purpose instruments called 'the general set', with a few special ones when necessary. You can handle additions to the general set in three ways:

(1) You can keep special instruments in the cupboard, and sterilize them when needed. It is useful for the theatre staff that you have cards indicating which instruments you need for which operations. If you don't know in advance what you will need, you can sterilize as many of your basic instruments as you can, lay them out on a sterile towelled trolley, and select immediately before each operation what you will need. You then cover the trolley with a sterile towel till you are ready for the next operation. Obviously, take care not to contaminate the trolley between operations. This method has been very successfully used in Manama, Zimbabwe, where the sterilizer took the better part of the day to heat up!

(2) You can make incomplete special sets, such as a burr hole set or an orthopaedic set, with their special instruments, which you use with the general set when necessary. The advantage of this method is that you will have these special instruments ready when needed in a hurry, and you don't waste time re-sterilizing instruments not required. You can use this method in combination with (1) and (3).

(3) If you have enough instruments, particularly haemostats, you can make complete special sets. This is the best method, and the one which we follow here, but it requires many more instruments, and it is very important that someone trained puts the right instruments in the sets. If this is not possible, revert to system (1).

You can do an occasional emergency operation with only one general set, but when you have a list of patients to operate on, you will need several general sets, if you are not to wait too long between operations. Boiling a set takes at least 15mins, and autoclaving 30mins. A set costs between US$750 and US$1000; about 30% is the cost of the haemostats.

If instruments are limited, start by collecting a general set adapted for Caesarean section and laparotomy, and also the more important special instruments.

Once you have all these, try to complete a chest drainage set, a tracheostomy set, 2 cut down sets, and a 2nd laparotomy set. When you have these, your next objective should probably be a minor set for such operations as wound repairs and circumcisions. If you perform many uterine evacuations, 2 or more sets would be useful.

A Caesarean section is only a particular kind of laparotomy. The set differs mainly in that it includes 2-6 Green-Armytage (or sponge-holding) forceps, and the large round-ended Doyen's retractor, which is specially designed for pelvic operations, replaces Balfour's. (A wide Deaver or Morris retractor is an alternative.)

The sets below mostly start with 6 towel clips and a towel holder, which you can also use to hold the sucker tube. Next come 4 Rampley's sponge holders, the first 2 of which are used for preparing the patient's skin, after which they can be used to hold towels. The remaining 2 are for 'swabs on sticks', and for swab dissection. Then come toothed and plain dissecting forceps, 2 scalpel handles, and a heavy and a light needle holder. There are also 4 pairs of Allis tissue forceps, and various retractors, depending on the set. The expensive items, because of the large number you need, are the haemostats, straight, curved, big, and small, clipped together in groups of 6 on Mayo's pins. The more experienced you are, the fewer of these you will need. We list 6 of each, which is a generous number for a beginner. Finally, there is the Pool's sucker and its tube; this is a perforated suction tube which does not suck up bowel. *Don't use haemostats as towel clips!*

Keep an inventory of equipment and a checklist for each set posted where the set is packed and stored. Nice instruments tend to disappear. One aid to keeping instruments together is to provide them in pairs, or in even-numbered quantities where possible. For example, the nurses will find it useful to remember that haemostats and towel clips should always be in half-dozens.
The theatre is the best place in the hospital for sterilizing equipment. So try to develop a simple 'central sterile supply' service which can prepare sets for the wards.

INSTRUMENT SETS You will want the following sets, some of which are described elsewhere: a uterine evacuation set (2 if possible), a general-purpose set (preferably 2 sets), a Caesar set, a cut down set, an abscess set, several minor sets (for hernias, etc.), an orthopaedic set (for drilling for osteomyelitis, etc.), an intestinal clamp set (for resecting bowel), a fine instrument set (for hand surgery), an eye set (28.1), a burr hole set, a chest drain set, and a tracheostomy set.

SHARP EQUIPMENT needs to be kept separately, because it gets blunt if it is autoclaved too often. Keep scissors separate from other instruments. Keep osteotomes and gouges in a cupboard and put them in sterilizing fluid 30 minutes before you use them. Autoclave the bone saw when you want it. Keep the bone drill and the twist drills to go with it in a special sterile pack.

CAUTION! Always re-autoclave the packs and drums regularly. A pack which has not been re-sterilized for some time is a risk, especially if it is only covered in towels. You may find termites inside it!

THE CONTENTS OF PARTICULAR INSTRUMENT SETS

THE GENERAL SET (including the instruments for laparotomy).
6 towel clips.
1 Backhaus towel forceps.
4 Rampley's sponge holders.
1 toothed dissecting forceps (Treves).
1 plain dissecting forceps (Bonney's).
1 #4 & 1 #5 scalpel handle.
2 needle holders, a heavy and a light.
2 Allis tissue forceps.
2 Lane's tissue forceps.
6 200mm curved haemostats (Spencer Wells).
6 120 or 140mm straight haemostats (Halstead's or Crile's).
6 120 or 140mm curved haemostats (Halstead's or Crile's).
2 Kocher's artery forceps.
2 Czerny's (or Langenbeck's) retractors.
2 Morris' retractors.
Poole's sucker tube.
1 20cm receiver & 2 gallipots.
Desirable additions include Lahey's curved gallbladder forceps.

CAESAR SET (US$950).
6 towel clips.
1 Backhaus towel forceps.
4 Rampley's sponge holders.
1 18cm toothed dissecting forceps.
1 18cm plain dissecting forceps.
2 #4 scalpel handles.
2 180mm needle holders.
2 Allis tissue forceps.
6 Green-Armytage forceps.
12 150mm straight Spencer Wells haemostats.
6 230mm curved Spencer Wells haemostats.
1 Morris retractor.
1 Doyen's retractor.
Poole's sucker and tube.
1 300mm bowl (for blood clot),
1 200mm receiver & 2 gallipots.
Desirable additions include a tenaculum, and a self-retaining retractor.

MINOR SET (US$750).
6 towel clips.
2 Rampley's sponge holders.
4 Backhaus towel forceps.
1 #4 & 1 #5 scalpel handle.
1 toothed dissecting forceps (Treves).
1 plain dissecting forceps (Bonney's).
4 Allis tissue forceps.
1 West's self-retaining retractor.
2 Czerny's retractors (or Langenbeck's).
12 125mm curved haemostats (Spencer Wells).
6 200mm curved haemostats (Spencer Wells).
1 20cm receiver & 2 gallipots.
Desirable additions include a dissector and a Volkmann's spoon.

ABSCESS SET.
2 Rampley's sponge-holding forceps.
4 towel clips.
1 knife handle.
1 sinus forceps.
1 Mayo's scissors.
1 toothed dissecting forceps.
1 150mm receiver.
2 gallipots and some gauze swabs.
2 towels.

UTERINE EVACUATION SET.
2 ovum or sponge-holding forceps (without ratchets).
1 Sims' vaginal speculum.
1 vaginal speculum (Sims, Auvard's or Collin's).
2 Teal's vulsellum forceps.
1 set of Hegar's dilators.
Karman suction curettes.
Uterine curettes with sharp and blunt ends (several sizes each).
1 200mm Kocher's forceps.
1 toothed dissecting forceps.
Have intra-uterine contraceptive devices (IUDs) available.

ORTHOPAEDIC SET.
6 towel clips.
4 Rampley's sponge holders.
4 dissecting forceps: (1 heavy toothed 180mm Lane's or Charnley's, 1 light Adson's 125mm, 1 plain 180mm, 1 McIndoe's 180mm).
6 curved 150mm Spencer Wells haemostats.
6 curved 200mm Spencer Wells haemostats.
1 #4 & 1 #5 scalpel handle.
4 220mm light bone levers, Lane's or Trethowen's.
4 275mm heavy bone levers.
1 Faraboef's elevator.
1 large & 1 small periosteal elevator (for the femur and humerus).
1 Size C double-ended Volkmann's bone scoop.
1 350g mallet.
1 sequestrum forceps.
1 180mm Read Jensen bone nibbler.
1 bone file or rasp.
1 220mm Liston's bone cutters.
1 200mm bone hook.

BURR HOLE SET.
1 Hudson's standard perforator 12mm.
1 Hudson's set of conical burrs 13mm and 16mm.
Hudson's brace.
1 West's self-retaining retractor.
1 60mm brain sucker.
1 Ch14 soft rubber catheter.
1 20ml syringe for washing out.

SMALL (Hand) INSTRUMENT SET.
2 small sponge-holding forceps.
1 plain 150mm McIndoe dissecting forceps.
1 plain 100mm Silcock's ophthalmic dissecting forceps.
1 toothed Adson's 120mm dissecting forceps.
4 165mm Gilles skin hooks.
1 light 190mm McIndoe dissecting scissors.
1 light 140mm curved Aufrecht's scissors.
12 curved Crile's mosquito haemostats.
1 Bard Parker #4 scalpel handle.
2 114mm Derf needle holders.
2 small 178mm Meydering retractors.
2 114mm Harlow Wood tendon hooks.
1 small curette.
2 assistant's scissors.
1 fine probe.

KIRSCHNER WIRE PACK.
6 wires of each size 0·75mm, 1·0mm, 1·5mm.
1 Pulvertaft's Kirschner wire introducer.
1 pair of Kirschner wire cutters.

5 The impact of HIV on surgery

"One night after I had been doing some blood tests in a rural area with some local medical colleagues, they went off with some girls from the town. They slept with them, and only one of them used a condom. In the morning I asked them how they could possibly have taken such a risk, since we all knew the prevalence of HIV was quite high in the region. They laughed, saying that you couldn't give up living just because you might get a disease."
A research worker in Central Africa, PANOS Dossier, 1987 (March).

5.1 Introduction

SURGICAL OVERVIEW
Since the dramatic appearance of a completely new and growing range of pathology in 1981, and the identification of the Human Immunodeficiency Virus (HIV) in 1983 by Françoise Barré-Sinoussi, Luc Montagnier and colleagues at the Institut Pasteur in Paris, enormous efforts have been made to combat this new disease. Control is limited in many low-income countries. The reasons are complex and vary in individual countries, but poverty and lack of resources are the biggest drawbacks.

In these environments, HIV-related disease continues to worry medical resources and still presents one of the greatest single challenges to a medical practitioner seeking to alleviate suffering in poor-resource settings. As a result, the practice of surgery cannot ignore the impact of HIV and must assess the implications of this new disease. Although much has been written of HIV-related surgical pathology, the preponderance of the literature reflects the experience of surgeons working in well-equipped hospitals in the First World, where HIV prevalence is low, and where there is ready access to a multiplicity of laboratory testing, drug therapies and nursing back-up. From personal experience we try to give you guidelines to help you in this new medical minefield.

The practice of surgery is everywhere a challenge, and is so especially in poor-resource settings where improvisation often is the order of the day. Be sure therefore to consider the *balance of risk* inherent in any surgical procedure. An operation which is seen as routine in a well-equipped teaching centre may be a serious risk in a rural hospital; likewise an operation traditionally considered routine in an environment of low HIV incidence may prove to have great risk where HIV is common. *You must not underestimate complications expected in HIV patients, particularly those not on treatment,* and once a commitment is made to surgical intervention, you must treat these complications aggressively if they arise. Thus you may need to restrict your elective surgery, particularly in certain anatomical regions, considering that HIV disease is progressive, particularly if no treatment has been started.

Nonetheless, ***don't deny emergency surgical intervention to the HIV patient***, who often requires more aggressive and urgent resolution of sepsis.

Many trauma victims are HIV+ve, but their management should proceed along standard lines regardless. Indeed, surgical intervention may buy a patient valuable time to begin the process of treatment.

Consequently, examine the impact of surgical intervention in certain HIV-related states carefully, and question the standard practices of surgical orthodoxy in relation to HIV disease.

Consider *performance status* and *life expectancy* carefully. This is especially true in those parts of the world where HIV prevalence is high but where testing is irregularly available, and where their HIV status is generally not known by patients themselves.

Avoid elective surgery (especially in patients with clinical signs of immunosuppression) in the following (unless you can guarantee close supervision of effective antiretroviral (ARV) therapy and a CD4 count >200/μl):
(a) *cosmetic procedures, especially on the nose and mouth, including routine circumcision*
(b) *complex plastic surgery, especially free flaps*
(c) *neonatal intervention for complex abnormalities*
(d) *open brain surgery*
(e) *tonsillectomy*
(f) *open thoracic surgery*
(g) *open perianal surgery*
(h) *insertion of prosthetic grafts or metal.*

Note that after surgery, you often cannot re-start ARV therapy immediately, and this may be a problem (5.8).

Whilst this list is not exclusive, it is also not exhaustive; treat each individual case on its merits. Nonetheless, within a broad perspective, exercise great caution in the above types of surgery. Post-operative infection rates are doubled in asymptomatic HIV+ve patients, and more than trebled in symptomatic HIV+ve patients, especially where the CD4 count is <200/μl.

HISTORICAL OVERVIEW
The most compelling evidence to date suggests that HIV was transferred to humans through transformation of an almost identical simian (monkey) virus in the Congo region in the 1940s or 1950s. The oldest +ve HIV test is from a serum sample of an adult male in Kisangani (formerly Stanleyville), Congo, taken in 1959. Before that there were no deep freezers to store serum. Another +ve sample was found in a lymph node from Congo in 1960.

The emergence of certain diseases, such as Kaposi sarcoma, amongst the homosexual community and intravenous drug abusers in the USA in the late 1970s brought to light a series of ailments related to immune deficiency. Subsequently, an infective agent, one of a group of retroviruses, was identified, and positively linked to further conditions, especially a wasting syndrome seen in Central Africa, known as Slim disease.

It was then evident that HIV was spread heterosexually and that subsequently this mode of spread was to prove geographically and numerically far greater a threat to populations. The rate of spread is linked to the presence of co-existent sexually transmitted diseases, principally of the ulcerating variety.

Studies show that a relatively small pool of infected commercial sex workers could be responsible for 80-90% of the initial disease prevalence in a community. Numbers of cases of HIV disease have increased exponentially, and in many sub-Saharan countries doubled every 9-12 months. This trend has been followed in Southeast Asia and the Indian subcontinent.

Initial reactions amongst politicians to the scourge of HIV, which was known to result in inevitable, usually slow and agonizing death through an end-stage described as Acquired Immune Deficiency Syndrome (AIDS), were to deny the problem. The overlay of sexual promiscuity, and in the West, of heterodox lifestyles, served to *exceptionalize* HIV disease, which has been handled differently from other infectious diseases (especially with regard to counselling). Stress was on confidentiality and anonymity, and concerns about abuse of a victim's civil rights demanded private individual counselling prior to HIV testing. This has resulted in isolation of the sufferer, contrary to the prior tradition (as in Africa) of understanding illness as a community problem to be discussed fully within the family and then within the village setting.

Thus, the HIV patient has often been secluded and even victimized in rural society and even within the family itself. Many women preferred not to know their HIV status, fearing ostracism, because they have little control over their lives and cannot make plans for the future. With the increased availability of antiretroviral medication, however, the exceptionalization of HIV disease has become an anachronism. In some countries, notably Uganda, the inexorable increase of HIV cases seems to have been reversed, principally through strenuous saturated educational coverage, propagated in the main by non-governmental organizations, and by popular fear of the disease.

The advent of ARV therapy has had a significant impact on HIV disease, even in the late stages. However, this therapy remains exorbitantly expensive long-term for many people in low-income countries, although WHO is making strenuous efforts to make low-cost drugs available. Single-dose treatments for antenatal women reduce transmission to the unborn child, after needle-stick injuries, and in rape cases. The emergence of resistant strains, however, remains a problem. The development of a vaccine is still at this stage a dream. Viricidal creams may offer some real hope in reducing transmission.

Thus HIV is a fact of life (and death) in many parts of the world, and surgeons in both cities and remote areas must know its implications.

5.2 Pathophysiology

The causal agent of AIDS is known to be HIV, which has two known types (HIV-1 and HIV-2), belonging to the family of primate *lentiviruses* (slow viruses), differing by the former having a *vpu* and the latter a *vpx* gene, absent in the other. There is great similarity with the Simian Immunodeficiency Virus (SIV), strongly suggesting a link between these viruses. HIV-2 is more similar to SIV, and HIV-1 has been found genetically to originate from a chimpanzee species. In conformity with other retroviruses, HIV contains a virus capsid whose hallmark is the enzyme *reverse transcriptase*. This enables a double-stranded DNA copy of the original genomic RNA to be made in host cells. The viral DNA is thus integrated into the lymphocyte genome. The glycoprotein (gp120) envelope of HIV binds to the glycoprotein (gp41) molecule on the surface of certain thymus-derived T-lymphocytes known as helper/inducer cells. This molecule called CD4 is also found on other cells, such as macrophages, monocytes, and even some antibody-producing B-lymphocytes, as well as in brain cells.

The helper/inducer T-lymphocytes are the kingpins of the immune response: when stimulated by antigen contact, they divide and produce lymphokines (such as *interleukin 2* and *interferon*) which control the growth and maturation particularly of cytotoxic/suppressor T-lymphocytes which have a CD8 glycoprotein molecule.

The ratio of CD4 to CD8 gives a good indication of immunological capability. Early on in HIV infection, the CD8 cell number may rise, but there is an inexorable fall in CD4 cell numbers; in the final stages of disease, the CD8 count will also fall.

Virus replication appears to occur mainly in dividing CD4 cells and these cells divide upon stimulation by micro-organism antigens (at least *in vitro*): thus intercurrent infections may stimulate viral replication. Paradoxically, in the final stages of the disease, when CD4 counts approach zero, there may be little active viral replication. *You can think of the CD4 count as the distance a patient is from death; the viral load the speed at which he is travelling there.*

The extensive genetic variability in HIV isolates and the inherent difficulty of blocking the CD4-HIV binding make vaccine development far from straightforward.

Although HIV core antigen can be detected and viral counts are very useful for monitoring antiretroviral therapy, these tests are rarely available in the developing world. The most widely used ELISA anti-Immunoglobulin antibody test for HIV infection will only become +ve 6wks to 9months after infection, thus producing a 'window' period when HIV is actually present in serum but *not* detected.

This has serious clinical significance; change in the test from -ve to +ve is known as seroconversion. The accuracy of the test and its sensitivity is high; most incorrect results arise from laboratory or deliberate errors, and if a result is clinically suspicious, you should organize a repeat test, preferably using a different laboratory or a different technique (*viz*. radio-immunoassay) with better specificity. The Western Blot method is expensive, however, and is probably not justifiable in most situations in the developing world.

5.3 Transmission and prevention

The 3 most prolific methods of transmission of HIV in the developing world are:
(1) by sexual contact, dominantly heterosexual,
(2) from mother to baby,
(3) by blood products.

Transmission may also occur through:
(4) transplanted tissue,
(5) sharps injuries and splashes.
This may occur between drug users sharing injection needles, especially when 'mainlining' (injecting) themselves with IV drugs.

Viral particles have, however, been detected in seminal fluid, and pre-ejaculate fluid, vaginal and cervical secretions, breast milk, tears, urine, and saliva, so caution regarding transmission is wise.

(i) Sexual contact
As simultaneous sexual promiscuity by men is common, there can be no clearly defined risk group; nonetheless certain groups have significantly higher prevalence rates than others, and therefore a high index of suspicion is justified. Such groups are:

army personnel,
those travelling widely in their employment,
e.g. *truck drivers, police, and itinerant salespersons,*
attenders at venereal disease clinics, and those tested +ve for syphilis, filariasis and leishmaniasis,
men working away from home,
those with high alcohol intake,
prisoners (through close and sexual contact),
divorced, separated, or young widowed women,
young widows and widowers.

As, however, the spouses of infected persons are at as great risk, the identification by history and direct social questioning of potential HIV individuals becomes at best difficult and time-consuming. Nonetheless identifying a girl as a virgin is helpful in minimizing HIV as a factor in reaching a diagnosis.

The estimated risk factor of transmission from a seropositive man to woman during a single unprotected sexual exposure is c.0·5-0·75%, but from a seropositive woman to man 0·25%. (There is a considerable range from 0·1% where the viral load is <1700 copies/ml to 20% where the load is >38500/ml.) The risk is zero if viraemia is undetectable.

The risk increases in a violent sexual encounter such as rape, in the deflowering of a virgin, if ulcerative venereal disease or filariasis is present (up to a factor x10), if non-ulcerative venereal diseases are present, if an intra-uterine contraceptive device is *in situ* and during pregnancy. It may be 500 times higher in the phase of acute HIV seroconversion. The risk increases x4-x7 in anoreceptive intercourse, and is further increased when jelly with the spermicide nonoxylon-9, which breaks down the rectal lining, is used. The risk is also present in oral sex, and with artificial insemination.

A condom (female as well as male) is protective, and reducing menstrual bleeding (by use of the combined pill and depot medroxyprogesterone acetate) will also reduce transmission. Male circumcision also reduces transmission.

Restricting sexual activity to a monogamous marriage without extra partners remains the only certain way to avoid HIV exposure by this route. Therefore you should advocate prenuptial HIV testing, and certainly before any pregnancy is considered.

(ii) Vertical transmission of HIV from mother to baby varies between 15-45% if there are no interventionist strategies used; estimates are that transmission occurs in ⅓ before delivery, in ⅓ during delivery, and in ⅓ after delivery. It seems possible to reduce transmission to 2-3% with the antenatal use of antiretroviral drugs (a single dose of nevirapine appears to be sufficient), arranging delivery by Caesarean section, and avoiding mixing breastfeeding with bottle feeding of milk substitutes.

Whilst the adoption of mandatory Caesarean section for HIV+ve mothers may have theoretical justification, the morbidity and mortality inevitable in such a policy in the developing world outweigh the advantages notwithstanding the costs of screening and surgery. Previous policies of restricting breastfeeding have actually been shown to be harmful, and *are not recommended*.

Other practices, however, reduce risks of transmission: protocols developed to prevent blood exchange from mother (high-pressure) to foetus (low-pressure) in potential rhesus sensitization are applicable up to the moment you clamp the umbilical cord:
(1) Treat infections which disrupt the placental barrier, particularly malaria and toxoplasmosis because these increase transmission of the virus. Malnutrition also allows increased transplacental viral transmission.
(2) Reduce prolonged labour by use of prostaglandins and oxytocin. Treat chorio-amnionitis with antibiotics.
(3) *Avoid external cephalic version and amniocentesis*; clamp the umbilical cord as early as possible. The longer the baby is protected in labour from direct contact with the mother's blood and secretions, the better.
(4) *Avoid artificial rupture of membranes* and make episiotomies at the last moment. If membranes are already ruptured, *reduce contact time by use of oxytocin.*

N.B. Foetal scalp electrodes and foetal scalp blood collections are contraindicated.

(5) Be very careful with instrumental deliveries, preferably using rubber cup vacuum extractors to prevent abrasions of the foetal head; better avoid them altogether. Washing of the vagina with povidone iodine before instrumental delivery or after rupture of membranes is probably a sensible precaution. Rinse babies immediately after delivery in warm water.
(6) During Caesarean section, try to deliver the foetus with intact membranes; *don't use the scalpel to open the whole thickness of the abdominal wall*, lest the baby is cut. Suctioning of the baby after delivery pushes maternal blood up its nose and is unnecessary; wiping is usually sufficient.

Transmission during breastfeeding appears to increase if the mother seroconverts during this time, if breastfeeding is mixed with other feeds, and if the nipple is cracked or eczematous, or the baby has mouth ulcers. Abandoning breastfeeding implies the ready availability of milk substitutes, rarely the case for the poor in low-income countries, and removes the natural transmission of protective immunoglobulin to the baby. However, expressed breast milk can be pasteurized (kept at 62·5°C for 30mins, or heated just up to boiling and then cooled) to eliminate HIV, as well as hepatitis B virus (HBV). Supply it then in a small cup rather than in bottles with teats as these are difficult to sterilize properly. Nutrients and micro-nutrients are preserved but IgA antibody activity is lost, and diarrhoea is then a frequent problem. Proper attachment of the baby to the breast and preventing nipple damage also reduces the risk. Heat treatment is not possible for colostrum, however, because it curdles and there is a high viral load in colostrum. Correct vitamin A deficiency, which increases the risk of transmission.

N.B. Transmission of HIV from seropositive baby to surrogate breastfeeding mother has occurred, and vice versa from seropositive surrogate mother to baby.

(iii) Transfusion of blood products entails a significant risk (3.6), especially where laboratory testing is unreliable. Because of the window period, apparently safe blood products may actually be contaminated. In order to reduce this risk, encourage longstanding donors whose HIV-ve status can be followed over a considerable period of time (and are therefore unlikely to seroconvert), unlike schoolchildren who may become newly sexually active. Discard blood from a new donor deliberately; accept it only if he tests -ve on a subsequent visit after nine months. Select blood donors on a voluntary basis, thus removing a financial incentive for donation.

The risks from transfusion are cumulative, rising with numbers of units transfused. Furthermore, the risk of using products pooled from many donors is also higher; thus *don't use fresh frozen plasma and pooled platelet concentrate*. The use of Factor VIII concentrate is likewise risky but may be essential in treatment of haemophiliacs requiring surgery; render it safe by heat treatment.

Use blood transfusion therefore very sparingly; educate anaesthetists concerning the safety of working with suboptimal Hb levels, and learn the appropriate strategies.

Various strategies can avoid risks:

(a) Autologous blood

Take 1L of blood from an adult; treat him with maximal doses of ferrous sulphate for 2wks; then take a further 1L at the same time as transfusing 500ml of the previously collected blood. In this way you can prepare 1500ml (3 units) of blood for elective surgery.

(b) Intraoperative haemodilution

Take 1L of blood immediately prior to surgery and replace it with crystalloid. The fresh and platelet-rich blood is then immediately available for re-infusion if needed; blood viscosity is also incidentally lowered and this may be an advantage, especially in vascular surgery.

(c) Peroperative blood salvage (autotransfusion)

Blood from clean traumatic injuries of the chest or abdomen, or from an ectopic gestation, is ideal for this treatment; it can be life-saving. The blood is warm, and still has its clotting factors; it carries no risk of hepatitis or HIV, and it will be perfectly cross-matched. Autotransfusion is not only simple but also very useful.

CONTRAINDICATIONS

Don't attempt autotransfusion if:
(1) There is an offensive smell when you open the abdomen.
(2) The abdomen is grossly contaminated.
(3) The blood is obviously haemolysed.
(4) A woman is more than 14wks pregnant with a ruptured amniotic sac. Her blood will be contaminated with amniotic fluid containing large quantities of thromboplastin. If you transfuse this, it could theoretically cause disseminated intravascular coagulation (DIC). Nonetheless you can use blood in a contaminated peritoneal cavity on occasion under antibiotic cover without untoward effect if you are absolutely desperate.

N.B. The presence of fresh clots is not a contraindication to autotransfusion.

THE VACUUM BOTTLE METHOD is the best. Buy vacuum bottles, or prepare them by closing blood-taking bottles containing 150ml 3·8% citrate-dextrose immediately after they have been sterilized, before the steam in them has had time to condense. Clamp a taking set, introduce one of its needles into the abdomen, as if you were doing a 4-quadrant tap, and then put the other needle into the bottle and remove the clamp. To fill the bottle insert another sterile needle connected by way of a heparinized suction catheter to a vacuum pump into the bung. You may be able to collect up to 3L of blood this way. If the vacuum is imperfect, and does not fill the bottle, apply low-pressure suction with a vacuum (water) pump connected to a sterile needle inserted through the bung.

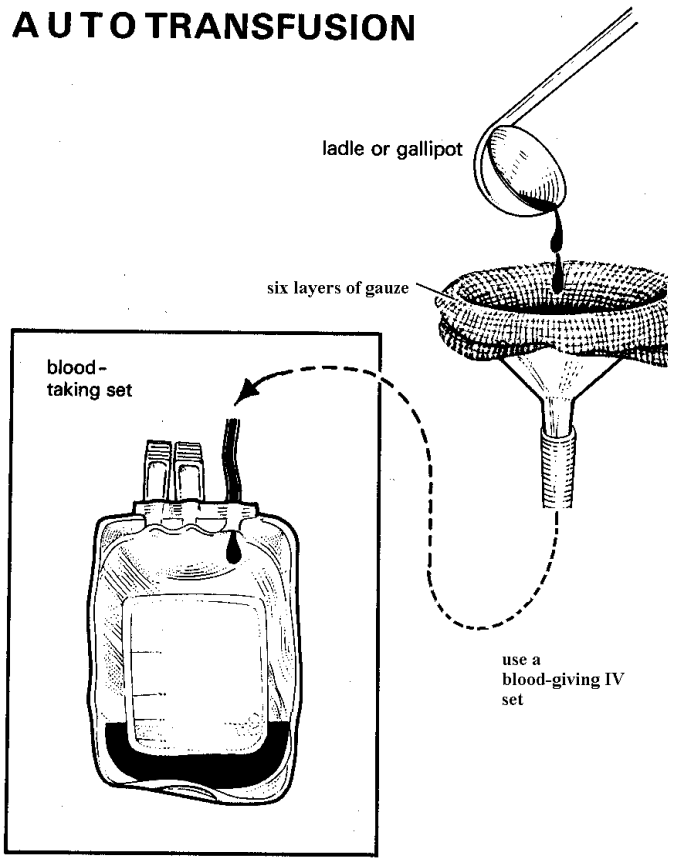


Fig. 5-1 AUTOTRANSFUSION using a funnel. This is also useful if the patient has a ruptured spleen. Use 6 large pieces of gauze, and collect the blood through a blood-giving IV line into an empty citrated blood-collection unit.
Kindly contributed by Stephen Whitehead of Maua Hospital, Kenya.

THE SOUP LADLE METHOD is less satisfactory, but is useful when you cannot use a vacuum bottle because there are too many clots. Keep the equipment (5-1) ready sterilized. Put the patient into a 15° head-down position, make a small opening in the peritoneal cavity to begin with, and be prepared to catch the blood, as it escapes, with a sterilized stainless steel soup ladle or gallipot. Then complete the incision and ladle out the rest of the blood. The right hypochondrium may be the easiest place to collect it. Pour it through a small metal funnel, and collect it through a blood-giving set into empty blood transfusion packs pre-primed with citrate anticoagulant. The filter in the drip set will remove smaller clots.

N.B. You can also use a filter made of 6 layers of gauze *but beware that if the gauze is contaminated by glove powder, the autotransfused blood may kill the patient!*

You can also squeeze blood out of soaked laparotomy pads after they have been agitated in saline. Alternatively, you will find a sump useful. This is a conical vessel with a handle and holes towards its tip. Insert it deep in the abdomen; blood will flow in through the holes and can be sucked out. Use low-pressure suction because the higher the pressure, the more haemolysis occurs.

CAUTION!
(1) Either transfuse the blood immediately, or throw it away.
(2) *Don't use it for someone else*.
(3) Use prophylactic antibiotics.
There are expensive commercial autotransfusion machines available, but they all work on the system of (1) aspirate, (2) anticoagulate, (3) filter, (4) centrifuge, (5) wash, (6) re-infuse.

N.B. Directed blood transfusion (where blood is collected from relatives or friends) has almost all the pitfalls of undirected transfusion.

(iv) Transplantation (of kidneys, allograft skin etc) carries the risk of HIV transmission.

(v) Sharps injuries and splashes
Risks of transmission of HIV to health personnel are small but real. Use routine double-gloving for surgical procedures, especially when you may encounter sharp pieces of bone, or use wires, drills or chisels; some prefer to use a glove half a size greater on the outside. The use of different-coloured gloves may highlight damage to the glove material more easily. Wear a non-sterile glove under a sterile one if economy dictates. (*Don't use recycled gloves for operating*, except as the first in double-gloving, unless economy dictates!) Long arm gloves are useful for surgery involving deep ingress into the abdominal cavity. Special Kevlar gloves are useful (and re-usable) as the interior glove because they cannot be penetrated by needles or blades: this makes their initial high cost worthwhile. They are however cumbersome to work with. Sterile cotton gloves can be worn outside the latex glove where wires are used in orthopaedic surgery; the wire will snag on the cotton before tearing the latex.

Blood splashes are also important especially to the open eye (with risk rates estimated at 1·5%). Use protective eyewear therefore, especially where spraying is likely, *e.g.* orthopaedic drilling. However, wrap-around plastic goggles are inconvenient for those with spectacles, and attachment of sides to the spectacles is a reasonable alternative. Masks also help protect the mouth from splashes.

Careful operating is, however, probably more important than trying to prevent injury. Avoid operating if possible when you are over-tired! Likewise drug users can avoid HIV transmission by using unused sterile needles, and discarding these carefully.

The estimated risk of seroconversion with a penetrating hollow needle-stick injury is 0·3% and with a solid needle is 0·03%. Reduce the use of cutting needles (you can close an abdomen readily using a blunt-ended needle) and preferably employ a no-touch surgical technique. Introduce rigorous adoption of theatre routines: *don't hand sharps from scrub sister to surgeon and vice versa*. Create a neutral zone where sharps are placed in a receiver by only one person at any given moment. *Don't use hands as retractors, and the surgeon's fingers to guide needles* (2.3).
Draw up multivial solutions using an unused sterile needle. Glove up for venepuncture and handling blood samples.

Most injuries to healthcare workers arise from careless handling of sharps:

Recapping of needle	40%
Improper disposal of sharps	32%
Contamination in open wound	25%
Other	3%

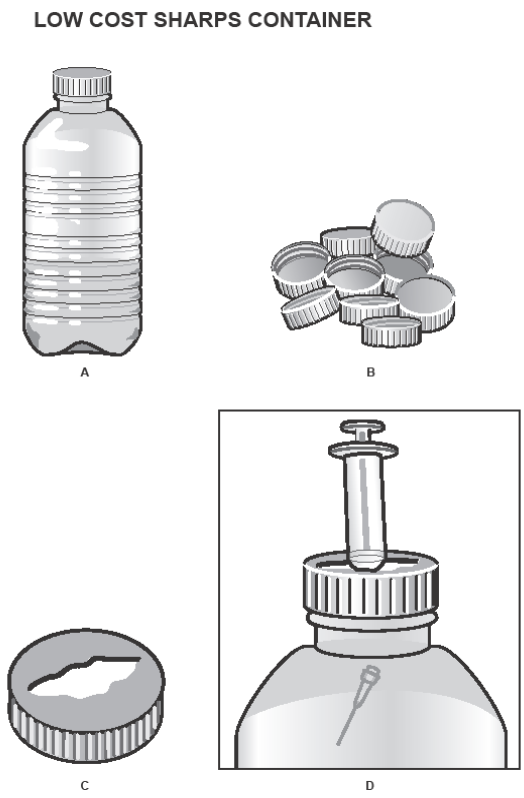


Fig. 5-2 LOW-COST SHARPS CONTAINER WITH NEEDLE REMOVAL DEVICE.
A, ordinary plastic bottle. B, bottle tops. C, bottle top with hole cut out. D, syringe inserted through hole and needle eased off inside bottle.
After Onayade AA, Omotoso AO, Olafimihan VB. Hospital biohazard control: low-cost device for safe collection and disposal of hypodermic needles. Tropical Doctor (2006):36(4)215.

Discard needles uncapped; separate and dispose of other sharps (*don't leave them for someone else*) in a dedicated, labelled, non-breakable, puncture-proof, watertight container with a keyhole opening large enough but too small to admit a hand. Fix this to a wall, and empty it when ¾ full, and incinerate it. *Don't over-fill the container*. (You should probably avoid expensive specially manufactured containers as they may be utilized for another purpose by cleaners.) A useful home-made container can be made from an ordinary mineral water bottle with its screw-top pierced with an elliptical hole (5-2A); thrust a needle attached to a syringe through this hole and with minimal manipulation pull the syringe back, thus detaching it from the needle (5-2D). Put non-sharps in other containers in order not to waste the space in the sharps container; *don't separate needles from syringes with your fingers!*

Adopt a hospital sharps-injury policy in order to advise *all* health workers on precautions and action to take after exposure, depending on facilities available. Discipline staff not taking precautions. Introduce a post-exposure prophylaxis/treatment policy if you don't already have one in your hospital. Remember to wash the part injured by a needle-stick immediately, and cleanse it with betadine. Use the low-cost de Montfort medical waste incinerator. In the laboratory, heat-treat serum at 56ºC for 30mins before testing; pipette solutions using a teat, *not your mouth!*

A practice that assumes every patient is an HIV risk, and all blood may be contaminated, is likely to result in far fewer accidental seroconversions than one that attempts to identify individual high-risk patients.

The HIV+ve health worker is extremely unlikely to transmit HIV to patients except sexually; the risk of a surgeon passing HIV to a patient has been estimated at 1:800,000.

5.4 Sterilization

Wear gloves when cleaning spills. Dilution by washing is important. If alcohol is used, wipe the surface several times because alcohol evaporates. Establish the rule, **"You spill it, you clean it."** Dispose of contaminated materials safely; *don't put them on a rubbish tip* where they may be scavenged! Ordinary laundry is effective for cleaning soiled linen after thorough soaking.

Chemical disinfection is acceptable only for instruments such as endoscopes which cannot tolerate heat. Pull out and clean light carriers and biopsy carriers, and wash internal tubes thoroughly before placing them in antiseptic. This may be:

(a) Chlorine-releasing solutions* (readily neutralized by blood or tissue),
(b) Ethanol 70% (for 15mins)
(higher and lower concentrations are less effective),
(c) Isopropyl alcohol (2-propanol) 70% (for 15mins),
(d) Povidone iodine 2.5% (for 15mins),
(e) Formaldehyde 4% (for 30mins),
(f) Alkaline-buffered glutaraldehyde 2% (for 10mins)
(*Cidex*, *Asep*, *Omnicide*, *Tegodor*): effective for 14 days, once prepared, if kept away from direct sunlight,
(g) Hydrogen peroxide 6% (for 10mins),
(h) *Virkon* (balanced blend of peroxygen compounds, surfactant and organic acids in inorganic buffer at pH2.6, sold as a stable powder, is non-corrosive, non-bleaching, non-toxic and not a transport hazard: a fresh 1% solution is used for 30mins).

CAUTION!
The following solutions are NOT recommended: spirit solutions <70%, especially <50%, cetrimide (*Cetavlon*) or chlorhexidine (*Hibitane*), formalin 0.1%, quarternary ammonium compounds (*e.g. Dettol*, *Roxenol*, *Flavine* etc).

* Chlorine-releasing solutions are excellent disinfectants. Their power is expressed in 'available chlorine': 1% = 10g/L = 10,000ppm (parts per million) = 3·33 chlorometric degrees

N.B. Chlorine corrodes iron and stainless steel, so these disinfectants must not be stored in stainless steel containers; equipment is easily corroded and should be rinsed well after disinfecting.

Sodium hypochlorite solutions (liquid bleach, *Javel*) are unstable:

Neat disinfectants (*Domestos*, *Chloros*, *Sterite*) contain c.100,000ppm

'Strong' hypochlorite solution contains >80,000ppm

Most supermarket brands contain c.50,000ppm

'Milton' contains c.10,000ppm (with salt)

HIV is inactivated by 5,000ppm solution in 1 minute, by 50ppm in 10mins; at this low dilution it is *very* unstable so must be freshly made, used and discarded.

Calcium hypochlorite (70% available chlorine) and bleaching powder (35% available chlorine), sold as tablets, granules or powder, both decompose gradually if not protected from heat and light.

Sodium dichloroisocyanurate 0·5% (NaDCC: 60% available chlorine) and tosylchloramide sodium (Chloramine T: 25% available chlorine), sold as powder or tablets, are comparatively stable.

Use solutions of 1,000ppm for general disinfection of wards, theatres and laboratory benches.

Clean contaminated surfaces with 5,000ppm which is left in contact for 30mins before rinsing off.

Recommended dilutions of chlorine-releasing agents			
	Available chlorine	Clean condition (*e.g.* cleaned medical equipment)	**Dirty condition** (*e.g.* blood spills, soiled equipment)
Available chlorine required		0·1% (1g/L, 1000ppm)	0·5% (5g/L, 5000ppm)
Dilution			
Sodium hypochlorite solution	5%	20ml/L	100ml/L
Calcium hypochlorite	50%	1·4g/L	7·0g/L
NaDCC	60%	1·7g/L	8·5g/L
NaDCC-based tablets	1·5g per tablet	1 tablet/L	4 tablets/L
Chloramme	25%	20g/L	20g/L

RECOMMENDED DILUTIONS OF DISINFECTANTS
WHO AIDS Series (2), Guidelines on Sterilization and High-Level Disinfection Methods effective against HIV. Geneva 1988

Standard autoclaving at 121ºC at 1 kg cm^2 or use of a hot air oven at 170ºC for 2h eliminates HIV (as it does the hepatitis B virus, which is much more easily transmitted). Thus place all surgical instruments which tolerate heat, and all reusable surgical sundries in disinfecting fluid and then clean them (someone wearing non-sterile gloves) free of blood or tissue and then sterilize them by heat.

5.5 Testing and visual recognition

Combo kits are now available which detect IgM as well as IgG, and so reduce the *window period* when a common screening test may show -ve in the presence of early infection (and high risk of transmission owing to high viral loads).

As most erroneous results are from laboratory errors rather than deficiencies of the test, repeat surprise results. Repeat confirmation of -ve results likewise for safety, after 3-6 months. Oral tests are now available.

Though the theoretical model of insisting on counselling for HIV tests as promoted in the First World is commendable, it may not be practical in the developing world situation where resources are few and trained manpower limited. It has been shown that a short description of the facts of HIV disease without full discussion of the social implications will lead to fear and despair rather than a positive attitude to the disease; thus limited counselling may be more detrimental than none at all.

If tests are only done when a patient has been fully counselled, many patients will go untested. Furthermore a possible HIV-ve result may thus be denied a patient who is too scared to ask for a test, assuming, as many do in areas of high HIV endemicity, that any severe illness is probably the dreaded disease that leads to certain early death. To put pressure on a patient to make up his mind on whether or not to have a test which may reveal a fatal illness is like asking a patient with a pathological fracture whether he wants an X-ray to be done if it might reveal a malignancy. Indeed even the counselling becomes something with a stigma attached for the patient.

In areas of high endemicity, an HIV test may be important to exclude HIV infection as a diagnosis, rather than confirming the presence of the disease, and thus give a patient hope when he had long given up ideas of recovering from illness.

Where HIV is prevalent, and testing difficult or impossible, an awareness of the clinical presentation of HIV-related disease is essential. As any body system can be affected, HIV does not manifest itself usually by a single identifying pathology. Therefore, look for the usual significant tell-tale signs in patients, especially to give clues as to the underlying pathology of the presenting condition. *HIV has made new diseases common, and changed the diagnostic spectrum.*

Certain conditions have a very high association with HIV, others less so; it is the sum total of the clinical picture that is important. Because HIV affects any system, always take a meticulous general history and make a full examination.

Perhaps the most striking features of untreated HIV patients recognizable before any medical interview is undertaken are the following (seen obviously mainly in the face):

(1) Facial rash, typically seborrhoeic dermatitis,
(2) Blue sclera,
(3) Lymphadenopathy,
(4) Herpes zoster scarring (5-3),
(5) Parotid swelling (5-12),
(6) Unilateral ptosis (5-14),
(7) Weight loss, hair changes and premature ageing.

Where you see these features, concentrate the remainder of the medical history and examination for diagnostic purposes on further HIV-related conditions. Although, of course, patients with HIV may have non-HIV-related illness, inherently it is more likely that the illness is indeed HIV-related. For example, a patient with clear signs of immunosuppression complaining

of headache is much more likely to have HIV-related causes of headache than a brain tumour.

Gradation of severity of symptoms and signs is related to dropping CD4 counts, but this test is not available in most hospitals, although you can gauge it by the total lymphocyte count.

5.6 New pathologies and new strategies

In every area, HIV has an impact; not only are new pathologies seen, but many well-recognized problems become more severe. We provide a brief overview here, but detailed discussion is in the respective relevant chapters. Of note is that prior to treatment, there may be several diseases present simultaneously with HIV. Consequently one symptom may be caused by different pathologies, and further, different symptoms may have separate pathologies. So in HIV disease, Occam's famous razor, "*Numquam ponenda est pluralitas sine necessitate*" ("Plurality must never be posited without necessity"), is inevitably blunted.

(a) Skin diseases (34.4)

In the skin, HIV directly attacks antigen-presenting dermal dendritic cells and Langerhans cells, which take up antigens, process them and present them to unexposed T-lymphocytes in lymph nodes, after which they themselves migrate to the skin to exert a protective immune effect. Impairment of this system leads to microbial invasion and malignant change. Furthermore, contact between HIV-traumatized dendritic cells and T-cells during antigen presentation causes a surge in HIV replication.

(1) **A fine facial rash** is virtually diagnostic; new skin affectations in HIV disease are very common, found in >50% of HIV patients presenting in hospital, and almost in 100% in the terminal stages of the disease. Idiopathic maculopapular eruptions are frequent and pruritic; these papular dermatoses must be distinguished from urticaria and lesions due to insect bites which occur on exposed skin. (Some of these may respond to dapsone; they don't respond to steroids.) Itching is often severe and needs a sedative or antihistamine.

(2) **Opportunistic infections, such as tinea, candidiasis, and scabies**, especially crusted and Norwegian types, may be florid and widespread.

(3) **Bacterial skin infections**, especially with *Staphylococcus aureus*, are more common. Likewise, allergic skin reactions are more common, and may be florid and life-threatening: frequent culprits are thiacetazone, sulphonamides, streptomycin, and pyrazinamide.

Some dermatitis may be secondary to other conditions, *e.g.* HIV-related malnutrition leading to pellagra. Some malignant skin conditions may show a more aggressive pattern: this has been reported with malignant melanoma.

Several skin ailments are common with high HIV association, with *herpes zoster* having a high predictive value, and *herpes simplex* on the vulva and buttocks; eosinophilic folliculitis has only been seen in HIV+ve patients, particularly where the CD4 count is <400/µl.

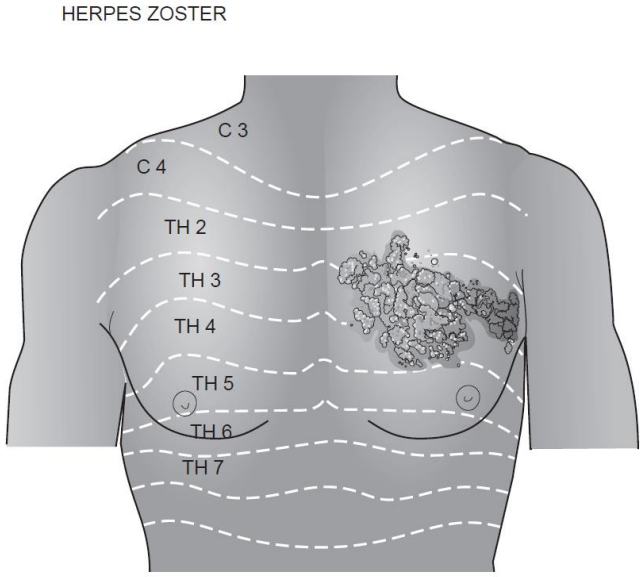


Fig. 5-3 HERPES ZOSTER. Blisters, classically ending in the midline.

Increased photosensitivity to sunlight and therapeutic irradiation is common.

Typical manifestations of skin disease (34.4) are:

Aggressive psoriasis (5-7),
Bacillary angiomatosis,
Candidiasis,
Condylomata (5-13),
***Cryptococcus* ulcers**,
Eosinophilic folliculitis,
Florid *tinea corporis*,
Herpes zoster (5-3),
Kaposi sarcoma (5-8,11),
Molluscum contagiosum (5-4),
Multiple *herpes simplex*,
Pyoderma gangrenosum,
Seborrhoeic dermatitis (5-6),
Stevens-Johnson syndrome (5-5).

It is probable that some of these skin infections destroy skin grafts, especially *herpes zoster* and *Molluscum contagiosum*.

(4) **Pressure sores** (34.16) are, alas, all too common in the debilitated advanced HIV+ve patient; these are often deep and resistant to healing: prevent them! Gloves, filled with water and tied, make excellent soft supports.

(5) **Malignant melanoma** in white people is 3 times as common in HIV disease.

MOLLUSCUM CONTAGIOSUM

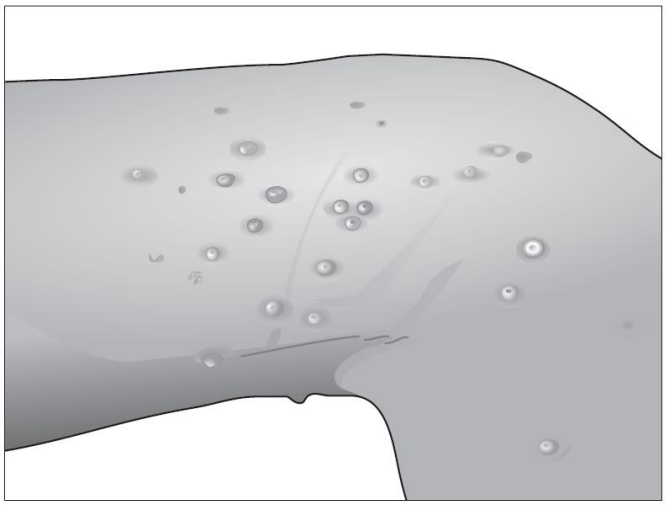


Fig. 5-4 MOLLUSCUM CONTAGIOSUM, producing multiple typically punctuate lesions.

STEVENS-JOHNSON SYNDROME

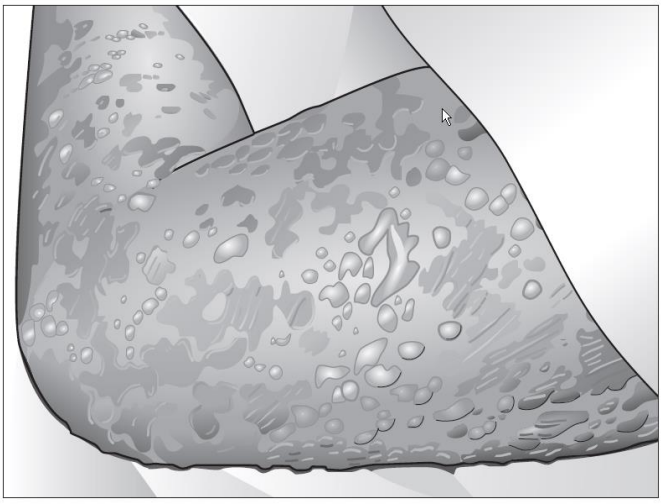


Fig. 5-5 BULLOUS EPIDERMOLYSIS (Stevens-Johnson syndrome) is a widespread blistering reaction that looks like a burn wound.

SEBORRHOEIC DERMATITIS

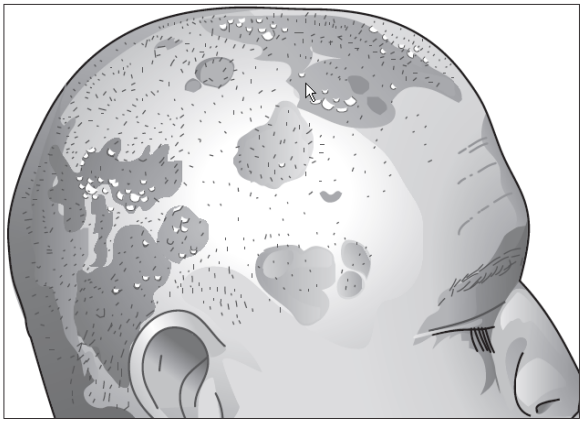


Fig. 5-6 SEBORRHOEIC DERMATITIS, which usually affects scalp, groins, and perineum, but can be widespread.

PSORIASIS

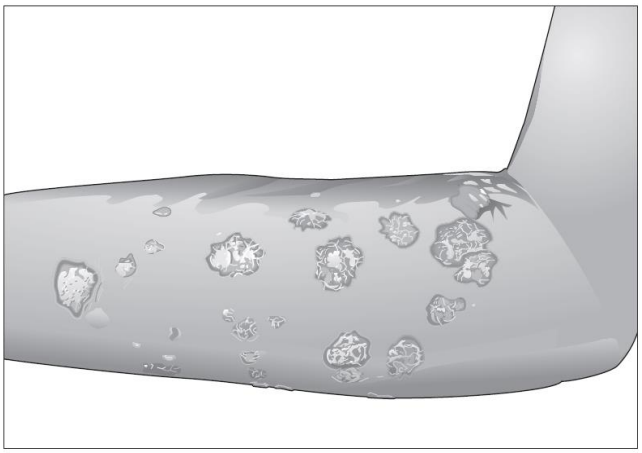


Fig. 5-7 AGGRESSIVE PSORIASIS.

(b) Soft tissue disease

(1) **Kaposi sarcoma (KS)** in its aggressive widespread form is now recognized as virtually diagnostic of HIV disease (34.10).

CLASSICAL KAPOSI SARCOMA

Fig. 5-8 CLASSICAL KAPOSI SARCOMA, typically on the leg, producing violet nodules and cutaneous ulceration.

(2) **Cellulitis** (6.22), arising without history of diabetes mellitus or trauma, is a common manifestation of HIV; the causative organisms remain *streptococcus* and *staphylococcus* and the disease responds to intravenous penicillin or cloxacillin, rest and elevation of the affected limb. There is, however, frequent skin necrosis requiring debridement and subsequent skin-grafting. This may occur with *pseudomonas aeruginosa* infection where the result is known as ecthyma gangrenosum. Facial cellulitis is potentially life-threatening owing to possible spread of organisms to the brain through the cavernous sinus, and requires aggressive intravenous antibiotic therapy.

NECROTIZING FASCIITIS

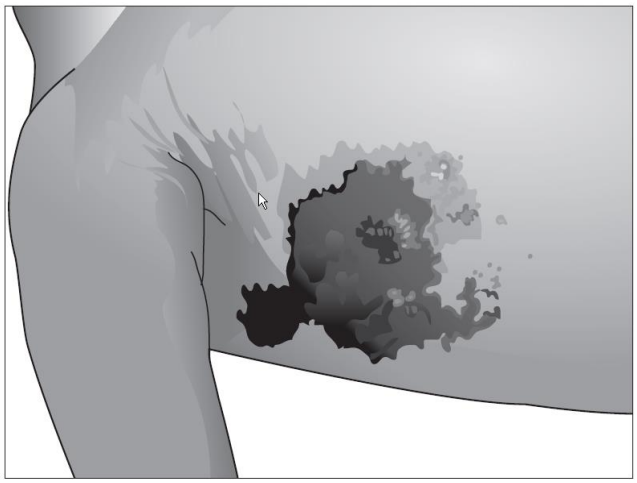


Fig. 5-9 NECROTIZING FASCIITIS, which is classically on the scrotum, but can appear in the perineum, abdominal wall, neck, limbs or indeed anywhere.

(3) **Necrotizing fasciitis** (5-9, 6.23) describes soft tissue infection initially remaining hidden until the blood supply to the skin is affected by increasing oedema and inflammation; thereupon there is rapidly advancing necrosis, if there is excessive collagenase production by haemolytic *streptococci* or *staphylococci* and *peptostreptococci.* The scrotum (Fournier's gangrene) and abdominal wall (Meleney's gangrene) are common sites, but you may also see necrotizing fasciitis associated with HIV in the limbs and neck. This may occur in infants as well as adults.

(4) **Pyoderma gangrenosum** represents a very painful necrotizing non-infectious ulceration, especially in a non-healing wound, often associated with fever. This responds to a short course of prednisolone 60mg/day (if the CD4 count is >50/µl), application of zinc oxide cream and maybe dapsone. *Debridement makes it worse!*

(5) **Recurrent infections and abscesses** (6.2), multiple and frequently recurring in skin and soft tissue, are also typical in HIV disease; standard methods of treatment are effective, but attention to every focus of sepsis is essential. Pus swab microscopy is useful; though the causative organism is most often staphylococcal, it may not be so and is sometimes Gram-ve. *Discourage the use of antiperspirant 'roll-ons'* because these may clog up skin pores, causing abscesses.
There is chronic staphylococcal carriage with decreasing immune competence, and therefore colonization of foreign bodies such as catheters is high. The incidence of post-operative wound infections increases dramatically in HIV+ve patients, especially if the CD4 count is <200/µl.

(6) **Abscess formation**, especially *de novo*, in normally clean anatomical sites should give rise to suspicion of HIV disease; in this category are breast abscesses in non-lactating women (6.13), muscle (pyomysositis) (7.1), thyroid (6.12), abdominal wall, penile (6.21) and retroperitoneal abscesses (6.15). Submandibular and neck abscesses (6.11) are often related to pre-existing lymphadenopathy and may be tuberculous.

Pressure sores (34.16) often arise from the combination of inertia, cachexia and neuropathy in HIV disease.

(7) **Leiomyosarcomas** in children are unusual lesions noted to be associated with HIV and specifically exposure to Epstein-Barr virus. The lesions occur subcutaneously, in the respiratory and gastro-intestinal tract, and even in the kidney. They appear not to be common in Africa to date.

(8) **Muscle atrophy** is frequent in debilitation; specific wasting syndromes are also seen with rises in CPK levels and increased numbers of macrophages in muscle biopsy specimens.

(9) **Lipodystrophy** is a generally abnormal degeneration of fatty tissue, seen in advanced HIV disease, where fat is lost in the extremities, buttocks and face (especially in men) and is laid down in the neck, abdomen, back and breasts (especially in women). This is not so well-recognized in poor-resource settings where malnutrition and HIV-related Slim disease are so common, and the condition appears to be related to the length of time on antiretroviral therapy. There is an associated tendency to type 2 diabetes mellitus. No specific therapy has yet been identified.

(10) **Other** rare malignancies found are embryonal tumours, and Merkell cell carcinoma.

(c) Lymphadenopathy (17.1)

(1) **Persistent generalized lymphadenopathy** has long been recognized as one cardinal feature of HIV disease and represents significant immunosuppression as related by depressed CD4 counts; the presence of epitrochlear lymphadenopathy is virtually diagnostic of HIV affliction.

Typically lymph node enlargement is symmetrical, with small rubbery nodes palpable; these show follicular hyperplasia. Cystic degeneration often occurs, especially in the parotid and submandibular regions. Where nodes are larger, non-symmetrical, matted and firm, other pathology is usually found, principally tuberculosis, Kaposi sarcoma, or lymphoma. These are usually large B cell anaplastic, Burkitt, or aggressive Hodgkin (Grade II) types.

Histoplasmosis in Latin America, *leishmaniasis* in South America and infection with *penicillium marneffei* in Southeast Asia are increasingly common associations of lymphadenopathy and hepatosplenomegaly with HIV. In children with HIV, BCG immunization produces a lymphadenitis.

(d) Oral disease

(1) **Oral candidiasis** (5-10) is a very well-known manifestation of HIV disease, which may present in erythematous, pseudomembranous, hyperplastic forms or angular stomatitis.

ORAL CANDIDIASIS

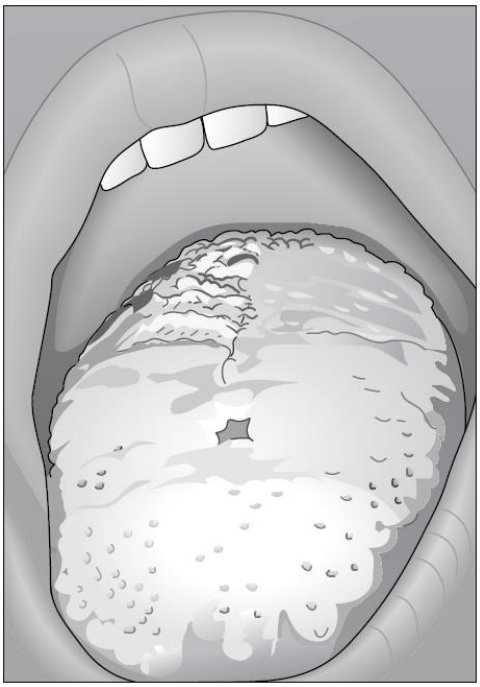


Fig. 5-10 AGGRESSIVE ORAL CANDIDIASIS, often extending into the pharynx and oesophagus.

(2) **White warty projections (hairy leucoplakia)** occurring particularly on the lateral aspects of the tongue and cheeks are diagnostic of HIV disease.

(3) **Periodontal disease** is common: linear gingival erythema worsens to necrotizing ulcerative gingivitis and periodontitis. Advanced necrosis may lead to external ulceration on the cheek, or even to cancrum oris (31.5). In these cases the demarcation of necrosis is usually clear.

(4) **Herpetic ulceration** of keratinizing epithelium is common.

(5) **Recurrent aphthous ulcers** are more severe and long-lasting. Some may be due to *histoplasmosis.*

(6) **Tonsillitis** is common and severe, often with ulceration, either in combination with generalized lymphadenopathy or alone. Development into a tonsillar abscess is not uncommon (6.7).

(7) **Oropharyngeal carcinoma** is 3 times as common with HIV disease.

(8) **Kaposi sarcoma** (31.8) lesions on the palate or gums (5-11) are manifestations of systemic gastro-intestinal involvement. Non-Hodgkin lymphoma is also frequently seen.

KAPOSI SARCOMA ON THE GUMS

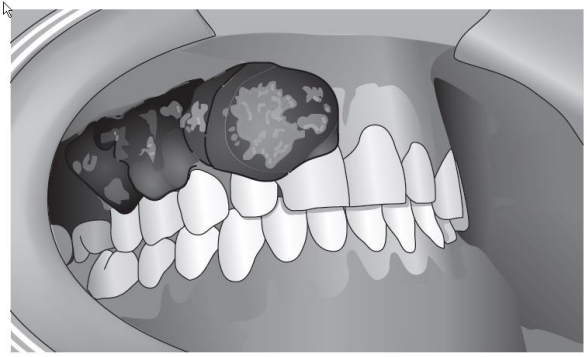


Fig. 5-11 HIV-RELATED KAPOSI SARCOMA, typically on the gums or palate (*remember always to look inside the mouth!*).

(e) Nasal disease

(1) **Recurrent rhinitis and sinusitis** are the consequences of mucociliary dysfunction in the nose and sinuses, with increased atopy, often complicated by bacterial or fungal infection (the latter if CD4 count is <50/μl).

(2) **Nasal tumours**, usually lymphomas or Kaposi sarcoma.

(f) Ear disease

(1) **Hearing loss** of both sensineural and conductive types can occur. There may be direct central neurological damage, effects of HIV directly on the VIIIth cranial nerve, but the causes below are more common. However, *don't forget that anti-TB drugs and ARVs may be directly ototoxic*.

(2) **Acute otitis media**, especially with effusion, owing to obstruction of the Eustachian tube by lymphadenopathy, is frequent and often recurrent, and may result in rupture of the eardrum. Almost all HIV+ve children have had at least 5 episodes by the age of 5yrs, the frequency being related to the drop in CD4 count.

(3) **Otosyphilis** leading to sensineural hearing loss occurs often suddenly with rapid progression in one or both ears: it appears that HIV disease may activate or accelerate pre-existing syphilis.

(4) **Otalgia and facial palsy** (Ramsay Hunt syndrome) is caused by *herpes zoster* affecting the geniculate ganglion; the herpetic rash appears in the ear, and the facial palsy never recovers.

(5) **Otitis externa** is often florid with necrosis, and may be accompanied by invasive fungal infection.

(g) Salivary gland enlargement (17.5)

(1) **Parotid enlargement** is a typical early sign of HIV disease; its cause is varied, including lymphadenopathy (as part of generalized lymphadenopathy), salivary and extraparotid lympho-epithelial cyst formation, and lymphocytic infiltration (due to direct infiltration by CD8 lymphocytes). This may represent a beneficial response to HIV infection, and patients with salivary gland enlargement seem to experience slower progression of the disease. Frequently, unilateral parotid swelling is followed some time later by swelling of the contralateral side.

PAROTID SWELLING

Fig. 5-12 PAROTID SWELLING, often bilateral, of cystic soft texture.
N.B. Nasogastric tube only* in situ *to help nutrition because of orophayngeal and oesophageal candidiasis.

(h) Eye disease

(1) **Keratitis** is a severe, rapidly deteriorating infection involving the cornea caused by bacteria, fungi, *microsporidia*, *herpes simplex* or *herpes zoster*. The cornea is affected in the latter through the nasociliary branch of the ophthalmic division of the Vth cranial (trigeminal) nerve. Progress occurs to multiple small dendritic and then geographic ulceration and frequently to perforation. Healing by scarring may give rise to iris adhesions leading to glaucoma, and inevitably corneal opacification. Once perforation occurs, however, or if a staphyloma develops, the eye is lost.

(2) **Bacterial conjunctivitis** comes as acute or subacute infection, either staphylococcal or gonococcal.

(3) **Conjunctival carcinoma** (28.15) was soon found as a more frequent pathology in Uganda, being first described in Guadeloupe as probably related to HIV, having been noted as an oddity much earlier. *(This pattern mimics the story of Kaposi sarcoma.)*
There appears to be an increased susceptibility to ultraviolet light in the presence of human papilloma virus-16 infection. A small lesion with an irregular surface 2-5mm in diameter appears on the medial side of the limbus usually, spreading onto cornea and underlying sclera. Recurrence after excision is fairly common. Where the tumour extends into the sclera, resulting in necrotizing scleritis, the eye is lost.

(4) **Kaposi sarcoma** appears as a slightly raised pigmented lesion found on the eyelid, conjunctiva or inside the orbit. This may be isolated or multifocal; recurrence after treatment is usual.

(5) **Molluscum contagiosum** consists of raised umbilicated lesions; when they affect the eyelids, they may become large and numerous; an associated follicular conjunctivitis may occur due to viral shedding.

(6) **Cytomegalovirus (CMV) retinitis** is the most common cause of impaired vision in HIV patients: in 30% it is bilateral; early signs are narrowing of the retinal vessels, resulting in perivascular exudation and haemorrhage prior to retinal infarction. CMV is common in those patients who have had TB; it does not appear to occur if *herpes zoster* was contracted earlier. Toxoplasma is a rarer cause of chorioretinitis.

(7) **Keratoconjunctivitis sicca** (extreme dryness of the conjunctiva), reminiscent of Sjøgren syndrome, occurs in HIV patients, and in particular in association with Stevens-Johnson syndrome, and toxic epidermal necrolysis.

(8) **Diffuse lymphocytosis syndrome** occurs as a malignant condition where there is perivasculitis of retinal vessels and lacrimal gland involvement.

(i) Cardiopulmonary disease

(1) **Cardiomyopathy** occurs, often with sudden dramatic cardiac collapse: its aetiology is multifactorial.

(2) **Spontaneous pneumothorax** (36.1) occurs especially in *pneumocystis carinii* pneumonia, which accounts for up to 60% of pulmonary infection in HIV disease. This occurs frequently in conjunction with *cytomegalovirus.* Other infections in the lung are mainly with bacterial pathogens and *mycobacterium*: in low- and middle-income countries, tuberculosis is extremely common as a manifestation of HIV disease. Pleural effusion is a common consequence, and empyema thoracis (9.1) likewise. However, not all effusions are due to tuberculosis: they may be secondary to lymphoma, Kaposi sarcoma, or serious bacterial infection. Open thoracic surgery is fraught with serious pulmonary complications and is ill-advised.

(3) **Tuberculous pericarditis and pericardial effusions** (9.2) are common.

(j) Oesophago-gastric disease

(1) **Oesophageal candidiasis.** Oral candidiasis (5-10, 30.4) is a very frequent manifestation of immune deficiency. Infestation with candida may spread further into the pharynx and oesophagus, where if very copious it will give rise to symptoms of dysphagia. It may be absent in the mouth though present in the oesophagus! Complete oesophageal obstruction can occur.

(2) **Diffuse oesophagitis** may be due to *herpes simplex*, and result in ulceration; discrete ulceration is more likely due to *cytomegalovirus*. There may be profuse haemorrhage. Some of these ulcers are, however, idiopathic. They often result in strictures.

(3) **Tuberculosis** may affect the oesophagus without being present elsewhere; a broncho-oesophageal fistula may result; the oesophagus is too friable to attempt stenting in this circumstance.

(k) The acute abdomen

(1) **Peritonitis** (10.1)
You will see the causes of peritonitis as in HIV-ve patients; HIV-positivity does not of course necessarily imply an HIV-related pathology as the cause. Indeed some common causes of peritonitis, such as gynaecological pelvic inflammatory disease (PID), are more common and more severe in HIV+ve patients. Likewise pelvic abscesses (from any cause, but especially PID) are more common and more extensive.

Nonetheless you may see HIV-related pathologies frequently; these include:
Primary peritonitis: most common,
Spontaneous bowel perforation, especially in the distal ileum (usually due to *CMV*) or colon,
Tuberculous peritonitis (16.1) in the following forms:
Multiple peritoneal seedlings with ascites,
Tuberculous mesenteric lymphadenopathy (with or without ulceration),
Ileocaecal tuberculous mass (tuberculoma),
Tuberculous colitis (mimicking ulcerative colitis),
Tuberculosis of Fallopian tubes and ovary,
Abdominal wall sinus,
Cryptococcal peritonitis, multiple superficial small white nodules seen on the omentum and serosal surfaces,
Mesenteric thrombosis (12.14), usually a venous infarction,
Colitis (in adults), from enteropathic *E. coli* or *CMV*, mimicking amoebic colitis,
Necrotizing enterocolitis (in adults and infants beyond the neonatal period; 14.4),
Acalculous cholecystitis (caused by *cryptosporidium*, *CMV*, *microsporidia*, lymphoma or KS).

In late stages, the gallbladder may perforate, but perforations may be multiple and small with inflammatory exudation.

Other HIV-related conditions may give rise to severe abdominal pain:
HIV-pancreatitis (15.13),
Severe HIV-cystitis,
Retroperitoneal abscess (6.15),
Necrotizing fasciitis of the abdominal wall (6.23),
Abdominal wall abscess,
Intestinal wall haemorrhage from Kaposi sarcoma (mimicking colitis).

There may be a complex mass of adhesions with all of the above, including bowel perforation. Of course tuberculosis may affect any abdominal organ, including the pancreas, liver and spleen. The classic 'doughy' abdomen occurs in c.50% of cases.

An ascitic tap will only demonstrate acid-alcohol fast bacilli (AAFB) in 25% of cases, but a raised adenosine deaminase level helps to confirm the diagnosis. However in areas of high endemicity, a high lymphocyte count in the ascitic fluid would be sufficient to justify TB treatment.

(2) **Intestinal obstruction** (12.2)
Causes of intestinal obstruction may again be non-HIV-related, but specific HIV causes are:
Tuberculous adhesions/mass/intestinal stricture,
Lymphoma of small bowel,
Kaposi sarcoma of small and large bowel,
Mesenteric lymphadenopathy,
Intussusception (12.7).

Tuberculous adhesions are often thick and unyielding (and may be detected as septa on ultrasonography in an ascites-filled abdomen). A tuberculous mass usually occurs in the right iliac fossa but any site may be affected; an intestinal stricture occurs in the ileum in 70%, in the jejunum in 15%, and in both in 15%.

In a few cases, the signs of intestinal obstruction may mimic a paralytic ileus thought to be related to an HIV-neuropathy.

(l) Abdominal mass

(1) **Lymphoma or tuberculoma** is likely to be the diagnosis in a younger patient; tuberculous abscess of the liver or spleen is not rare, but common local conditions should still head the diagnostic list in HIV+ve patients, especially where, as in schistosomiasis, HIV appears to have little impact on the disease pattern.

Don't assume a right iliac fossa mass to be a walled-off acute appendix! Whilst the diagnosis of an abdominal mass follows standard principles, *keep HIV-related conditions in mind*.

(m) Hepato-biliary disease

(1) **Liver abscess** (15.10), especially tuberculous, is not uncommon.

(2) **Hepatitis** is common: either with *hepatitis B* or *herpes* virus, *cryptococcus*, or induced by drugs. Granulomatous hepatitis occurs with fungal infections or *mycobacteria*.

(3) **Hepatoma** is 7 times as common with HIV disease, but the effects of hepatitis virus exposure are probably more important.

(4) **Acalculous cholecystitis** has been discussed previously under "The acute abdomen".

(5) **Cholestatic jaundice** (15.9) may arise from several types of HIV-related pathology:
papillary stenosis,
sclerosing cholangitis,
lymphadenopathy in the porta hepatis, especially TB.
Cryptosporidium and *cytomegalovirus* have been implicated; this is not necessarily a late complication of HIV disease.

(6) **A pancreatic mass** may be tuberculous, lymphoma or adenocarcinoma, behaving more aggressively, often associated with portal vein thrombosis.

(n) Gynaecological disease

(1) **Pelvic inflammatory disease**, **pelvic lymphadenitis and pelvic sepsis** (23.1), especially post-abortal (23.2), are more common and more virulent in HIV disease. They are promoted by the use of intra-uterine contraceptive devices; recurrent miscarriages, primary subfertility due to HIV disease and permanent infertility due to previous infection are very frequent consequences. Recurrent sexually transmitted infections are very common.

(2) **Tuberculous infection of tubes and ovaries** is common. Dense matted adhesions are frequently found with perforation into bladder, small, large bowel or rectum. Low recto-vaginal fistulae unrelated to obstetric trauma are seen in sexually active women and children <5 years.

(3) **Cervical carcinoma** is 10 times more frequent, affects younger females, and is more aggressive; recurrence is common.

(4) **Herpetic vulvovaginitis**, often ulcerative, is common and huge extensive vulval condylomata very often seen. Their presence in young girls does not necessarily imply sexual abuse; longstanding condylomata may however develop into carcinoma.

(5) **Ovarian lymphomas** of Burkitt type are seen.

(6) **Non-specific chronic pelvic pain** has been a difficult but regularly seen problem in HIV patients.

(o) Obstetric problems

Pregnancy worsens the HIV condition if in the late stages; wasting contributes to maternal and perinatal mortality; puerperal sepsis (22.14) is more common and more severe.

Although Caesarean section reduces the transmission of HIV to child, *it is not practical to advocate such a general policy*. Introduce practices to reduce transmission (5.3).

Unusual infections, *e.g.* peritonitis after postpartum tubal ligation, or pubic osteomyelitis after spontaneous labour, are seen.

Puerperal psychosis may be difficult to differentiate from HIV-cerebral encephalopathy.

Good contraception (*and that usually does not mean the contraceptive pill*) is needed in HIV+ve women. *The contraceptive pill is a bad option* because ARV therapy and antibiotics (especially rifampicin) interfere with its absorption and so it becomes far less effective (unless 2 pills a day are taken). Fever, vomiting diarrhoea and the AIDS dementia syndrome also interfere with effective use. The dangers of IUDs are overstated, but Depo-Provera is probably the drug of choice.

(p) Urological disease

(1) **Neuropathic bladder** is a common problem in HIV disease; it may present with irritative symptoms of urgency and frequency, which respond to anticholinergic therapy *e.g.* imipramine. Less commonly there are obstructive symptoms leading to urinary retention. Where the neurogenic bladder is due to Guillain-Barré syndrome or transverse myelitis, expect spontaneous recovery. Otherwise a trans-urethral incision of the prostate provides a remedy in men, and intermittent self-catheterization in women.

(2) **Urethral stricture** (27.9) may also cause acute urinary retention; the stricture is usually more severe than in non-HIV patients. This commonly presents in HIV+ve patients through its complications, namely periurethral abscess and fistula formation. The development of 'watering can' scrotum and perineum is frequent. *Don't perform an open urethroplasty because it has a high complication rate*.

(3) **Fournier's gangrene** (6.21,23) is a very high risk in HIV patients following urethral injury. It may often, however, occur *de novo.*

(4) **Prostatic abscess** (6.19), **tuberculous and non-specific prostatitis** are specific HIV-related problems, frequently resulting in urinary retention. Tuberculous prostatitis mimics prostatic carcinoma completely, even to the extent of giving rise to raised prostate specific antigen (PSA) serum levels.

(5) **Focal segmental glomerulonecrosis** is the commonest cause of HIV-related renal impairment; renal tuberculosis remains rare but consider it in chronic sterile pyuria.

Although common in the HIV population, epididymitis shows no real differences in presentation and treatment with the non-HIV population.

(6) **Tuberculous epididymitis** is however more common; the lesion is typically firm.

(7) **HIV-related cystitis** may be aggressive and extremely debilitating. The predominant symptoms are painful urinary frequency, suprapubic pain and haematuria (micro- or macro-scopic) without any demonstrable urinary tract infection. Cystoscopy reveals a highly characteristic uniformly congested appearance with no ulceration and no significant reduction in bladder capacity. The histological appearance is like a non-specific interstitial cystitis without mast cells, with no *cytomegalovirus* found.

(8) **Urinary tract infections** occur in c.15-20% of males with advanced HIV disease (CD4 <200/μl), most commonly with *pseudomonas aeruginosa.*

All types of sexually transmitted infections are inevitably common in HIV+ve patients, and therefore a combination of diseases is frequent.

(9) **Balanitis co-existing with chancroid, condylomata or with malignancy.** This may be in the form of squamous carcinoma or Kaposi sarcoma. There may be a continuum of histological change from condyloma to squamous carcinoma, suggesting a synergistic interaction between the papilloma virus and HIV. Malignancy of the foreskin however remains rare; frankly necrotic ulcerative penile lesions are usually due to chancroid. Patients may request circumcision (27.29) hoping thereby to avoid recurrent penile ulceration; this may then of course occur on the glans penis itself. The operation of circumcision is not without risk: severe necrotizing fasciitis of the penis can occur post-operatively. There appears, at least in certain cases, to be a microangiopathy associated with balanitis; this may be the predisposing factor in the development of necrotizing fasciitis and it may be exacerbated by increased tension when LA is used in a penile block. You should therefore perform the operation only under GA or using a caudal block.

(10) **Penile abscess** (6.21) *de novo* is diagnostic of HIV infection. The infection usually spreads from the penis to the scrotum, rather than the reverse as in the classical Fournier's gangrene. In the absence of urethral stricture or diabetes mellitus, necrotizing fasciitis of the penoscrotal tissues is likewise diagnostic of HIV disease.

Don't necessarily refuse a request for circumcision in HIV+ve patients on traditional or social grounds; there may be a protective role in HIV transmission in the act of circumcision. The epithelium of the exposed glans penis in the circumcised male changes from columnar to stratified squamous, and may thus be more resistant to ulceration.

However, the morbidity and, in some cases, mortality of circumcision, especially where medical resources are scarce, may make this procedure dangerous.

Don't underestimate the possible complications of elective circumcision, especially if you use an LA ring block: necrotizing fasciitis, abscess and also the risk of haemorrhage, particularly where thrombocytopenia is present, are serious problems. Though these complications are quite common with HIV+ve patients, they can also occur to others.

Recommendations for circumcision to prevent HIV transmission fall in the same category as the use of condoms: although it may help, it does not get to the root of the problem. You must carefully counsel your patient that circumcision does not protect him from HIV infection, but may just lower the risk.

(11) **Condylomata** may be very profuse on the foreskin and may encroach onto the glans penis and into the urethral meatus.

(12) **Erectile dysfunction** is very common in HIV disease, and seems to have a multifactorial origin; treatment with sildenafil and related drugs poses huge moral and ethical issues.

(q) Anorectal disease (26.2)

You will find a variety of anorectal lesions in HIV+ve patients, and their severity relates closely to CD4 levels; in all patients they are common, although they are particularly numerous (c. 30%) in homosexuals, where they have a somewhat different pattern. As many practitioners have a natural reluctance to examine the anal region, they are often referred to as 'piles'; however, haemorrhoids are *per se* not part of the spectrum of HIV anal pathology.

Many of the lesions are resistant to treatment and their aetiology is not known, but this does not mean that you can do nothing for patients with these conditions. However, don't undertake elective anorectal surgery lightly: many authors have reported poor or absent wound healing often after many months. Distal septic complications such as meningitis may also occur.

(1) **Idiopathic anorectal ulcer** appears first as a mucosal laceration within the anal canal, and gives rise to symptoms identical to the classical anal fissure, *i.e.* pain and bleeding *per rectum.* However, you will see no anal skin lesion on gentle parting of the buttocks, because the lesion is internal, usually just proximal to the dentate line. Furthermore there is rarely anal sphincter spasm, and often diarrhoea rather than constipation. Pain is persistent, usually associated with some intermittent bleeding *per rectum*, particularly after defecation. The mucosal defect then deepens and becomes palpable as an ulcer with smooth benign-feeling edges. As this ulcer deepens further, it may penetrate into the vagina or urethra or appear as a large fistula externally. No single agent has been implicated in this lesion, although in some cases *cytomegalovirus*, *chlamydia trachomatis* (26.11), and *herpes simplex* virus have been found. There is commonly associated infection, with patients reporting pus draining *per rectum*.

(2) **Superficial breakdown of perianal skin with excoriation** is often associated with chronic diarrhoea; control of loose stools is therefore obviously important. Vesicular excoriation is due to *herpes simplex*. Careful examination to exclude fistulae and abscesses is vital.

(3) **Fistulae** (26.3) in HIV+ve patients are often complex and multiple. They are frequently high or intersphincteric (intermediate) and are therefore not amenable to simple laying open. In fact, even for low superficial fistulae, the laying open may result in non-healing perianal wounds, especially if the CD4 count is <200/µl. Many fistulae arise from sepsis, but some as a result of extension of the idiopathic anal ulcer described above. In these cases, the fistula is wide and may readily admit the examining finger. Fistulation can occur to the outside skin, but also to the vagina or bladder. This occurs in both adults and small children. If the fistula was not present at birth, it is pathognomonic of HIV disease.

(4) **Anal and perianal warts** (26.6) are often very extensive; their excision or diathermy ablation surprisingly results in rapid wound healing, presumably due to an epithelial growth factor in the papilloma virus. Contact tracing in poor-resource environments is a pipe-dream, and therefore recurrence by reinfection is frequent. Moreover, if not all condylomata are removed, and they can extend far up in the anal canal, they quickly re-establish themselves. *Beware when using diathermy on these lesions: HIV may be transmitted by the smoke,* so always wear a mask and aspirate away the fumes.

PERIANAL CONDYLOMATA

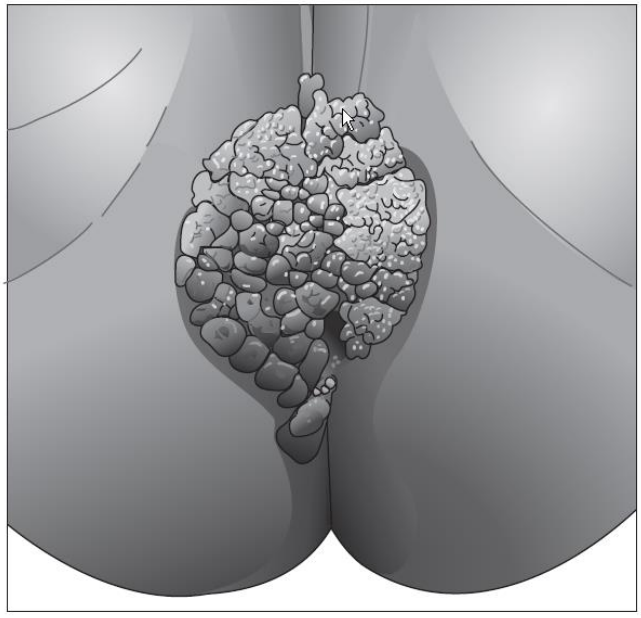


Fig. 5-13 CONDYLOMATA (warts), often extensive with underlying neoplastic change.

(5) **Squamous carcinoma** (26.7) may be heralded by the neoplastic change seen in anal intra-epithelial neoplasia (AIN), or be the result of chronic infestation by condylomata, especially if florid, or arise *de novo*. The incidence of HIV-related anal carcinoma is rising and it is 60 times more common than without HIV disease, but this may be mostly due to the risks of ano-receptive sexual intercourse.

(6) **Lymphoma** and **Kaposi sarcoma** may also be found at the anus.

(7) **Proctitis.** Just as in colitis, the rectum may be affected by a severe inflammatory process; *cytomegalovirus*, *herpes simplex*, *chlamydia* or enteropathic *E. coli* may be the cause.

(r) Vascular disease

Any major artery can be involved; the pathology affects mainly the adventitia with leucocytoclastic vasculitis of *vasa vasorum* and periadventitial vessels, proliferation of slit-like vascular channels, chronic inflammation and fibrosis. There is associated medial fibrosis with loss and fragmentation of muscle and elastic tissue, and similar fragmentation in the internal elastic lamina of the intima, with calcification.

(1) **Arterial occlusion** (35.2) or **aneurysm formation** (35.9) are the end results; the former is much more common, but increasing numbers of aneurysms are seen in HIV+ve patients. Arterial occlusion in limbs results obviously in gangrene; in poor-resource countries, patients rarely present with claudication, and the deterioration of symptoms is usually too rapid to allow early presentation. Thus arterial reconstruction is hardly ever an option; you should also have serious qualms about using prosthetic material in HIV+ve patients as the vessels take sutures poorly, and secondary infection of the graft is a very definite risk, often with fatal outcome. Results of surgery for atheromatous disease (*i.e. not HIV-related*) in HIV+ve individuals may however be more successful.

Thrombosis may also occur in mesenteric vessels, or cerebral arteries resulting in a cerebro-vascular accident. Aneurysms tend to occur in the carotid and superficial femoral arteries, although any artery may be involved and multiple lesions are seen. Spontaneous arteriovenous fistulae also result.

(2) **Deep vein thrombosis** occurs with 10 times greater frequency, though you will detect less than 1% of cases clinically. Risks of surgery are obviously further increased when you take this statistic into consideration, especially as you can use prophylactic anticoagulants only with reluctance in the presence of thrombocytopenia.

(s) Orthopaedic problems

There is increased risk of infection especially when implants are used; you may see late infection long after implants have been inserted, where surgery has often been done before seroconversion. The larger the implant, the bigger the problem: bone infections then often fail to respond to antibiotics, removal of the implant, debridement and subsequent sequestrectomy. *Never put implants in open fractures in HIV patients!*

Thus non-operative methods are usually more suitable, especially when there are obvious signs of immunosuppression (CD4 levels <200/µl); external fixators are preferable if practical. Remove implants as soon as possible, once their effectiveness is over. Explain fully the merits and demerits of internal fixation before you carry out any such operation.

(1) ***Adult*** **bone infection (osteomyelitis)** (7.3) occurs usually in the lower femur or upper tibia, often bilaterally; *staphylococci* are usually found, but *salmonellae* and gut organisms are often seen. Despite appropriate treatments, infections frequently don't resolve and amputation may be necessary.

(2) **Septic arthritis** (7.16) occurs more frequently in HIV disease, especially if joint replacements have been inserted. The knee, hip, shoulder, ankle, elbow and wrist are commonly affected by the same organisms as osteomyelitis.

(3) **Tuberculous arthritis** affects HIV patients similarly to non-HIV: primarily the spine (32.4), and then the hip and knee are involved. Relapse is not uncommon after treatment; except where immune competence is reasonable (CD4 >200/µl), avoid surgery to decompress the vertebral column to relieve paraplegia or arthrodese painful destroyed joints.

(4) **Reactive HIV-arthritis** causes painful swelling and joint effusion, especially of knees and ankles, and may be acute (mimicking septic arthritis) or more insidious, usually bilateral and sometimes migratory; recurrence frequently occurs in the same joint which had been quiescent for months.

The arthritis may arise as a result of reaction of diarrhoea bacterial fragments carried in the circulation: aspiration yields opalescent fluid filled with leucocytes. Chronic debility results with permanent joint stiffness where relapse occurs (often with resolution of physical signs).

Where rheumatoid arthritis, Reiter's disease or ankylosing spondylitis occur with HIV disease, their response to anti-inflammatory drug treatment is usually poor.

(5) **Inflammatory conditions of tendons and ligaments**, *e.g.* tennis elbow, Achilles tendinitis, plantar fasciitis, are common, and usually recur after treatment.

(t) Neurological disease

(1) **Transverse myelitis, motoneurone disease, leucoencephalopathy, progressive dementia, and encephalitis** occur through the strong affinity of HIV for neuronal cells.

UNILATERAL PTOSIS

Fig. 5-14 UNILATERAL PTOSIS, usually without pupillary changes.

(2) **Neuropathies and myelopathies** are common, resulting in facial palsy (especially at seroconversion), ptosis (5-14), impotence, paraparesis, urinary retention or incontinence.

(3) **Opportunistic cerebral infections** with *toxoplasmosis*, *cytomegalovirus*, *herpes simplex*, and *blastomycosis* are common.

(4) ***Cryptococcus*** **meningitis** is a typical manifestation of advanced HIV disease; tuberculous meningitis is more common in HIV patients and often results in secondary hydrocephalus involving the basal cisterns.

(5) **Herpes zoster** may affect the *motor* roots in HIV disease: a claw hand may result.

(u) Haematological disease

(1) **Pyrexia** without obvious cause is frequent.

(2) **Chronic anaemia** is common with bone marrow suppression of single or multiple cell lines. Infiltration of bone marrow with leishmaniasis or toxoplasmosis is seen. There is a drop in levels of interleukin 4 and 5, needed in haemopoiesis.

There is an increased risk of bleeding in HIV disease; when thrombocytopenia is overt this may be catastrophically serious. Platelet numbers may be satisfactory, but their function not so.

(3) **Idiopathic thrombocytopenia** responds to splenectomy, but in HIV patients the risks of *pneumococcal* and other sepsis, including malaria, outweigh the advantages.

All these complications may be correlated to CD4 cell counts and can therefore give an indication of the stage of advance of the disease, and also of its regression with treatment:

CD4 count	**Infectious complication**	**Non-infectious complication**
>500/µl (WHO stage I)	Acute HIV syndrome	Persistent generalized lymphadenopathy Skin rashes Salivary gland swelling Guillain-Barré disease Myopathy Aseptic meningitis Colitis
200-500/µl (WHO stage II) (WHO stage III)	Pneumonias Pulmonary TB Herpes zoster Kaposi sarcoma Oral *candidiasis* Recurrent skin infections Primary peritonitis	Carcinoma cervix B-cell lymphoma Anaemia Mononeuritis multiplex Lymphocytic interstitial pneumonitis Oral hairy leucoplakia Idiopathic thrombo-cytopaenia Gingival erythema Seronegative arthritis Vasculitis
<200/µl (WHO stage IV)	*Pneumocystis carinii* pneumonia Extra-pulmonary/ miliary TB Necrotizing fasciitis Cellulitis Chronic mucocutaneous herpes simplex Oesophageal *candidiasis* Anal ulcer/ perianal excoriation	Wasting Peripheral neuropathy Non-Hodgkin's lymphoma Cardiomyopathy Encephalopathy Myelopathy/ radiculopathy Dementia
<100/µl	Disseminated herpes simplex *Toxoplasmosis/ blastomycosis* *Cryptococcal* meningitis Chronic *cryptosporidiosis* Chronic *isosporiasis* Oesophageal *candidiasis* Respiratory *candidiasis* Salmonella (non-*typhi*) septicaemia	Lipodystrophy

The T-cell profile changes with drop in levels of CD4 cells and rise in CD8, with drop in ratio CD4/CD8 <2; there is a total drop in T-cells late in the disease. However, a T-cell leukaemia may occur with rise in numbers.

(4) **Leucopenia** is common with especially a lymphopenia; a leucocytic response to infection is often not present. However, **leukaemia** can develop after ARV treatment is started.

Immunoglobulins, especially IgE, are raised, and so plasma viscosity goes up, with rise in ESR. Total globulin levels rise, with drop in albumin/globulin ratio.

5.7 HIV and tuberculosis

Tuberculosis (TB) can affect any organ in the body, and so is found in surgical patients either as coincident pulmonary disease, or as primary cause of their complaint (*e.g.* TB lymphadenitis (17.4), TB arthritis (32.3), abdominal TB (16.1), gluteal sinuses, epididymal TB (27.23) etc.).

The advent of HIV disease has severely increased its incidence throughout the world, and in many countries TB is a strong indicator of HIV disease. You may see bovine TB where immunization of cattle and pasteurization of milk are not routine.

Extra-pulmonary TB is an even stronger indicator of HIV. It may be difficult to detect, and not be associated with pulmonary TB, so use focused ultrasound: look for ascites, micro-abscesses in the liver or spleen (38.2b,g), para-aortic and periportal lymphadenopathy (38.2h), pleural or pericardial effusions, often with fibrinous strands visible.

Test for HIV in every TB patient, and suspect both in prisoners. Dissemination is more common as the CD4 counts fall <200/µl. There may be TB outside the lung without it being inside the lung!

The clinical diagnosis of TB can be difficult; especially in lymph nodes, but also in pus and other solid organs, aspiration for AAFB by direct smear microscopy or using Ziehl-Neelsen (ZN) staining is useful, especially if histology is not available. Use simpler cold staining methods: flood the smear with concentrated carbol fuchsin for 10mins without heating, and wash with water; then flood the smear with Gabbet's methylene blue for 2mins and again wash with water. Dry the smears as for ZN staining. Recent methods (Gene Xpert) detecting DNA sequences by using a polymerase chain reaction (PCR) through nucleic acid amplification tests are very sensitive even in HIV+ve patients and can detect rifampicin resistance; if the equipment is available, the cartridges are now inexpensive, easy to use and recommended by WHO.
The average delay before TB is diagnosed is 18 months!

Gabbet's methylene blue: Methylene Blue 1G, Absolute Alcohol 30ml, Concentrated Sulphuric Acid 20ml + Distilled Water 50ml.

Naked eye appearances of caseation are virtually diagnostic, but may be confused with necrotic lymphoma. Tuberculin (Mantoux and Heaf) testing is no longer reliable, except for children <3yrs who have not had BCG.

In areas of high TB endemicity, you may be able to diagnose TB by a lymphocytosis on pleural fluid, pericardial fluid. The main differential diagnosis is cancer or brucellosis.

It is important to screen sputum also for AAFB ('open PTB') in every patient for good infection control, especially in the community. A chest radiograph may not show classical apical disease, but rather lower lobe infection; there is less cavitation and a miliary pattern is common (especially when the CD4 count is low). It is important to perform radiography when treatment is finished, and to file films properly for future cross-reference.

Don't start treatment without arranging contact tracing, especially for babies and infants, and notification.
Special charts are available in many countries. Follow your regional regime or, otherwise, the WHO-approved standard, using a directly observed treatment scheme (DOTS):

Intensive Phase: 2 months isoniazid (H), rifampicin (R), pyrazinamide (Z), and ethambutol (E), followed by
Continuation Phase: 4 months HR.

Extend this continuation phase to 5 months for TB epididymitis, 6 months for spinal TB with neurological problems, TB pericarditis and meningitis, and 9 months for renal TB. (An alternative is 6 months of isoniazid and ethambutol.) *Don't use ethambutol in children <10yrs.*

If the patient has had treatment before, initial treatment is probably best in hospital: use longer treatment phases:

Intensive Phase: 2 months streptomycin (S), plus HRZE, followed by 1 month HRZE, and then
Continuation Phase: 4 months HRE.

Don't use streptomycin in pregnancy; or (and ethambutol) in children <10yrs. Thiacetazone is no longer routinely used.

Dosages are weight-dependent: as the patient improves and he gains weight, so you may need to alter the dosage; these are daily oral doses:

Weight	Isoniazid (H)	Rifampicin (R)	Pyrazinamide (Z)	Ethambutol (E)	Streptomycin (S)
5-9kg	50mg	75mg	250mg	-	-
10-20kg	100mg	150mg	500mg	-	-
21-33kg	200mg	300mg	1000mg	800mg	500mg
34-50kg	300mg	450mg	1500mg	800mg	750mg
>50kg	300mg	600mg	2000mg	1200mg	750mg

Fixed dose combinations may be available, and help patient compliance; twice- or thrice-weekly regimens are being introduced to make DOTS easier, but dosages will then differ. Resistant TB may be as common as 15%, and needs 20 months' treatment.
Control neuropathy with isoniazid with pyridoxine (vitamin B6) 50mg tid; prophylactic treatment is 20mg od.

Most of the problems with HIV treatment occur with rifampicin, which induces liver enzyme breakdown of ARVs, making them less effective; generally start antiretrovirals after the intensive 2-month phase of TB treatment. Rifabutin is an alternative to rifampicin.

If the CD4 count is <50/µl, however, start ARV treatment as tolerated, but avoid nevirapine and substitute efavirenz which is much more expensive. Otherwise start ARV treatment after 2wks of anti-TB therapy.

The main drawback with TB drugs is their side-effects, which are often shared by antiretroviral drugs:

Toxicity	***TB Drug***	***ARV Drug***
Neuropathy	H	d4T, ddC, ddI
Hepatitis & rash	R, H, Z, S	NNRTIs
Nausea	Z	ddI, AZT, PIs
Visual loss	E	-
Hearing & balance loss	S	-

(N.B. Names of ARV drugs in 5.8)

(Note, however, that visual loss is usually the result of CMV retinitis rather than a side-effect of TB drug therapy.)

If a patient is already on ARV treatment when you diagnose TB, *don't stop the ARV drugs!*

If patients don't complete their treatment courses, or if many different treatment regimens are used, resistant strains are likely to develop. Multi-resistant TB has surfaced in some parts of the world: 2 months of streptomycin are then recommended but get specialist help; the possibilities of untreatable TB, if combined with HIV, would be disastrous. For these reasons, prophylactic treatment of HIV+ve patients with isoniazid is only recommended where tuberculosis is not so prevalent: *follow national programme guidelines!*

5.8 Treatment

The virus multiplies at an alarming rate: within a week of seroconversion there are 10^{7-8} RNA copies/ml. In 6-12 months the viral load reaches an equilibrium where it can usually be maintained by medication for several years.

ARV therapy has proved remarkably successful, though eradication of viral reservoirs has not been possible. You should maintain long-term treatment, though this is still expensive; however, costs have come down dramatically through WHO campaigns, so use this resumé if you can. Unfortunately still c.10% of people with HIV needing treatment worldwide are not getting it.

A willingness and commitment to long-term therapy is essential; consider the financial costs, and the potential barriers ahead. Treat co-morbidities, and manage psychosocial issues: *the drugs are not the whole story!*

A combination of drugs is necessary; otherwise early drug resistance is inevitable, and further treatment practically impossible.

Reduction of viral loads by 70-80% is usually possible with at least 95% adherence to drug regimes, but about 30% of patients default treatment.

Agents can be divided into:

Type	**Abb^{n}.**	**Function**	**Name**	**Abb^{n}.**
Nucleoside Reverse Transcriptase Inhibitors	NRTI Cat I	Mimic normal building blocks of HIV-DNA	stavudine zidovudine emtricitabine	d4T AZT FTC
	NRTI Cat II		didanosine lamivudine zalcitabine	ddI 3TC ddC
	NRTI Cat III		abacavir	ABC
Nucleotide Reverse Transcriptase Inhibitors	NtRTI	*as NRTIs*	tenofovir	TDF
Non-Nucleoside Reverse Transcriptase Inhibitors	NNRTI	Directly inhibit early stages of replication	delavirine efavirenz nevirapine etravirine	DLV EFV NVP ETR
Protease Inhibitors	PI	Directly inhibit last stages of replication	amprenavir indinavir nelfinavir ritonavir saquinavir atazanavir lopinavir darunavir	APV IDV NFV RTV SQV AZV LPV DRV
Dideoxy-Nucleotide Reductase Inhibitor		Promote NRTI activity	Hydroxy-urea	

The main drawback of these drugs is their side-effects and interactions with other drugs (especially TB treatment, which often means taking 6 or more drugs), and hence their tolerability.
Avoid efavirenz during pregnancy.
Certain combinations are contraindicated because they either work as antagonists, or are excessively toxic to peripheral nerves or bone marrow.

Other toxic side-effects, potentially fatal, are: pancreatitis, hypersensitivity reactions (including Stevens-Johnson syndrome) and lactic acidosis (with d4T, ddI or AZT): this presents with abdominal pain and dyspnoea.
Long-term side-effects are lipodystrophy, osteodystrophy and insulin resistance.
Occasionally you will need to substitute one drug for another of the same type: get advice about this.
Nonetheless you should warn patients of potential side-effects, *e.g.* vivid dreams with starting EFV, rash with NVP, anaemia with AZT, peripheral neuropathy with d4T or ddI.

Recommended initial therapy is one NRTI from Category I, one from Category II, and one NNRTI. Use an extra NRTI from Category III if the viral load is <55,000 copies/ml: monitoring of viral load and CD4 counts is important.

Previously, treatment was only started if the CD4 count was <200/µl or <500/µl, though evidence now suggests it may be better to start whatever the count. Treatment is necessary regardless for clinical stages III and IV, co-infection with TB or hepatitis B (include TDF and 3TC or FTC), pregnancy (avoid AZT if Hb is <80g/L) or where the partner is HIV-ve.

Other combinations are 3 NRTI; 2 NRTI + PI; 2 NRTI + 2PI; or NNRTI + PI. *Don't use two NRTIs alone*; PIs are metabolized by cytochrome P450 which is also inhibited by ritonavir. This enhances their efficacy if used in combination, but adds significant side-effects, especially metabolic. Combination drugs are available. Both d4T and ddI are being phased out because of their toxicity.
Follow nationally agreed guidelines, as with TB therapy.

New types of drugs are *fusion inhibitors* and *integrase/CCr-5 inhibitors*.

Screen patients for anaemia, TB, hepatitis B and syphilis and treat these before starting ARV therapy. Try to get renal and liver function tests done. *Don't forget to do the pregnancy test in women of reproductive age!*

Treatment for children should also follow nationally agreed guidelines: use first-line therapy with RTV-boosted LPV for babies <3yrs and EFV for those >3yrs, together with 2 NRTIs.

Sudden discontinuation of all therapy usually results in viral rebound within a month with 'wild-type' HIV strains; discontinuation of therapy even when no detectable virus is found for 3yrs almost always results in viral rebound in 3 months. This means that if you have severe complications after abdominal surgery resulting in your patient not being able to take his medication, all the problems described may emerge. Consider carefully therefore before you undertake complex elective procedures in the abdomen.

Recurrence of HIV-related disease, if no antiretrovirals are used, is virtually inevitable with many complications, but especially so with TB and malignant disease such as Kaposi sarcoma or lymphoma. Co-infection with hepatitis B hugely increases the mortality.

Change of therapy may be needed if there is intolerance to drugs, deterioration of the patient's condition, and drug interactions. This, however, is a very specialized field!

Treatment to reduce mother-to-child transmission (if the mother is HIV+ve but not on treatment) is now standardized: AZT bd (or AZT + NVP, as monotherapy may mask neonatal HIV positivity at 6wks of age) at 14wks gestation or as early as possible afterwards.

Further, use infant doses of AZT and NVP to the new-born baby (in liquid form) from 48 and 72h after delivery, till 1wk after ceasing breastfeeding. For babies under 2kg, reduce the dosage. If the baby vomits <1hr after taking the medication, repeat the dose. Should the baby be born <2h after the mother had her dose, supply an immediate dose to the baby, and repeat this at discharge.

Post-exposure prophylaxis (PEP): after a sharps injury or splash onto mucosal surfaces, wash the affected part immediately in warm water. For a case of rape, obtain specimens, but *don't use a douche*. Clean a human bite wound by copious rinsing. Significant injuries warrant ARV treatment: a single drug regime of AZT 200mg tds for 4wks reduces the risk of seroconversion by 80%, but adding another NRTI drug (*e.g.* 3TC 150mg tds) will further reduce the risk. Severe injury, gang rape, rape with immediate life-threatening injury, or inadvertent HIV-affected blood transfusion warrant addition of a PI drug also despite side-effects.

Obviously HIV testing of the victim and the source is mandatory; stop treatment if an HIV-ve result is confirmed from the source (but this may still only signify the 'window' period). An HIV quick test may be -ve even with high viral loads in the period of early seroconversion. A hospital policy on PEP is advisable: some may claim a needle injury otherwise to obtain antiretroviral drugs surreptitiously.

Adjuvant therapy: in Kaposi sarcoma and HIV-related lymphoma, chemotherapy will not eliminate malignancy, so you should give antiretroviral therapy in addition. *If you don't, recurrence is inevitable*.

You might also consider antiretroviral treatment when inserting metal into bone, in order to prevent septic complications, but this area is still controversial.

6 The surgery of sepsis

6.1 'Where there is pus, let it out'

Draining pus is the commonest surgical operation in low- and middle-income countries all over the world. It is also one of the most useful and usually one of the simplest. Quite a small district hospital can expect to drain 200 large abscesses each year, some containing up to 3 litres pus. Although pus can collect almost anywhere, particularly important sites are the breast (6.13), muscles (7.1), bones (7.2), joints (7.16), hand (8.1), pleura (9.1), peritoneum (10.1), and eye (28.3). The most serious consequence of pyogenic infection is septic shock.

Why sepsis of all kinds is so common is not altogether clear, but malnutrition, anaemia, HIV, diabetes and poor hygiene may all play a part. Over 50% of patients with surgical sepsis are malnourished (with protein and calorie deficiency): the malnutrition is either primary or arises because of the sepsis. It may not be immediately visible in fat patients! This malnutrition increases the risk of further infection, pressure sores (34.16), pneumonia and multi-organ failure.

Abscesses are more common in children and young adults, and a patient may have a dozen or more at the same time. *Staphylococci* are almost always responsible, except in the perineal and perianal region, which is commonly infected by coliforms and anaerobes. Some abscesses are tuberculous (5.7), or from actinomycosis. In the presence of HIV disease, Gram-ve organisms may be responsible at any site.
Initially, when there is cellulitis (bacteria multiplying in the tissues), antibiotics will be effective. Infection should then abate within 24h, or develop an abscess, which needs draining. Before pus has collected, drainage is not possible. Antibiotics and drainage thus both have their proper time and place, and *you must not confuse them*.
The tighter the space for an abscess, the more urgent the need for drainage. If a patient has pus in the bones, joints, tendon sheaths, or the pulp space of the fingers, draining it early is particularly urgent. Elsewhere, you have more time, but then pus may not present itself so obviously! If pus gathers in loose tissues near the surface of the body, you can usually detect fluctuation.

But you will not detect fluctuation, or only detect it very late, if pus is under tension in some tight compartment, or if it is inside a large fatty region such as:
(1) the pulp spaces of the fingers or toes (8.5),
(2) the fascial spaces of the hand (8.12) or foot (8.17),
(3) the ischiorectal fossae (6.17),
(4) the lobules of a woman's breast (6.13),
(5) the neck or iliac regions (6.16),
(6) the parotid gland (6.10).
Incise abscesses in any of these places without waiting for fluctuation, or for pus to point.

For fluctuation to be a useful sign, a minimum quantity of pus must be present, and it must be near the surface. *Don't wait till a huge bag of pus has formed and much tissue has been destroyed. Use a needle to aspirate: you will be surprised how often you find pus!* Even if you don't find pus, an incision will allow infection to drain more readily (by the path of least resistance).

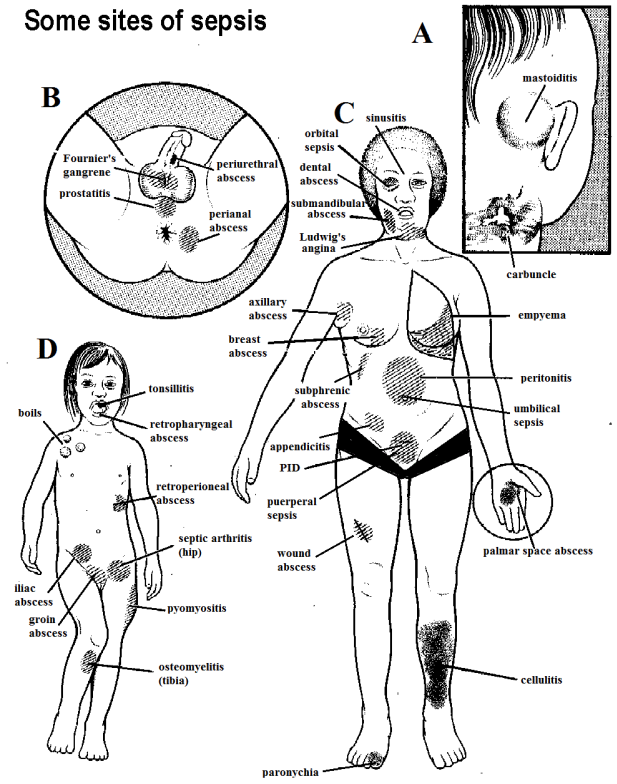


Fig. 6-1 SOME SITES OF SEPSIS. Pus can gather almost anywhere, but here are some of the commoner places: A, behind a child's ear. B, in the male perineum. C, in an adult female. D, in a child.

WHERE THERE IS PUS, LET IT OUT

6.2 Abscesses

The typical symptom of an abscess is severe throbbing pain. The infected part is tender *(dolor)* and swollen *(tumor)*, and the skin over it stretched, shiny, and red *(rugor)*, although this may not be evident on pigmented skin. Touching an abscess is acutely painful. If it is large or there are several abscesses, fever, weakness, toxaemia, and anaemia may be present. The usual signs of inflammation and suppuration suggest the diagnosis, but *don't necessarily expect to find fluctuation in the sites where pus is in a tight compartment*. Severe pain is a useful sign that an abscess is ripe for incision, but pain may be mild when the tissues are loose. If diagnosis is difficult, try aspirating it with a syringe and a wide bore (1·5mm) needle, but remember that pus may be present *even if you fail to aspirate any*. Always aspirate a lump if there is the slightest hint of diagnosing an abscess: tubercular abscesses are often *not* warm, hence the term 'cold abscess', but they are not actually cold! *Never try to treat an abscess by one aspiration alone*. An ultrasound scan may be useful in detecting a localized fluid collection.

Even if an abscess has ruptured spontaneously, adequate drainage by incision is necessary. *There is no need to curette the walls of an abscess*, except in the hand where you want inflammation to resolve particularly rapidly and completely. Suspect a wound abscess if a suture line becomes indurated and tender; it may not be fluctuant (11.13).
So incise an abscess adequately and let the pus flow out; break down any septa in a large cavity and open up any smaller cavities (loculi) off the main one.

Abscesses are often placed at the end of an operating list of otherwise 'clean' cases, and are often left to very junior staff. They are often not treated as genuine emergencies, even though the great risk of septicaemia is ever present. *Don't therefore underestimate the seriousness of abscesses!*

Nevertheless, *be careful:*
(1) The diagnosis can be difficult, *e.g.* an iliac abscess (6.16).
(2) Drainage has its risks, especially severe bleeding when there is a large abscess or many of them, so watch blood loss carefully.
(3) A superficial abscess over the tibia, femur, or humerus may turn out to be pyomyositis (7.1) or, more seriously, osteomyelitis (7.2).
(4) A 'chronic abscess' may turn out to be a solid tumour. Some cancers may present as infections (24.4; 34.15).
(5) *Don't forget the possibility of TB!*

ULTRASOUND will readily demonstrate a collection of fluid: use this if you can when the diagnosis is unclear.

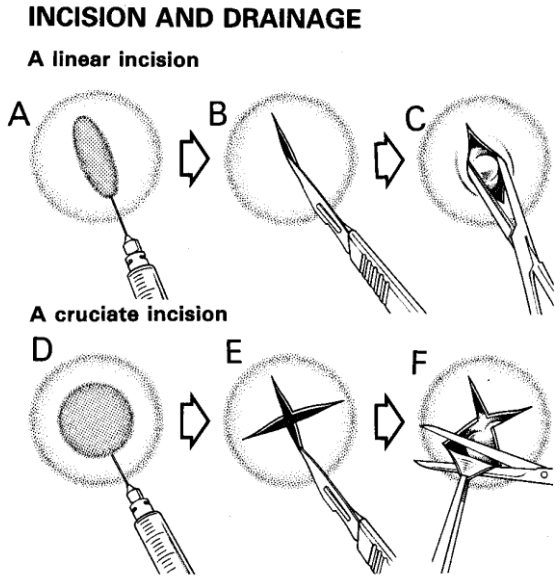


Fig. 6-2 INCISION AND DRAINAGE (I&D).
A-C, linear incision being made and its edges spread. D-F, cross-shaped incision, cutting off the edges of the skin, and so removing the roof of the abscess. *After Hill GJ. Outpatient Surgery, WB Saunders 1973 Fig. 5.12, with kind permission.*

EXAMINATION. Assess the general condition carefully, especially if there are many abscesses, or large ones. Look for anaemia.

SPECIAL TESTS
(1) **If the infection is severe**, take blood cultures. You may be able to isolate the causative organism (this is important in osteomyelitis).
(2) Test the blood or urine for sugar; this may be the first presentation of diabetes: always do this if there is more than one septic infection.
(3) **If there is a particularly large or unusual abscess** (especially in the hand in the absence of trauma or penetrating foreign body, thyroid, muscle, abdominal wall, retroperitoneal space, penis and scrotum, or in the breast of a non-lactating woman), or recurrent ones, test for HIV.

ANTIBIOTICS *are not usually needed*. Use them *only* if:
(1) there is a severe constitutional disturbance with high fever and toxaemia;
(2) there are signs that the infection is spreading: increasing erythema, cellulitis, lymphangitis, severe lymphadenitis, or fever;
(3) the abscess is in the groin (a 'bubo') related to *chlamydia* (lymphogranuloma venereum): use doxycycline;
(4) the abscess is deep-seated, *e,g.* in the brain or liver (15.10).

DRAINAGE OF AN ABSCESS (GRADE 1.2)
INDICATIONS. A collection of pus anywhere accessible.
If you suspect that there is a foreign body in an abscess, this is an added reason for exploring it. Try to remove the offending object and drain the cavity at the same time.
If you are not sure if pus is present or not, aspirate the lesion with a wide bore needle to see if you can withdraw pus. If pus is present, drain it.
If you fail to aspirate pus with a needle, *this does* **not** *mean that there is no pus present!*
Signs that an infection is spreading are not a contraindication to drainage; if you suspect pus is present, drain it.

ANAESTHESIA
(1) You don't need muscular relaxation, so ketamine is very suitable.
(2) If an abscess is already pointing, but the superficial skin is not paper-thin, you can infiltrate the skin at the site of the incision with LA (6-2A). Alternatively you can infiltrate all around the circumference of the abscess, if this is not too big.
(3) Use morphine or pethidine beforehand if the abscess is big or in a sensitive spot, especially for inpatients.
(4) Ethyl chloride local spray is the least satisfactory, but you can use it for very superficial abscesses where the skin is so thin that LA infiltration is virtually impossible. It makes the tissues hard and difficult to incise.
(5) For babies <6-9 months, a quick incision is safer (and kinder) than multiple needle pricks to establish LA or GA.
(6) IV diazepam with pethidine has the hazards of a proper GA and requires all the usual precautions, and *has no advantage over ketamine*.

INCISION
Drain the abscess at the site of maximum tenderness and try to follow Langer's lines (34-1E).
If an abscess is superficial, use a pointed (#11) blade (6-2).
CAUTION!
(1) **If the abscess is deep**, try to incise parallel to any nerves or vessels, not across them.
(2) *A common mistake is not to make the incision large enough*, so extend the incision the whole diameter of the abscess!

HILTON'S METHOD is indicated if there is anything near the abscess which you might possibly injure. Incise the tissues down to the deep fascia; then push blunt scissors or a haemostat into the softest or most prominent part of the swelling. Open them out inside the abscess. If necessary, enlarge the wound by blunt dissection inside the tissues.

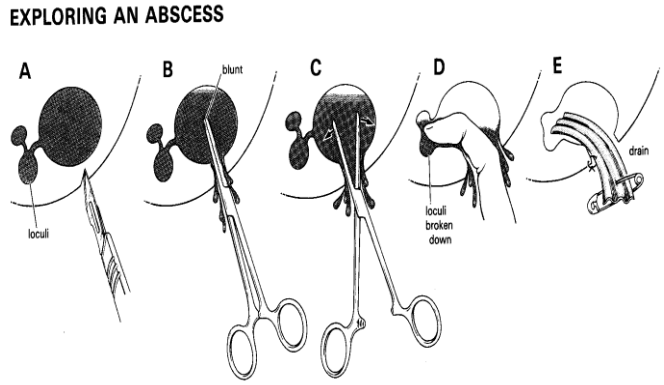


Fig. 6-3 EXPLORING AN ABSCESS BY HILTON'S METHOD. A, incise the abscess at its lowest point, if this is practicable. B, push blunt scissors or a haemostat into it. C, open the haemostat. D, explore the abscess with your finger. E, insert a drain.

DRAIN THE PUS by putting your finger into the abscess, and breaking down all the loculi, so that there remains only one cavity. Use your little finger if the abscess is small.

If there is much pus, suck it out or clean out the cavity with a swab. Make sure you remove all the pus: rinse the cavity thoroughly with water: you may need to squeeze for some time till all the pus comes out: this is painful for the patient so use adequate analgesia. *Provide free drainage*. Make sure that any more pus which collects can drain from the bottom of the cavity.

If the abscess you are draining has a tendency to heal over and leave a cavity, deroof it (6-2F). This is especially necessary with perianal (6.17) and Bartholin's abscesses. Cut away some skin, particularly any dead skin. Allow drainage with a soft rubber drain with a suture to hold it in place.

If pus has to drain downwards, as in the breast, try to incise the lowest part of the abscess. This is better than making a counter incision at its lowest point, and it also avoids making 2 incisions.

If the drained abscess site bleeds, pack the cavity (3.1). If necessary, infuse IV 0.9% saline. You rarely need to transfuse blood unless there are multiple abscesses or severe pre-existing anaemia.

GENERAL MEASURES. **If the abscess is in some critical place**, such as the lateral pharyngeal space (6.9), or the mid-palmar space (8.9), admit the patient. Make sure the fluid intake is adequate, and *don't forget to supply an analgesic*: abscesses are painful!

POST-OPERATIVE CARE. Rest the affected part, and where possible raise it. For example, put the hand in a St John's sling, or, for an inpatient, raise the hand in a roller towel.

ALWAYS INCISE AT THE POINT OF MAXIMUM TENDERNESS

If the foot is infected (8.17), raise the foot of the bed. Make sure your nurses wash the abscess cavity and *don't merely put a dressing on the surface*: the wound will then close over the cavity and the abscess will recur. Make sure free drainage remains possible.

DIFFICULTIES WITH ABSCESSES
If there is severe prostration without a fever, suspect that resistance to infection is low and treat with particular care. Check the HIV status.

If there are many abscesses, with pyaemia, multiple sites of pyomyositis, or septicaemia, bleeding may be profuse when you drain the abscesses. For anaemia, transfuse pre-operatively and, if necessary, again during the operation. Draining multiple abscesses is a major procedure, particularly if a child is severely anaemic or malnourished, so be careful before you incise too many abscesses at one sitting: *children have been known to bleed to death!*

If there is a huge abscess in a very ill patient, he will not tolerate an extensive procedure. It may occasionally be necessary to take him to the theatre several days in succession for repeated drainage, slowly increasing the exposure.

If an abscess fails to heal, *don't forget the possibility of* diabetes, TB (5.7) or HIV (5.6), or a combination of these, syphilis, an underlying tumour, or occasionally actinomycosis (which produces yellow so-called 'sulphur' granules). In Southeast Asia, think of melioidosis (6.10). Check that no foreign body, *e.g.* part of a drain or suture, has been left *in situ*.

N.B. The best instrument to find and pull out a suture knot in a chronically infected wound is a crochet hook!

If you suspect a TB or syphilitic lymph node abscess, perform an ULTRASOUND and look for perinodal fluid, necrosis of the lymph node and calcification. Drainage is not necessary and will only risk causing a non-healing sinus *if you don't treat the underlying cause.*

6.3 Pustules (Boils)

Pustules, as well as carbuncles (6.4), are contagious skin infections which are usually caused by penicillin-resistant *staphylococci*. There may be a crop of them, and in a closed community they may become epidemic.

Clean the skin round the boil with water, and cover it with a dry dressing. Let it burst spontaneously. If it is pointing, a small incision will let it discharge and will reduce the pain. You can use a sterile needle to do this.

CAUTION! Never squeeze a pustule; especially on the face, never let the patient squeeze it.

If there are many pustules, advise washing thoroughly with soap and water, and to shower bd. The bath, shower and toilets must be clean. Advise a daily change of underwear, and washing it by boiling. Exposure to the sun is one of the best cures. *Avoid using 'roll-on' deodorants.*

6.4 Carbuncles

A carbuncle is typically the result of neglected skin infection in a dirty, malnourished, and underprivileged patient, particularly a diabetic or one with HIV. A staphylococcal infection starts in one of the hair follicles, usually at the back of the neck or on the back of a finger (8.1), and then spreads. In doing so, the infection lifts the skin above it on a sea of necrotic fat and pus. At presentation, pus will probably be discharging. *Antibiotics don't cure a carbuncle*, although they may stop it spreading. You will probably have to let the slough separate slowly, and then remove it.

Be sure to test the blood or urine for sugar. Consider HIV testing.

If a collection of pus forms, cut down on it and drain it.

If the skin around the carbuncle is hairy, shave it with as little trauma as you can. Wash it with water, apply dry gauze, and change this frequently. A large slough will form in the middle of the carbuncle. You may be able to lift the slough off painlessly without an anaesthetic.

If the slough is slow to separate, excise it, and apply a dressing of petroleum jelly gauze.

If the bare area is large, apply a split skin graft, as soon as it is clean and granulating.

If a black central pustule with surrounding vesicles forms, consider ANTHRAX and treat with penicillin IV.

If multiple pustules enlarge and spread exuding 'sulphur granules', this is botryomycosis (*not a fungal infection, though it looks like mycetoma*): treat this with debridement and longer-term broad spectrum antibiotics.

6.5 Extradural abscess

Pus may gather between the skull and dura as the result of:
(1) The spread of infection from sepsis nearby.
(2) Exposure of the bone as the result of an injury.
(3) Metastatic spread from elsewhere in the body.

If the abscess is large, there will be fever with signs of raised intracranial pressure (impaired consciousness and pupillary changes) and localizing motor signs, usually on the other side of the body, but not always so. Locally, there may be a diffuse inflammatory oedematous swelling of the scalp over the lesion (Pott's puffy tumour). If the abscess is not so large, the only symptoms may be confusion. Making burr holes should be one of your basic skills, so draining the pus should not be too difficult.

If you have limited imaging facilities, your problem will be to diagnose an extradural abscess in the first place and to know where it is: the abscess is underneath the swelling.

SPECIAL TESTS: The skull radiograph will only show changes if an extradural abscess is chronic, or if there is osteomyelitis of the bone. If you can perform a carotid arteriogram (38.1), this will localize the abscess beautifully.

DRAINAGE (GRADE 3.3)
Drain the extradural abscess through a burr hole. Make this on the edge of the area of swelling on the skull (where present), and nibble away the skull around it until the abscess is well drained.

If the abscess is secondary to osteitis, and there is a sequestrum, removing it will drain the abscess adequately. Likewise, if it is secondary to a neglected compound depressed skull fracture, elevation of the bone fragments will locate and drain the abscess.

6.6 Infections of the orbit

Acute suppurative infection is common near the eye, especially in children. It can occur in front of or behind the orbital septum. This is a sheet of fibrous tissue which stretches from the edges of the orbit into the eyelids, and divides the periorbital region from the orbit. Infections of both these regions usually start acutely with erythema and oedema of the eyelids; distinguish between them as described below. The danger with any infection in this region is that infection may occasionally kill the patient by spreading to the cavernous sinus or the meninges.

(a) Periorbital cellulitis occurs in front of the orbital septum, is more common than orbital cellulitis and occurs in younger children. It can be primary, or secondary to: (1) local trauma, (2) skin sepsis, (3) a recent upper respiratory infection often with *H. influenzae* (associated with bacteraemia).

(b) Orbital cellulitis occurs behind the orbital septum, and is less common but more serious. It is usually due to spread from the paranasal, commonly the frontal or ethmoid, sinuses.

(c) Subperiosteal abscesses may form when bacteria spread from the adjacent sinuses.

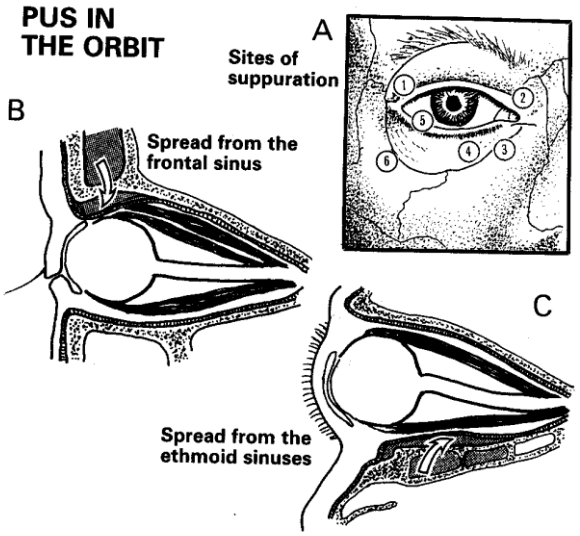


Fig. 6-4 PUS IN THE ORBIT.
A, some important infections around the eye. B, pus spreading under the periosteum from the frontal sinus. C, pus spreading under the periosteum from the ethmoid sinus.
(1) lachrymal gland (dacryoadenitis). (2) frontal sinus and anterior ethmoidal air cells (sinusitis). (3) tear sac (dacryocystitis). (4) tarsal cysts. (5) stye (hordeolum). (6) periostitis of the margin of the orbits and suppurating tarsal cysts can occur anywhere on the lids, and periostitis anywhere in the orbit.
After Dudley HAF (ed) Hamilton Bailey's Emergency Surgery, Wright 10th ed 1977 Figs 187-9, with kind permission.

(d) Cavernous sinus thrombosis can be:
(1) Occasionally, aseptic as a result of trauma, tumours, or marasmus.
(2) More commonly, septic as a result of the spread of infection from the nose (a nasal furuncle is the commonest source), face, mouth, teeth, sphenoid or ethmoid sinuses, the middle ear, or the internal jugular vein.

A cord of thrombus spreads from the site of the infection to the cavernous sinus, and sometimes to the cerebral veins and meninges to cause:
(1) A rise in pressure in the veins draining the eye, resulting in severe oedema and proptosis.
(2) Paralysis of the IIIrd, IVth, VIth (commonly) and the first 2 branches of the Vth cranial nerves.
(3) Meningeal irritation.
(4) Depressed conscious level. If treatment starts late, visual impairment, ocular palsies, and hemiplegia may result.

RANGIT (60yrs) was admitted with a history of septic teeth for many years. Recently he had had fever, headache, rigors, and gradual swelling of the mandible. He was ill, dehydrated, shocked, jaundiced, and confused. Pus discharged from his mouth, the submental glands were enlarged, the neck was stiff, and Kernig's test was postive. Both globes were proptosed, particularly the left, which was fixed; the forehead and cheek were oedematous, and the CSF turbid. Despite vigorous antibiotic treatment he died. Postmortem examination revealed left dental and mandibular abscesses; the left orbit and cavernous sinus were full of pus.
LESSONS (1) This is a very dangerous condition. (2) Proptosis in the presence of facial sepsis is a sign of danger. (3) The organisms responsible are often penicillin-resistant.

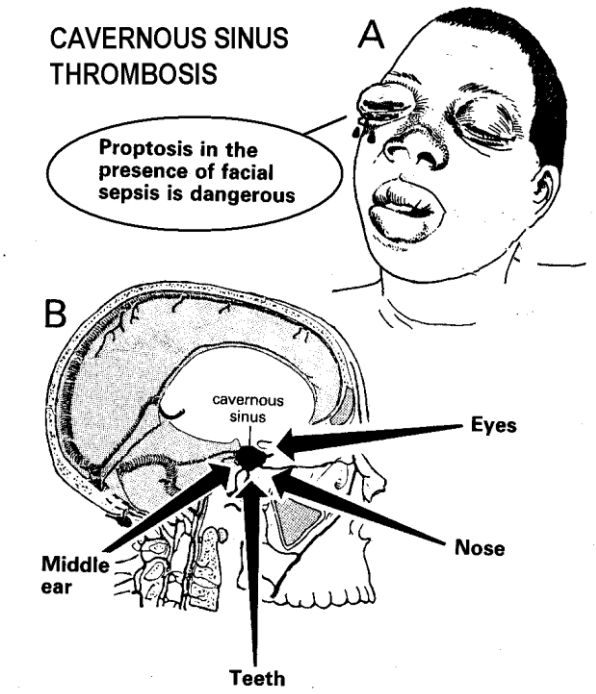


Fig. 6-5 CAVERNOUS SINUS THROMBOSIS.
A, orbital oedema and proptosis may be associated with paralysis of the IIIrd, IVth, VIth (commonly), and the first 2 branches of the Vth cranial nerves, and also with meningeal irritation. B, infection may spread to the cavernous sinus from the eyes, nose, teeth, middle ear, or the paranasal sinuses.

Don't be frightened of operating in the orbit. Because of the danger of cavernous sinus thrombosis, you must drain pus early. A negative exploration will not cause harm, and you are very unlikely to damage the globe.

EXAMINATION
Gently separate the eyelids. Examine for induration and tenderness of the lids, chemosis (subconjunctival oedema), proptosis (his globe is pushed forwards), limitation of ocular movement, and loss of visual acuity.

RADIOGRAPHS. Infection may have spread from the paranasal sinuses, so consider X-raying them (if this is possible), to see if you can find a loss of translucency on the affected side (29-8). The films may be difficult to interpret, especially in children in whom the sinuses are small.

TREATMENT. If you suspect orbital cellulitis, take blood cultures and start IV penicillin with cloxacillin or chloramphenicol. Or, use a cefalosporin immediately!

CAUTION!
(1) Oedema and erythema of the lids are common to both orbital and periorbital cellulitis.
(2) If the treatment of orbital cellulitis is delayed or incorrect, cavernous sinus thrombosis may follow.

DIFFICULTIES WITH ORBITAL SEPSIS

If the globe is displaced by an inflammatory swelling, and its movement impaired, perhaps accompanied by loss of visual acuity, suspect a subperiosteal abscess of the orbit. For example, an abscess above the eye will displace it downwards. Try aspirating the pus from the roof of the abscess with a needle. The eye may go back into place. Then incise and evacuate the abscess through a conjunctival fornix: the inferior fornix if swelling is maximal inferiorly, and the superior fornix if it is maximal superiorly. Pus will probably be coming from a paranasal sinus and you may find the track through which pus has spread. Insert a drain.

If there is an inflammatory swelling in the upper, outer part of the orbit, involving the outer 3rd of the upper lid, suspect that the lachrymal gland is infected (DACRYOADENITIS). Incise the abscess through the upper fornix of the conjunctiva, or through the eyelid.

If there is an inflammatory swelling below the medial aspect of the lower lid, suspect an abscess in the lachrymal gland (DACRYOCYSTITIS). Press it; pus may exude through the punctum. If it suppurates, incise it through the skin of the lower lid. When the infection has subsided, arrange for a dacryocystorhinostomy which will usually re-establish the flow of tears.

If the conjunctiva becomes increasingly congested with bloody tears, the globes protrude, the ocular movements become more and more impaired, accommodation paralyzed, the pupil fixed and dilated, and the cornea anaesthetic, this is a CAVERNOUS SINUS THROMBOSIS. It will probably involve both eyes. Early vigorous treatment may avoid death. Use high-dose IV penicillin with chloramphenicol or a cefalosporin, *together with diuretics* (furosemide or mannitol) *to reduce cerebral oedema. Don't forget to deal with the cause of the sepsis!*

6.7 Peritonsillar abscess (Quinsy)

Abscesses round the tonsils are quite common, and follow tonsillitis. The patient, who is usually a child, has a tense swelling above and behind one of the tonsils, displacing it downwards and forwards. Non-operative treatment is almost always successful and is much safer than draining, which is a heroic procedure and is seldom necessary, because much of the swelling is inflammatory oedema.

NON-OPERATIVE TREATMENT

Treat as an inpatient with IV penicillin, ampicillin, or chloramphenicol, as well as IV fluids and morphine or pethidine. Expect a response within 24h: the abscess will probably burst spontaneously, or the inflammation will subside sufficiently to make drainage much easier.

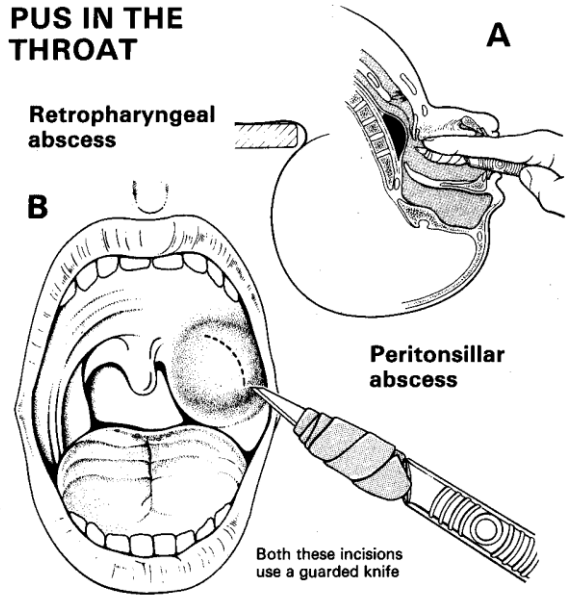


Fig. 6-6 TWO ABSCESSES IN THE THROAT.
A, the danger with a retropharyngeal abscess is that an unconscious child may inhale pus and get bronchopneumonia. Avoid this by incising it while the head is hanging over the end of a table. B, peritonsillar abscess occasionally follows tonsillitis, and may need draining. Do both these incisions with a guarded knife that cannot cut too deeply.

INCISION (GRADE 1.4)

In the unlikely event that non-operative treatment fails, sit the patient upright in a chair with the head supported, and a gag in the mouth. Get a very good headlight.

CAUTION!

(1) *Don't allow inhalation of pus*.

(2) Have suction instantly available.

Spray the pharynx with LA solution, such as 4% lidocaine. If opening the mouth wide enough is impossible, you may have to use GA and intubation with the head on the side as low as possible. Place a swab over the tongue. Pack the pharynx. *This can be very hazardous anaesthesia*. Have a tracheostomy set (29.15) and suction ready.

Use a guarded scalpel to incise the abscess over its most prominent part (6-6B). Divide only the mucosa; then use sinus forceps to find pus by Hilton's method (6.2).

If severe bleeding follows and you cannot control it, try firm compression through the mouth with a tightly rolled swab. You will then be faced with a very difficult intubation, keeping pressure on the tonsillar fossa in order to insert tight 'figure of 8' sutures around the bleeding points.

6.8 Retropharyngeal abscess

Occasionally, an abscess forms in the lymph nodes behind a child's pharynx which bulges forwards. Sometimes an abscess is the result of infection round an impacted fish bone. If the swelling is large enough, asphyxiation may result. If it bursts, aspiration pneumonia may result. The major differential diagnosis is a chronic tuberculous abscess, which may have spread from the cervical spine.

TREATMENT
If the patient is dehydrated, correct the deficit with IV fluids.

INCISION (GRADE 1.5)
ACUTE ABSCESS IN A CHILD
The great danger of a GA is that the patient will inhale pus. Ketamine is relatively safe because the cough reflex is less suppressed. Use it IV, and keep the head down.

Have a tracheostomy set (29.15) and suction ready. Put the child supine with the head over the end of the table, so that the pharynx is as nearly upside down as possible.

If the abscess is pointing, you may be able to open it with sinus forceps alone. If you can get a really good view, you may be able to aspirate it with a needle. If this is impractical, open the abscess with a guarded knife (6-6A). Put your index finger into the mouth, and slide the knife along it. Drain it by Hilton's method (6.2), as for a peritonsillar abscess.

CAUTION! Don't allow inhalation of pus: aspirate immediately you incise.

If severe bleeding follows, and you cannot control it, apply local pressure for 15mins. If that fails (rare), be prepared to tie the external carotid artery (29-7).

ACUTE ABSCESS IN AN ADULT
Anaesthetize the mucosa over the abscess with 4% lidocaine, preferably as an aerosol, and incise it with the head down and on one side, as in a child.

TUBERCULOUS RETROPHARYNGEAL ABSCESS is usually subacute and follows infection of the body of a vertebra. Only consider drainage if obstruction to the airway is a real danger. Drain the abscess through an external incision in front of the sternomastoid down to the prevertebral fascia. Displace the thyroid gland and trachea anteriorly, as in a cervical oesophagostomy (30-5).

6.9 Dental abscess

The classic presentation is with a painful, throbbing, swollen, red face (a 'fat face'), perhaps with fever, trismus and lymphadenitis; this is probably an acute dental or oral infection, most probably an alveolar abscess.

There may be:
(a) An alveolar (peri-apical) abscess: an infection which spreads to bone from a dead tooth after suppuration of the pulp of the tooth. There is severe pain and the tooth is tender to percussion, and may be slightly extruded from its socket. There is pyrexia and facial swelling develops (and trismus if the molars are involved). If drainage is delayed, the pus in the abscess discharges spontaneously through a sinus (31-9) in the gum or face, which may become chronic.
(b) A periodontal abscess at the side of a tooth, caused by spread from an infected gum. This may cause dramatic destruction of alveolar bone resulting in a loose tooth; *it is not usually tender to percussion*.
(c) A pericoronal abscess caused by infection of the gum over the crown of an unerupted and impacted tooth, usually a lower 3rd molar (an infected 'wisdom tooth').
Often, an abscess does not form, and the gum round the tooth is merely inflamed. *Extraction of the tooth does not promote drainage and may spread the infection.*

Pus from all 3 of these spaces, especially the first, can track in towards the cheek, the tongue, or the palate, or downwards into the neck. Pus can discharge inside or outside the mouth. It can collect:
(1) On any of the surfaces of the gum ('gumboils').
(2) In the buccal sulcus of either jaw on the oral or deeper side of the attachment of the buccinator muscle (common).
(3) On the surface of the face superficial to the buccinator attachment.
(4) On the palate (less common).
(5) In the submasseteric space between the masseter and the ascending ramus of the mandible.
(6) In the pterygomandibular space between the medial pterygoid and the ascending ramus of the mandible.
(7) In the sublingual space above or below the mylohyoid muscle.
(8) In the submandibular space superficial to the mylohyoid.
(9) In the submental space in the midline under the jaw.
(10) Anywhere down the side of the neck.
Don't be daunted by the complexity of this anatomy. Some of these spaces communicate with one another and more than one space may be involved.

Infection can spread in some particularly dangerous directions:
(1) From the upper jaw (or upper lip or nose) to cause cavernous sinus thrombosis, perhaps fatal (6.6).
(2) From the lateral pharyngeal space up towards the base of the skull, down to the glottis or into the mediastinum. Infection of this space is one of the most dangerous conditions in dentistry. There is difficulty swallowing and speaking.
(3) From the lower jaw, via the sublingual and submandibular spaces, to the tissues of the neck, where it may cause oedema of the glottis, respiratory obstruction and death. This is Ludwig's angina (6.11).

HISTORY AND EXAMINATION
A patient of any age >5yrs who has a swollen face, looking ill and distressed. He has usually had toothache in the past, and now he tells you that he has had pain for 1wk. He has fever, trismus, and a unilateral, tender, shiny, warm, indurated swelling. Looking at him will tell you which side of the face and which jaw is involved. Feel for warmth with the back of your index finger and test for fluctuation.
A tooth with large holes in it probably has an apical abscess under it. It may be firm, but is usually loose.

If there are either obvious periodontal disease or several loose teeth, suspect a periodontal abscess.

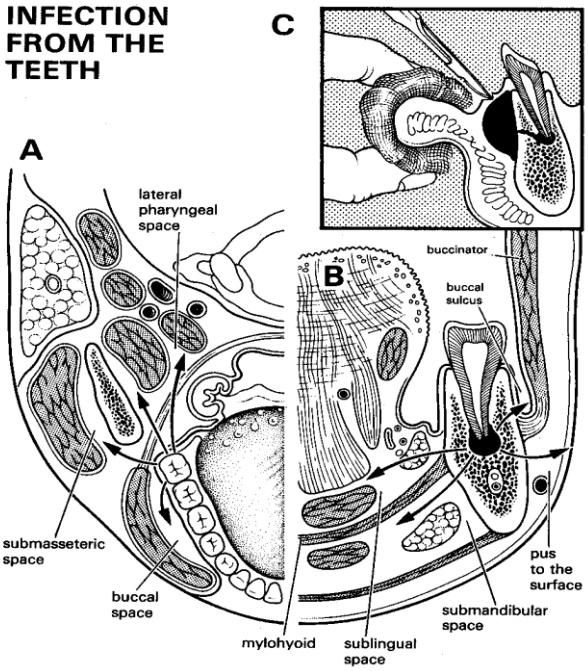


Fig. 6-7 THE DIRECTIONS IN WHICH PUS CAN SPREAD.
A,B, views of the same structures at 90º to one another. The attachments of the mylohyoid and buccinator muscles determine whether pus, orginating in the lower jaw, points inside or outside the mouth. A, pus from the lower third molar spreading into the buccal space, the submasseteric space, and the lateral pharyngeal space. B, attachments of the mylohyoid and buccinator muscles. The attachments of these muscles determine whether pus spreads into the sublingual space, the submandibular space, the buccal sulcus, or on to the surface of the face. C, incision of an abscess in the buccal sulcus. *Partly after Dudley HAF (ed) Hamilton Bailey's Emergency Surgery, Wright 10th ed 1977 Fig. 151 with kind permission.*

BEWARE OF CAVERNOUS SINUS THROMBOSIS AND LUDWIG'S ANGINA

If you are in doubt as to which of the teeth is the site of infection, tap them with some metal object or press them with your gloved index finger. A tooth which is much more painful than the others is probably the source of an alveolar infection. It may also be slightly raised in its socket. A tooth with a periodontal abscess is usually not tender to percussion, but often loose.

N.B. It is quite difficult sometimes to localize the affected tooth; be gentle and patient to be certain which tooth is the offending one. It is a tragedy to remove the wrong tooth!

RADIOGRAPHS. If possible, X-ray the offending tooth. You may see:
(1) A radiolucent area at its apex when an apical abscess has been present for 2-3wks.
(2) Caries between two adjacent teeth which may not be visible from the mouth.
(3) The impacted tooth which is responsible for a pericoronal abscess.
(4) Some other source for the infection, such as an infected cyst, or a fracture.

DIFFERENTIAL DIAGNOSIS includes acute inflammation of the salivary glands (6.10), mumps, Burkitt's lymphoma (17.6), lymph node swellings and glandular fever, as well as snake bite, and trigeminal neuralgia.

TREATMENT. Make sure fluid intake is adequate because drinking may be difficult.

CAUTION! Don't apply poultices or any kind of local heat to the face: that may spread the infection. If an abscess is pointing inside the mouth, hot saline mouth washes may ease the pain.
Antibiotics are often unnecessary, because many dental infections can be treated by local drainage only. Use IV penicillin if there is surrounding cellulitis or actinomycosis (31.6). When you have drained an abscess, culture the pus and change the antibiotic if necessary.

CAUTION! Explain that a course of antibiotics is not sufficient treatment for the abscess, and that review is essential, even if the swelling improves.

ANAESTHESIA
(1) 2% or 4% lidocaine spray or a swab soaked in lidocaine solution.
(2) Inject LA solution into the outer wall of the abscess over the proposed site of the incision.
(3) Ethyl chloride local spray is suitable for an abscess which presents on the face or in the labial or buccal sulci. Isolate the infected area with gauze packs, and then spray on ethyl chloride until crusting occurs. Then open the abscess with a #11 blade.

CAUTION! Avoid GA, unless it is expert (especially if there is danger of respiratory obstruction), with intubation throat packing.

A dentist may be able to save the tooth by draining the abscess through it, and later filling its root. If you cannot refer to a dentist, remove the tooth. Many abscessed teeth are loose, and you can then easily pick them out of their sockets.

Removing the tooth to allow pus to drain through the socket may be sufficient. *Don't incise a non-fluctuant swelling*. If it is not yet fluctuant and ripe for incision, use hot saline mouth washes, as hot as can be borne without the risk of being scalded, several times a day. Treat with cloxacillin and metronidazole and wait till the cellulitis settles.

CAUTION!
(1) *Don't pull out the tooth* (31.3) *before starting treatment for periodontal cellulitis*.
(2) **If there is a tense inflammatory swelling of the upper part of the neck**, suspect Ludwig's angina and treat urgently (6.11).

PUS POINTING INSIDE THE MOUTH can point in several places:
If an abscess is pointing on the alveolus, open it into the mouth.

If it is pointing in the labial sulcus (6-7C), make a 1·5cm incision through the mucous membrane parallel to the alveolar ridge. Push a fine haemostat into it and open the jaws.

If it is pointing in the palate, make an antero-posterior incision, parallel to the nerves and vessels, remove an ellipse of tissue and let the pus flow out.

If it is pointing in the floor of the mouth, push flat forceps through the midline muscles.

If there is pus in the pterygomandibular, lateral pharyngeal, or submasseteric spaces, drain it through a vertical incision inside the mouth parallel to the ascending ramus of the mandible, taking care to avoid the parotid duct. This runs in the cheek under the middle ⅓ of a line between the tragus of the ear and the commissure of the lips, and opens in line with the first molar tooth. Push forceps to the lingual or buccal side of the ramus, wherever the pus seems to be pointing. If it is under the masseter, insert a drain deep to this muscle down to the mandible from outside the face. Insert the drain through an incision just below the inferior border of the mandible.

PUS POINTING OUTSIDE THE MOUTH
Drain it through one of the incisions below, as soon as you have started antibiotics for any cellulitis present. Removing the tooth to let the pus drain is not enough, even if it does drip from the root canal. If the abscess is fluctuant, it needs draining too.

If you are not sure whether it is ready for drainage or not, insert a wide bore needle under LA. If you aspirate pus, incise it by Hilton's method (6.2) where it points at the softest and most tender spot. To minimize scarring, make an incision below the inferior border of the mandible, where possible. Make an incision on the face in line with the creases in the skin. These may not always be over the most fluctuant part of the abscess.

INCISION FOR DENTAL ABSCESS (GRADE 1.3)
CAUTION! When you plan your incision, consult 6-8 and remember important features of the anatomy:
(1) The extension of the lower pole of the parotid gland into the side of the neck.
(2) The mandibular branches of the facial nerve. These run horizontally and cross the lower border of the mandible, just anterior to the masseter, deep to the platysma muscle in the anterior mandibular region and deep to the fascia posteriorly.
(3) The facial artery and vein. These enter the face from between the submandibular salivary gland and the lower border of the mandible; they cross the ramus of the mandible 3cm from the angle of the jaw and then run obliquely across the lower third of the face superficially on the buccinator muscle. You may have to compromise between choosing the best site for dependent drainage and an inconspicuous scar in the crease lines of the face. Here are some likely sites:

If there is a submental abscess, drain it through a small midline transverse incision under the chin.

If the abscess is under the body of the mandible, drain it through a horizontal incision 1-2cm below the lower border of the mandible, taking care to avoid the mandibular branch of the facial nerve and the facial vessels. Push sinus forceps towards the lingual side of the mandible to drain the pus there.

If the abscess points external to the buccinator, drain it through a small incision over the swelling.

DRAINS. Suture a drain into the wound for 2-5days, or leave it open with its edges separated by gauze.

For a PERIODONTAL ABSCESS, refer to a dentist for a conservative operation, or pull out the tooth (31.3). For a PERICORONAL INFECTION (infected ‘wisdom tooth’), see 31.4.
POST-OPERATIVELY, after you have incised any intraoral abscess, treat the patient with warm mouth washes to help the incision stay open as long as is necessary.

DIFFICULTIES
If the mouth cannot open to let you get at the abscess (trismus), irrigate the mouth with warm water for 15-20mins and try again.

6.10 Parotid abscess

Although a parotid abscess can occur without any obvious cause, it occurs most often in debilitated or HIV+ve patients, or after major surgery when mouth care has been neglected. The parotid is painful and is usually much swollen; the skin over it is tight and shiny. You may see pus coming from the parotid duct (inside the cheek level with the first molar tooth). Pus forms in several lobules of the gland between its septa, and does not form a single abscess. This, and the division of the facial nerve into its five branches within the parotid gland, make drainage difficult; it is however essential. *Don’t wait for fluctuation*.

In Southeast Asia, melioidosis (due to *Burkholderia pseudomallei*) may be the cause, especially in children. This needs IV ceftazidime and can be difficult to eradicate.

INCISION (GRADE 1.4)
Start incising anterior to the pinna. Keeping close to it, proceed towards the mastoid and then continue in the angle between the pinna and the neck until you reach a skin crease, then cut along this for up to 10cm. Raise a flap of skin and subcutaneous tissue, so as to expose the parotid gland. Make multiple incisions into this in line with the branches of the facial nerve. Explore each incision by Hilton's method and clean out each abscess cavity with gauze. Close the wound with continuous or interrupted sutures of 3/0 monofilament, leaving a dependent Penrose drain emerging from the inferior part of the incision.

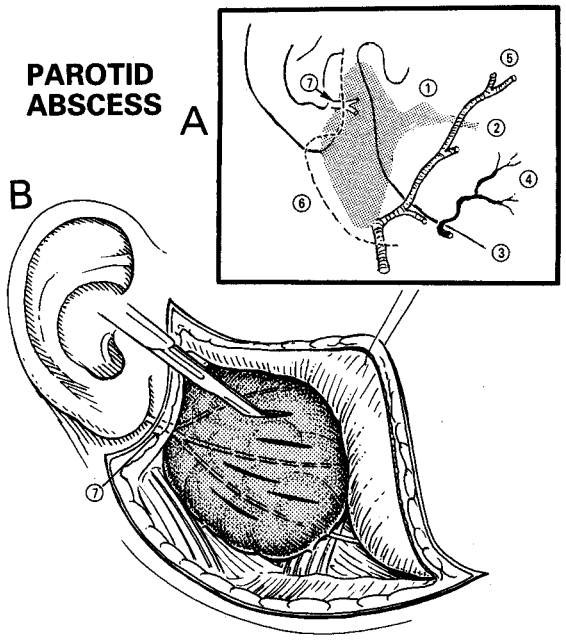


Fig. 6-8 DRAINING A PAROTID ABSCESS.
A, anatomy of the parotid gland. The facial nerve (7) enters the substance of the parotid so that, if you only incise the skin and subcutaneous tissue superficial to the gland when you reflect the flap, you will not injure it. Note that it extends well down into the neck. Incise where the pinna meets the skin of the face and neck and continue on in a skin crease. B, turn back the flap and incise radially to avoid the branches of the facial nerve (7).
(1) parotid gland. (2) parotid duct. (3) border of the mandible. (4) facial artery crossing the mandible about 3cm anterior to its angle. (5) facial vein. (6) incision. (7) VIIth cranial (facial) nerve.

DIFFERENTIAL DIAGNOSIS is mumps or parotid cysts (17.5). There is no pus at the orifice of the parotid duct, mumps is usually bilateral, and the skin over the swelling is less shiny. *Mumps parotitis does not require surgical drainage*, it resolves spontaneously. Simply aspirate HIV-related parotid cysts.

6.11 Pus in the neck: Ludwig's angina

You may see these acute suppurative infections in the neck:
(1) **Suppuration in a lymph node**, especially a deep cervical one, is common in children, and is much like suppuration in any other lymph node.
(2) **Suppuration arising from an infected tooth (Ludwig's angina)** occurs in children and adults: it is a severe bilateral brawny cellulitis of the sublingual and submandibular regions, and may extend as far as the clavicles. It usually starts as a dental abscess in the mandible, which results in fever and severe toxicity. If the infection is neglected, it may obstruct the respiration by causing oedema of the glottis, and by pushing the tongue up against the roof of the mouth. Anaerobes and spirochaetes may be responsible. Death from septicaemia is likely. Urgent intensive antibiotic treatment is mandatory, together with drainage to decompress the tissues at the floor of the mouth, even if no pus is aspirated.

If you see chronic suppuration in the neck, think of:
(1) Tuberculous lymphadenitis (17.4),
(2) Actinomycosis (31.6).
If breathing is not significantly obstructed, you may be wiser to wait for 24h for the antibiotics to act and the oedema to subside a little, before you drain the lesion.
If breathing is significantly obstructed, you may be forced to do a tracheostomy (29.15). This is difficult, because the tissues of the neck are firm and oedematous.

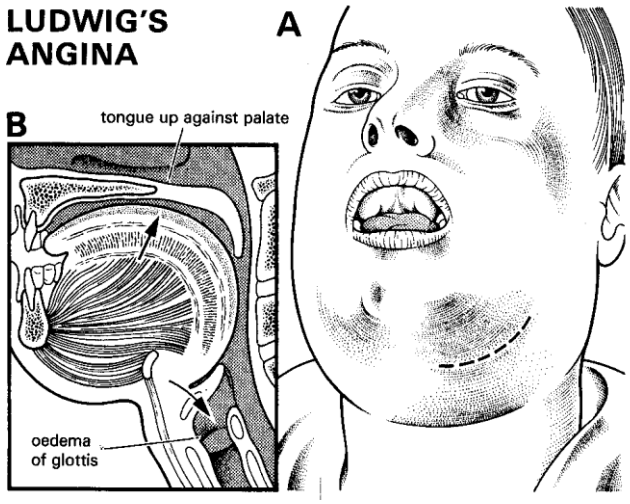


Fig. 6-9 LUDWIG'S ANGINA.
A, note the massive swelling of the chin. B, swollen tissues have compressed the tongue against the palate. The infection may spread to cause oedema of the glottis.
Partly after Dudley HAF (ed) Hamilton Bailey's Emergency Surgery, Wright 10th ed 1977 Figs.153,154 with kind permission.

ANAESTHESIA
(1) Use LA, but it will be painful and distressing, so add a little ketamine, unless the airway is almost totally obstructed.
(2) *Don't administer an inhalation anaesthetic*. The voluntary muscles are needed to maintain the airway, and you will be unable to pass a tracheal tube without great difficulty.

TREATMENT
This is an acute emergency: use high doses of penicillin, metronidazole and chloramphenicol IV.

INCISION FOR LUDWIG'S ANGINA (GRADE 1.4)
Make a generous incision below the angle of the mandible, over the point of maximum tenderness, taking care to avoid the facial artery and in the line of a skin crease if possible. The abscess will be surrounded by inflammatory oedema. Cut through the skin and deep fascia, and explore it by Hilton's method (6.2). You may need to do some careful blunt dissection to release a little pus at the centre of the abscess. *Don't be alarmed if you don't actually find pus*: it will drain spontaneously. Leave the wound open. If there is much bleeding, wash the wound with hydrogen peroxide. Later, remove the offending tooth (if this is the cause, 31.3), and when infection has settled secondarily suture the incision wound.

6.12 Thyroid abscess
(Acute bacterial thyroiditis)

Abscesses of the thyroid are not uncommon in the developing world, especially in the HIV patient. Presentation is with a wide, very painful, oedematous swelling of the neck which is maximal over the thyroid. The pus is too deep for you to be able to detect fluctuation. Inflammatory oedema may be so marked as to cause Ludwig's angina (6.11).

DIAGNOSIS. Confirm the presence of pus by needle aspiration, if necessary under ultrasound guidance.

ANAESTHESIA. Use IV ketamine or a GA with intubation. LA is not satisfactory, unless the pus is pointing, but if your anaesthetist is not expert, you may have to use it. An alternative option in this case is repeated aspiration (preferably under ultrasound guidance). *The anaesthetist must be experienced to administer a GA.*

INCISION (GRADE 1.5). Use a scalpel to make a transverse incision ≥5cm over the area of maximal swelling. Insert a haemostat and drain the pus by Hilton's method (6.2). Insert a drain and treat with an antibiotic (chloramphenicol or a cefalosporin) for 5days.

N.B. There may be perforation of the trachea, so be prepared to aspirate the airway vigorously!

6.13 Breast abscess

The importance of a breast abscess is less for a mother than for the child, who may cease to be breastfed as a result of it, and develop marasmus. So your main objective must be to see that when you have treated the abscess, mother continues to breastfeed.

Acute septic breast infections usually occur during the 2nd week of the puerperium, in a breast which is either engorged or has a cracked nipple. Antibiotics alone are only effective if you use them early, during the phase of acute cellulitis. As soon as there is a definite lump or the presence of pus found by aspiration, incise the breast.

Avoid these common mistakes:
(1) *Don't delay incision,* and *don't continue with antibiotics alone after an abscess has formed*. The mass may fail to resolve, and become so hard (an 'antibioma') that you cannot distinguish it from carcinoma.
(2) *Don't wait for fluctuation*, or for the abscess to point. If you do, she will suffer much unnecessary breast destruction.
(3) Provided that the mother does not present so late that breastfeeding is impossible, *don't take the baby away from the breast unless pus is actually draining from the nipple*. A suckling baby is much the best tool to keep the breast from being engorged.
(4) *Don't suppress lactation with diethylstilbestrol*; its effects are temporary anyway.
(5) *Don't forget to insert a drain*.

Subacute or chronic recurrent abscesses are unrelated to lactation, and are less painful. Frequently they are a presenting sign of HIV disease, or the result of nipple-piercing. They are usually close to the areola, are often associated with inversion of the nipple, and they commonly involve both breasts, either simultaneously or one after the other. A mammary fistula may be present. Actinomycosis (31.6) or filariasis may be the cause. If the lesion is localized, excise it (6-10).
Beware of the highly malignant condition MASTITIS CARCINOMATOSA, which occurs in pregnancy and mimics breast infection (24.4). The breast is inflamed and hard.

ANAESTHESIA. Use GA or ketamine. You should use LA, which is not very satisfactory, only for very superficial small abscesses. Be sure to add premedication with pethidine.

ABSCESSES IN LACTATING BREASTS
INDICATIONS FOR INCISION
(1) An area of tense induration. You will feel this most easily when the breast is empty.
(2) Pain which is severe enough to prevent sleep.
Use the tip of your finger to feel for the point of maximum tenderness. Run your finger firmly across the oedematous swelling: you may feel that its centre is slightly softer than its edges. If you are in doubt, aspirate it with a needle (6.1).

INCISION (GRADE 1.4). If an abscess points at the areola, or near it, make a circumferential skin incision at its margin. Elsewhere in the breast, a circumferential incision is preferable to a radial one, which leaves an uglier scar. In order to get a finger to break down loculi, the incision will have to be at least 2cm wide.
CAUTION! Don't wait for fluctuation.

If you are still in doubt, try to get an ultrasound scan.
Cut through the skin and subcutaneous tissue. Push a long haemostat into the abscess, and open its jaws. Pus will ooze out. Feel every part of the breast against the haemostat, and try to enter all its loculi. Remove the haemostat, and use your gloved finger to break down any septa between the loculi. If it is in the subcutaneous tissue, feel for a deeper extension.

Insert a soft drain, suture it in place, and apply a dry dressing. Wash the cavity bd. You may pack a cavity initially if there is significant bleeding, but remove it after 24h.

If there is a large abscess in a lower quadrant, make a single incision in the lower part of the breast. *There is no need to make a main incision, and another counter incision inferiorly to provide free drainage*.

If you cannot find any pus, the lesion may be an anaplastic carcinoma or the highly aggressive inflammatory carcinoma of the young lactating woman; so send a biopsy for examination.

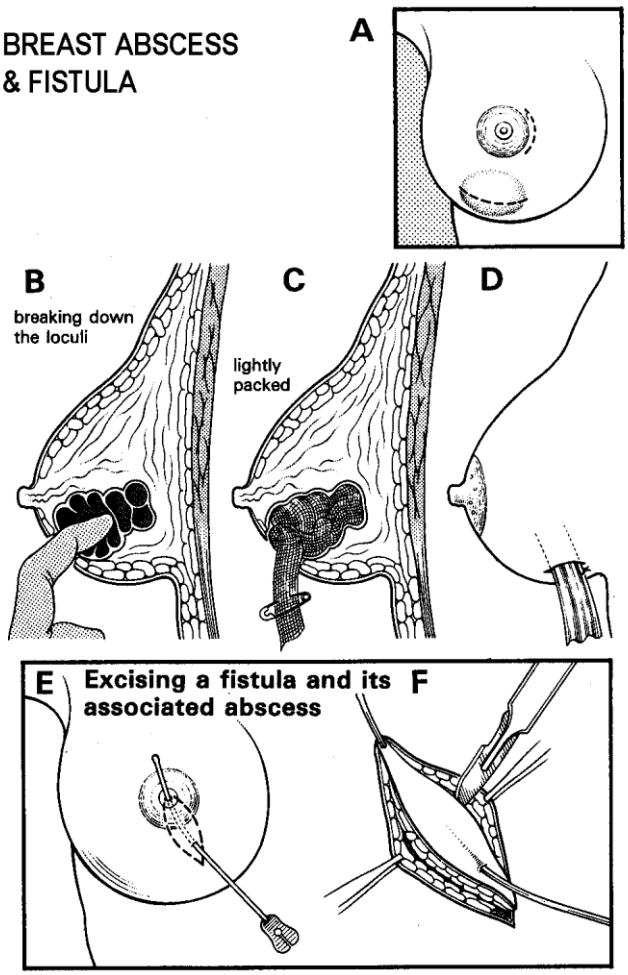


Fig. 6-10 BREAST ABSCESS AND FISTULA.
A, if an abscess points at the areola, or near it, make a circumferential skin incision at its margin. Elsewhere in the breast, a circumferential incision is preferable to a radial one, which leaves an uglier scar. B, insert your finger and break down all loculi. C, loosely pack the cavity. D, insert a dependent drain if the cavity extends below the incision. E,F, excise both ends of a mammary duct fistula, including 2cm of skin distal to the distal opening.
After Hughes LE in Rob C and Smith R, Atlas of General Surgery, Butterworth, 2nd ed 1981 p.121 Fig.25,26 with kind permission.

If milk flows from the wound, advise that it will stop, provided breastfeeding is re-established.

CAUTION!

(1) If there is no fever, or throbbing pain, consider the possibility of a carcinoma.
(2) Do a careful follow-up. Another abscess may form.

BREASTFEEDING must not stop! Let the baby continue to suck from the normal breast and, as soon as possible, from the infected breast. But *don't let him suck from an infected breast if:*
(1) Its nipple is cracked.
(2) Pus comes from it.
If so, express the milk, by hand or with a breast pump. Discard it if it is obviously mixed with pus, otherwise pasteurize it. As soon as the baby can fix onto the nipple, encourage him to suck from it.

If presentation is late, when breastfeeding has become impossible, incise and drain the breast, and use an antibiotic to hasten the resolution of inflammatory oedema. Start expressing the breast as soon as possible, and *follow up until breastfeeding has been re-established*.

SUBACUTE AND CHRONIC ABSCESSES
Be sure to take a biopsy for tuberculosis and cancer, and examine pus for acid, alcohol-fast bacilli.

If there is a small opening discharging pus, at or near the areolar margin, or recurrent abscesses continue to reappear at the same site, near the areola, this is a MAMMARY FISTULA (or sinus). Examine the patient during a quiescent phase. See if you can pass a probe from the site of the abscess, through to the nipple. If you can, a fistula is present and you may be able to excise the whole lesion (6-10E,F). Make the incision round the fistulous track, and continue it 2cm distal to the fistula. There is no need to remove more than ½cm of skin on either side of the track. Deepen the incision to expose the underlying tissue, and excise the fistula. Be sure to excise the central part of the duct, because if you leave it behind, the lesion is sure to recur.

If there is necrotizing fasciitis of the breast, there is widespread tissue destruction. This is a sign of advanced HIV disease. The options are extensive debridement or mastectomy (24.5): blood loss may be extensive, so be prepared to transfuse!

RE-ESTABLISH BREASTFEEDING QUICKLY IN AN INFECTED BREAST

6.14 Axillary abscess

Suppuration in the axilla can take several forms:
(1) Pus can form superficially in the apocrine glands.
(2) It can form more deeply in the lymph nodes under the *pectoralis major*. Open a deep abscess promptly, because pus can track along the nerve trunks into the neck.
(3) It can arise in the scent glands (hidradenitis suppurativa) as a result of the use of deodorant 'roll-ons' which block the excretory ducts.

TREATMENT
Abduct the arm.
If the abscess is superficial, incise over it.

DRAINAGE (GRADE 1.4)
If the abscess is deep, make a 3-5cm incision just behind the fold of the *pectoralis major*, so as to avoid the axillary vessels. Push a haemostat upwards into the swelling, open its handles parallel to important structures, and open the abscess. Insert a drain, and suture it in place.

If the whole axilla is a bag of pus, incise low in the axilla.

If there is a large subacute or chronic abscess, consider the possibility of tuberculosis, especially if the surrounding tissues are indurated, sinuses are present, and the breast is swollen from lymphoedema, perhaps with the sign of *peau d'orange*.

If there are multiple recurrent small abscesses in the skin, the cause may be:
(1) tuberculosis, so take a biopsy. Otherwise start a therapeutic trial with chemotherapy for tuberculosis;
(2) fungi or actinomycosis (31.6);
(3) hidradenitis suppurativa (34.9): avoid incision and drainage, and treat with cloxacillin and metronidazole or rifampicin. Regular swabbing with surgical spirit after showering helps to open up the excretory ducts.

N.B. Chronic hidradenitis results in sinuses, keloid formation and contracture, and may need wide excision leaving a 2cm adjacent and *deep margin of soft unaffected tissue.*

6.15 Retroperitoneal abscess

Retroperitoneal abscess is a common feature of HIV disease; it may become very large in size because it often remains undetected for a long time. It does not necessarily arise from the cortex of the kidney as does the perinephric abscess. The latter is mainly staphylococcal, but the former may have a wide variety of organisms, including *salmonella* and anaerobes.

The patient, who may be any age, presents with fever and a tender swollen area in the loin or subhepatic area. **If the abscess is small and related to the upper pole of the kidney**, there may be no localizing signs.

ULTRASOUND (38.2h) is the best way of diagnosing and defining a retroperitoneal collection, and can distinguish this from a subphrenic collection. You can also gain information on the kidney in this way, and use ultrasound to localize where to insert a needle for diagnosis and a therapeutic drain.

RADIOGRAPHS. A plain radiograph may show obliteration of the *psoas* shadow, and scoliosis with a concavity towards the abscess. Look also for disease of the spine, especially narrowing of intervertebral discs and erosion of the bodies of the vertebrae nearby, especially anteriorly (osteomyelitis, an important differential diagnosis). *An IVU is not usually necessary*; it may show a normally functioning kidney which may be displaced, especially medially or posteriorly, or a hydro- or pyo-nephrosis, but ultrasound is the imaging of choice.

DIFFERENTIAL DIAGNOSIS
(1) Pyomyositis of the abdominal wall or paraspinal muscles.
(2) Pyonephrosis.
(3) Subphrenic abscess.
(4) Osteomyelitis of the spine, with spread to the paraspinal tissues.

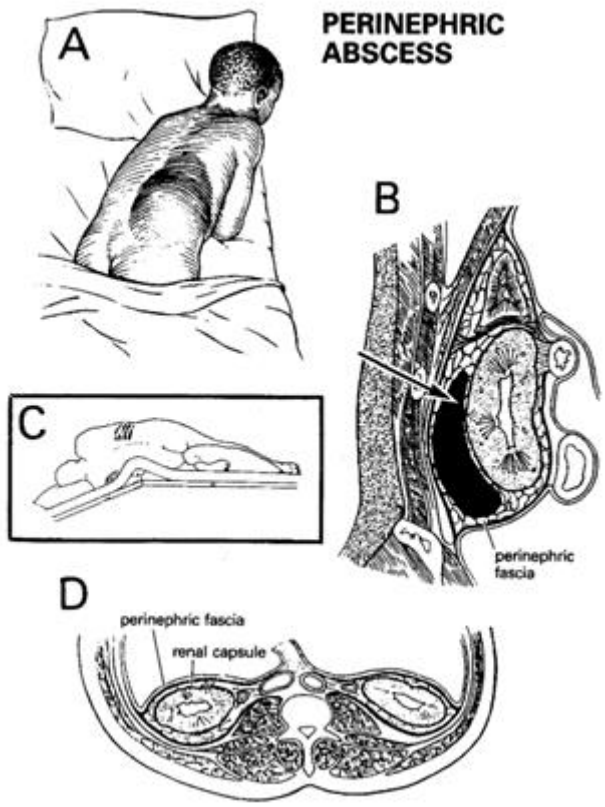


Fig. 6-11 PERINEPHRIC ABSCESS.
A, unusually large perinephric abscess. B, approach a perinephric abscess through the bed of the 12th rib. C, patient in the left lateral position. D, true renal capsule is closely applied to the surface of the kidney. Outside this, the perinephric fat is surrounded by the perinephric (Gerota's) fascia. *After Flanigan RC in Rob's Operative Surgery. 1981 Figs.1b,3,12a with kind permission.*

(5) Retroperitoneal sarcoma: this is rare, but if you incise into the tumour, you will lose the chance to excise it properly.

TREATMENT. The pus must be drained. You may not know for certain if it is perinephric, subphrenic (especially in the posterior or subhepatic spaces, 10-5B), or has spread from osteitis of the spine. Treat with chloramphenicol or a cefalosporin. If you can, insert a tube drain under LA with ultrasound guidance.

POSITION. Lateral, as for a nephrostomy (27.14).

INCISION FOR RETROPERITONEAL ABSCESS (GRADE 3.2)
The retroperitoneal abscess of HIV may become so superficial that dissection is not necessary. Otherwise, make a 15cm lumbotomy incision slightly below the 12th rib just lateral to the *sacrospinalis* muscle (about the midpoint of the rib) extending down obliquely towards the posterior iliac spine. You can extend this laterally just above the line of the posterior iliac spine if necessary. *Take care to avoid the iliohypogastric nerve* at the lower end of the incision. Retract *latissimus dorsi, external* and *internal oblique* and *transversus abdominis* muscle origins, and cut through the deep fascia onto retroperitoneal fat behind the kidney.

If the pus is in the muscles (pyomyositis), you will discover this before you reach the rib (unless it is in the *psoas* or *quadratus lumborum*). If it is spreading from the spine or is subphrenic, you will also find it.

N.B. The lumbotomy incision is easier than the 12th rib bed incision, but gives poorer access to the kidney itself; it is, however, perfectly satisfactory for drainage of an abscess.

Drain the pus by Hilton's method (6.2). Insert a wide bore tube or corrugated drain and close the wound in layers.

6.16 Iliac abscess

When you see a child or young adult with a painful flexed hip, and c.7-day history of fever, anorexia, pain, and swelling in the inguinal area, think of iliac adenitis. The infection may have reached the iliac nodes from the leg, the perineal area (including the genitalia), or the buttocks. The abscess lies near the *psoas* muscle; this goes into spasm and sharply flexes the hip, so that extension beyond 90º and walking is impossible. There is a tense, tender, hard mass in the iliac fossa, which is lower, and closer to the anterior iliac spine, than an appendix mass. Fluctuation is rare, and only occasionally will you find the site of the primary infection.

It is useful to distinguish 'periadenitis' without suppuration (common), which resolves on antibiotics and does not need drainage, from an iliac abscess (less common), which needs drainage and which can follow periadenitis, or pyomyositis of the *iliopsoas*, or be an extension from osteomyelitis of the spine. An appendix abscess is quite different, and is inside the peritoneum, whereas all these other conditions are retro-peritoneal. Consider brucellosis if you don't find any pus.

Iliac abscess is also known as iliac adenitis, deep inguinal adenitis, extraperitoneal iliac abscess, or suppurating deep iliac nodes. It has several important differential diagnoses, and is often misdiagnosed.

DIFFERENTIAL DIAGNOSIS is that of the 'sick child with the painful flexed hip'. It is more difficult if the right hip is flexed, because the diagnosis on this side includes appendicitis.

Suggesting inguinal (groin) **abscess:** a septic lesion in the skin or groin lymph nodes which may be minimal or extensive, and may complicate TB or malignancy. You should look for the origin of the sepsis on the foot, leg, perineum, or abdominal wall. If there is pus, drain it following recommendations as for an axillary abscess (6.14).

Suggesting pyomyositis of the *iliopsoas*: the same signs as iliac adenitis. The differential diagnosis may be impossible, and is not important because the treatment is the same.

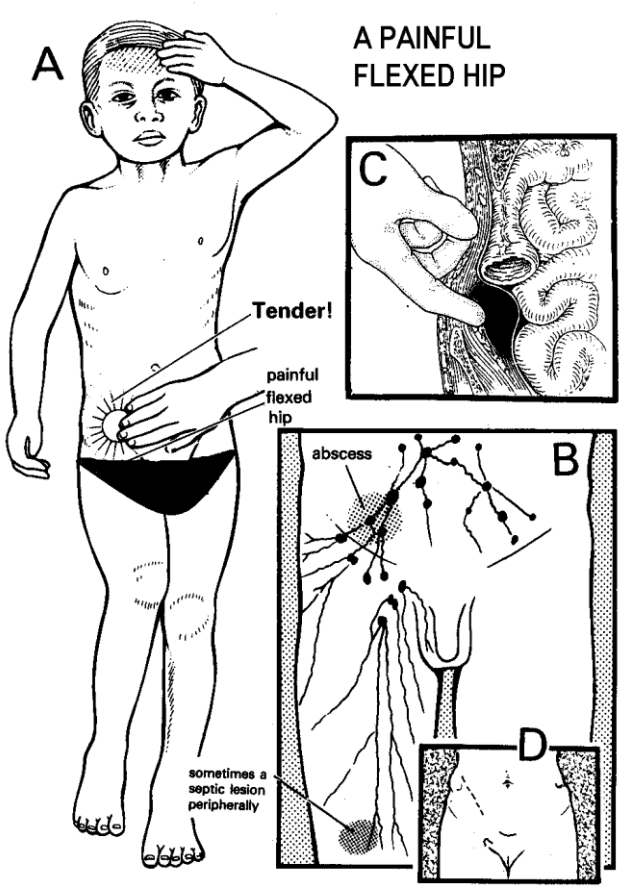


Fig. 6-12 A PAINFUL FLEXED HIP in an ill patient has a variety of differential diagnoses. A, typically the hip is more flexed than is shown here. B, iliac abscess forms in the iliac nodes. C, exploring extraperitoneally for iliac suppuration. D, incision for an iliac abscess. *C,D, after Dudley HAF (ed), Hamilton Bailey's Emergency Surgery, Wright 10th ed 1977 p.287 Fig 26.1 with kind permission.*

Suggesting an appendix abscess: a different anatomical site: intraperitoneally in the right iliac fossa, with nausea and vomiting, less spasm, and only mild flexion of the hip (14.1).

Suggesting septic arthritis of the hip: severe joint spasm, acute pain on percussing the greater trochanter, no palpable mass, no movement of the hip owing to severe pain, and a radiograph showing a widened joint space. This is equivalent to osteomyelitis because the epiphyseal plate is inside the capsule of the hip joint (7.18).

Suggesting tuberculosis of the hip: a chronic history and radiograph signs of tuberculosis (5.7). Other possibilities include Perthes' disease (32.14), a slipped epiphysis, and a fracture.

Suggesting a tuberculous *psoas* abscess arising from the spine: a chronic history, radiographic changes in the spine. A *psoas* abscess does not usually need drainage, unless it is very large and causing pain. It will resolve slowly on therapy for tuberculosis; incising it can lead to secondary infection.

Suggesting acute and usually staphylococcal osteomyelitis of the spine (uncommon): more pain, spasm of the *sacrospinalis*, radiographic signs in the spine. Drain the lesion as for osteomyelitis (7.2).

If the diagnosis is difficult, and you suspect an abscess, you can: (1) Make an examination under GA, with the abdominal muscles relaxed. Feel the exact site of the mass and its consistency and boundaries, and feel for fluctuation. (2) Aspirate the mass with a large-bore needle, medial to the anterior superior iliac spine.

NON-OPERATIVE TREATMENT. *Deep inguinal (iliac) adenitis with periadenitis and without pus formation does not require drainage*. The hip is flexed as when an abscess is present. You can feel deep tender glands above the inguinal ligament. Treat with penicillin or chloramphenicol. If infection is slow to resolve, use skin traction ($^1/_7$th of the body weight) to avoid contracture and raise the foot of the bed.

DRAINAGE (GRADE 2.4). If you have aspirated pus with a needle, you can safely open up the deeper layers. The abscess will have pushed the peritoneal lining of the right iliac fossa medially and superiorly. Make an incision 5-10cm or more over the swelling about 2cm above the inguinal ligament, starting just medial to the antero-superior iliac spine (6-12D). Take a long haemostat and push this through the muscle over the abscess until you find pus. Then, using your fingers, enlarge the opening. Take a specimen, drain the lesion, and continue antibiotics. If the leg remains in spasm, apply traction as above.

CAUTION! Draining an iliac abscess is potentially dangerous: you may injure the caecum or the iliac vessels. So follow the method above and aspirate first. Ultrasound guidance (38.2) will help.

6.17 Anorectal abscess

An anorectal abscess usually originates in an anal gland, and may communicate through a tiny opening with the anal canal, at the pectinate line. A connection between the skin and the anus (a fistula) is the reason why about half of these abscesses recur, or discharge persistently. Abscesses (with no opening to the skin), sinuses (with an opening to the skin, but not to the anus), and fistulae (with openings to both) are thus part of the same disease process (26.3). Most abscesses settle by discharging spontaneously, or being drained, but a serious life-threatening infection can sometimes spread in the soft tissues, or deeply into the pelvis.

Presentation is usually acute because the pain is intense: severe throbbing pain keeps the patient awake at night. On examination, you find a tense tender swelling near the anus. Sometimes, there may be little to see and no fluctuation to feel, except mild tenderness at the anal margin, or the whole perineum may feel tense and tender. If the pain suddenly resolves, the abscess has probably spontaneously ruptured. But there may now be a persistently discharging sinus or fistula opening on to the skin near the anus.

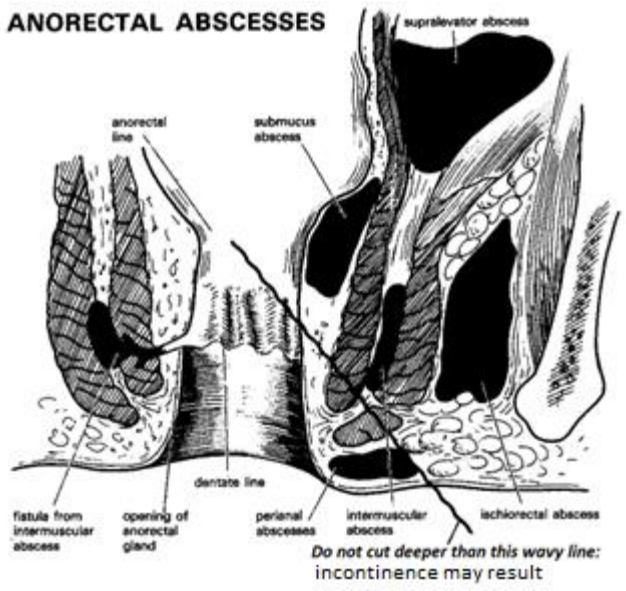


Fig. 6-13 AN ANORECTAL ABSCESS forms in the anal glands. The pus can track in any of the directions shown. When an abscess bursts into the anal canal and on to the skin a fistula may form.
After Macleod JH. A Method of Proctology, Harper &Row 1979 Fig.7.9 with permission.

As anal glands are mostly posterior, most abscesses and most fistulae are posterior. These glands extend into the sphincters, so that pus can track in various directions:
(1) downwards to cause a perianal abscess;
(2) laterally, through the sphincters, to cause an ischiorectal abscess. The ischiorectal spaces connect with one another behind the anus, so that infection on one side can spread to the other side (horseshoe abscess);
(3) rarely, medially under the mucosa of the anal canal to form a submucous abscess, or
(4) upwards between the sphincter muscles to form a high intermuscular abscess, or further above the *levator ani* muscles to form a supralevator abscess.

Here are the classical types of anorectal abscess, but you may see combinations, and the diagnosis can be difficult. Only the 1st two are common.

(a) A perianal abscess presents as a red tender swelling close to the anus. On rectal examination, there is little or no tenderness, induration, or bulging in the anal canal. There may be a fistulous track, going straight through or above the subcutaneous external sphincter, and usually through the lowest part of the internal sphincter.

(b) An ischiorectal abscess lies deeper than a perianal one, is larger and further from the anus; it forms a deep tender brawny swelling and is not fluctuant until late. The patient is likely to be toxic, febrile, and debilitated. On rectal examination you may feel a tender induration bulging into the anal canal on the same side. The infection may spread posteriorly and then to the other side as a horseshoe abscess, so that there now are signs on both sides. The presentation may then be with urinary retention.

(c) A submucous or high intermuscular abscess (rare) presents with pain in the rectum and no external swelling, unless it is complicated by an ischiorectal or perianal abscess. On rectal examination you may be able to feel a soft, diffuse, tender swelling extending upwards from the pectinate line. You will often need to administer a GA to do a rectal examination: confirm and treat the condition by draining the abscess.

(d) A pelvirectal abscess (rare) presents with fever, but no local anal or rectal signs. Later, it may extend downwards into the ischiorectal fossa. With your finger in the anus, you may be able to feel fluctuation above and lateral to the anorectal ring.

Don't delay treatment in the hope that an anorectal abscess will cure itself: always incise it. If the abscess is large, warn that it is going to take weeks to heal. De-roof it and let it granulate. *Don't try to curette it, and close it by primary suture*. A large incision will not necessarily give a better result; recurrence depends on whether or not there is a tiny communication between the abscess and the anal canal.

PERIANAL ABSCESS

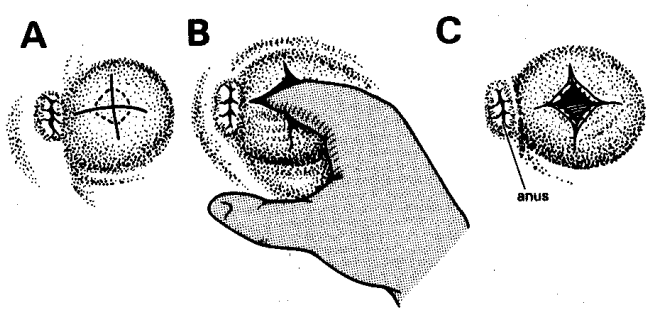


Fig. 6-14 DRAINAGE OF A PERIANAL ABSCESS.
A, cruciate incision. B, insert your finger and break down loculi. C, wound with its edges trimmed, being left to granulate.

CAUTION!
(1) **If there is an acute abscess**, *don't probe around looking for fistulae*: wait until the lesion has become chronic. *If you probe unwisely, you may create an iatrogenic extrasphincteric fistula* which will be very difficult to treat.
(2) **In the chronic phase**, look carefully for the tracks in the skin and rectum that show its presence. Unless you demonstrate the presence and course of the fistula, you cannot hope to cure it.
(3) **If an abscess lies anteriorly**, consider the possibility of a periurethral abscess in a man, or a Bartholin's abscess in a woman.
(4) **If there are multiple abscesses**, these are likely to be the result of inadequately draining fistulae.

INDICATIONS FOR INCISION. Operate immediately you can feel a tender swelling. *Don't wait for fluctuation*. If pain has deprived sleep, open the abscess.

ZBIG (50yrs) complained of painful defecation and passing pus and blood rectally. He was found to have a perianal swelling, given a course of antibiotics, and sent home for readmission later for examination under anaesthesia. He returned after 3 days with severe pain, swollen crepitant buttocks, and a black gangrenous scrotum. The urine was tested and was found to contain sugar. He was referred, but died en route.
LESSONS (1) Bacteria in anorectal abscesses come from the gut and anaerobic infections can be dangerous. (2) Never treat an anorectal or perineal abscess with antibiotics without also draining it. (3) Spreading anaerobic infections originating in the gut need metronidazole and in this case at least chloramphenicol, and early debridement of all the dead tissue. (4) Always test the urine for sugar and check for HIV. Serious infections are particularly common in diabetics. Recognize Fournier's gangrene early!

ANTIBIOTICS will not treat an abscess and are useful only if there are signs of spreading infection. If so, treat with chloramphenicol and metronidazole, and look if there are signs of necrotizing fasciitis (6.23) which needs wide debridement. Occasionally use prophylactic antibiotics if the patient has a hip prosthesis *in situ* or has had rheumatic fever. Rarely, if there is severe neutropenia due to bone marrow failure, you should use antibiotics rather than performing an incision, as in this case *there will be no pus*!

ANAESTHESIA
For a large abscess, use GA or ketamine: *make sure you put the legs up in the lithotomy position before you give the ketamine, otherwise the legs may be too stiff to elevate!*
N.B. LA is unsatisfactory, except for a small abscess.

EXAMINATION UNDER ANAESTHESIA
Use the lithotomy position. Put a finger into the anus and feel its entire wall between two fingers (26-2F). Feel if there is an indurated upward extension of the abscess under the mucosa 3cm or more above the internal sphincter. Feel the extent of the abscess, and for the point of maximum fluctuation. Insert a bivalve speculum and look for pus coming out of an internal opening near the dentate line. Press on the abscess: you may see a bead of pus escape from the internal opening. You may feel the opening as a localized tender depression in the anal canal in the place suggested by Goodsall's rule (26-6I).

DRAINAGE (GRADE 1.4)
Support the mass with your finger in the rectum. Make a cruciate incision the length of the diameter of the abscess over its most prominent or fluctuant part. This will be externally for a perianal or ischiorectal abscess, and inside the rectum above the anorectal line for a rare submucous or pelvirectal abscess. Make the incision large enough to admit one or two fingers, so that you can explore the abscess fully with your finger and break down all loculi (6.2). *Don't break down any natural barriers to the spread of infection*. If possible, send a specimen of the pus for culture. Now look again, but *don't probe*, to see if there is a fistulous opening.

If you do find a fistula, which you will only find in about 10% of cases, determine where it is in relation to the pectinate line. Make sure there is no foreign body in the rectum.
If the abscess is acute, you will not find a track. *Don't probe around*, you may make a false track!

If the abscess is chronic with a well-defined wall, and the patient is well anaesthetized, probe carefully to look for a fistula.
If there is no fistula, cut off the corners of the flaps to prevent the edges of the wound coming together and adhering. A linear incision is *hardly ever* adequate. Wrap your finger in gauze and clean the walls of the abscess cavity.
If there is a fistulous opening, pass a seton (26.3). *Don't lay open the fistula even if it is a low type*, unless you are certain of the patient's HIV-negative status.

POST-OPERATIVELY, insert a soft drain, suture it in place, and make sure the patient showers bd. Insert a pad inside the underwear. Recommend laxatives if there is a tendency to constipation.

DIFFICULTIES WITH AN ANORECTAL ABSCESS
If there is an abscess on both buttocks, use circumferential incisions 3-5cm apart on both sides and loop a drain between them to keep the space open (6-15). There is sure to be a track across the midline behind the anus. *But be sure not to cut in the midline either anteriorly or posteriorly* because healing will be very slow and you may damage the sphincter.

If there is pus draining from the anus, the abscess has either drained internally, or there is an infected HIV-related anal ulcer (26.2), or other underlying disease (*e.g.* tumour, amoebiasis, schistosomiasis, gonorrhoea, tuberculosis, inflammatory bowel disease or trauma).

If there is an internal opening which communicates with the ischiorectal fossa above the anorectal ring (rare), *don't cut externally*, or incontinence will result! Drain the abscess internally. You may then possibly avoid the complications of a fistula.

If the abscess extends submucosally (rare: 6-13), make an opening internally. *Don't lay it open* as it will probably bleed copiously, and if there is untreated HIV disease, it may never heal.

If there is a supralevator abscess (very rare), explore the abdomen and drain the abscess, preferably extraperitoneally.

If there are signs of spreading infection, such as gross inflammatory swelling, areas of necrosis, or crepitation, this is necrotizing fasciitis. Start urgent IV metronidazole plus chloramphenicol or a cefalosporin and perform a wide debridement.

If there is a recurrent abscess (common), there is almost certainly an underlying fistula. The opening may be very small, and you may have overlooked it when you drained the first abscess. Check the HIV status and glucose. Drain the abscess and attend to the fistula when the infection has settled.

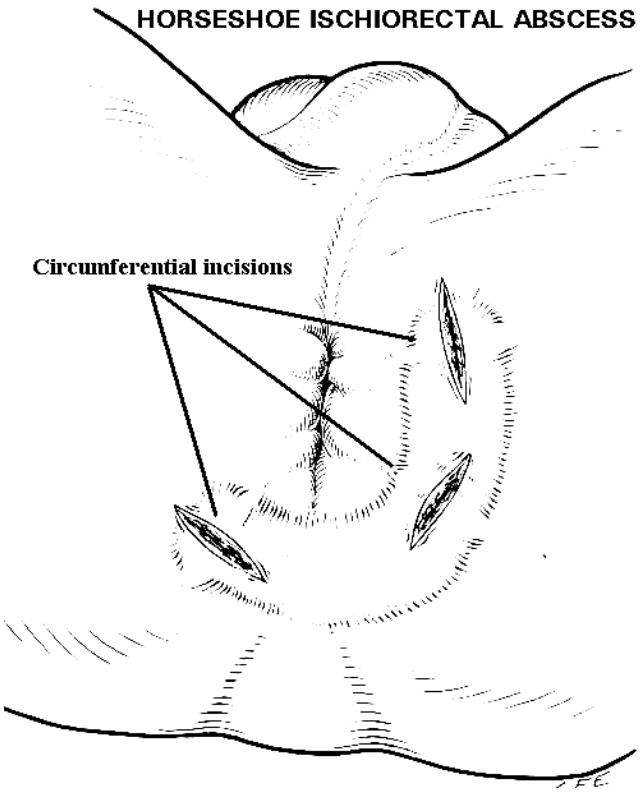


Fig. 6-15 DRAINAGE OF A HORSESHOE ISCHIORECTAL ABSCESS.
Incisions circumferential to the anal canal 3-5cm on both sides without crossing the midline: a loop drain between them keeps the space open.
Adapted from Dudley HAF (ed) Hamilton Bailey's Emergency Surgery, Wright 10th ed. 1977 p.384 Fig 39.5.

If a fistula develops later, pass a seton (26.3).

If there is gross faecal incontinence, fashion a defunctioning colostomy to allow the sepsis to settle, and later re-examine the remaining fistula(e).

6.18 Periurethral abscess

A periurethral abscess presents as a tender inflamed area in the perineum, or under the penis. The abscess commonly arises in the bulbar urethra, probably in Cowper's para-urethral glands, and is usually caused by *gonococci* to begin with, but these are soon replaced by secondary invaders. The danger is that urine may leak from the abscess cavity, extravasate widely, and cause extensive cellulitis or a fistula (27.11). The urine is infected, so this kind of cellulitis is more dangerous than that following traumatic rupture of the urethra. There may or may not be retention of urine due to an inflamed stricture, which will prevent you passing a catheter, so you may have to drain the bladder with a suprapubic cystotomy (27.8).

DIFFERENTIAL DIAGNOSES
(1) A perianal abscess.
(2) A scrotal abscess is in a different place and is not associated with urinary symptoms.
(3) Localized penile extravasation of urine.

ANTIBIOTICS. Use ampicillin or chloramphenicol, until you have the results of culture of the urine and pus, if this is possible.

DRAINAGE (GRADE 1.4). Try passing a soft rubber urethral catheter *(even if there is no urinary retention)*.

If catheterization is successful, drain the abscess by a midline perineal incision; be sure to open it widely, but *take care not to damage the urethra*.

If catheterization fails, as it probably will, and you cannot identify the urethra, perform a suprapubic cystostomy (27.8); then drain the abscess.

If the stricture is short and the sepsis minimal, gently pass a bougie until the stricture is reached. Open the abscess as before and feel for the bougie; display the urethra and perform an external urethrotomy by opening it longitudinally from the bougie distally across the stricture in order to pass the bougie into the bladder. *Don't cut into the roof of the urethra!* Replace the bougie by a urethral catheter. *(It will then be much easier to manage the stricture than if you leave it and try to dilate it later.)* ***Don't extend your incision in the bulbar urethra*** as massive haemorrhage may result, which will be very difficult to control. Insert a soft rubber drain and encourage showering bd. Manage the stricture by gently attempting to pass a bougie after 2-3wks.

DIFFICULTIES WITH A PERIURETHRAL ABSCESS
If the urine extravasates, treat with antibiotics and divert the urine (27.12).

If the abscess recurs, consider diabetes, HIV, tuberculosis or carcinoma of the urethra.

If a fistula develops, divert the urine (27.11).

6.19 Prostatic abscess

Gonococci or coliforms can infect the prostate. To begin with, they cause a prostatitis, and later a frank abscess. The patient presents with urgency, frequency, and dysuria, or with urinary retention. There is fever, rigors, and severe rectal or perineal pain, sometimes with tenesmus. The prostate is enlarged, usually more so on one side than the other, and is exquisitely tender. Untreated, the abscess may burst into:
(1) the urethra,
(2) the perirectal tissues, where it can present as an ischiorectal abscess,
(3) the perineum,
(4) the rectum, forming a rectourethral fistula.

DIFFERENTIAL DIAGNOSIS
Extreme prostatic tenderness should make the diagnosis clear. *Don't confuse a prostatic abscess with*:

(1) An ischiorectal abscess: the swelling is to one side of the midline.
(2) An abscess in a seminal vesicle: rectally, the site of maximum swelling and tenderness will be higher and more to one side.

SPECIAL TESTS. Test the urine for sugar, and culture it. Check the HIV status.

ANTIBIOTICS. Treat with ampicillin or chloramphenicol, until you know the results of culture.

MANAGEMENT
If the prostate is not fluctuant, see what antibiotics alone will do in 48h. Try to find an expert urologist, who can drain the abscess into the urethra with a resectoscope. Otherwise drain the abscess yourself, as follows. Fortunately, this is very rarely necessary.

DRAINAGE (GRADE 2.4)
The ideal, if antibiotics fail to cause a marked improvement in 48h, or the abscess is fluctuant, is endoscopic drainage by a urologist using a resectoscope. If this is not possible, use an exaggerated lithotomy position and administer a GA. Start by passing a rubber Jacques catheter. If this passes easily, leave it in place. If you cannot pass it, perform a suprapubic cystotomy.

To drain the abscess, pass a metal sound, and cut down on to this through a 5cm midline incision immediately in front of the anus.

Remove the sound and control bleeding. Put your finger through the incision into the prostatic urethra, and then through its posterior wall into the abscess cavity. If this contains several loculi, break down the septa between them.

Pack the wound loosely with a dry dressing and leave it open, or suture the skin edges loosely over it. Remove the catheter about the 7th day.

6.20 Abscess in the seminal vesicles

This is rare; the symptoms are the same as with an abscess of the prostate, but the warmth, the swelling and the tenderness, instead of being over the prostate, are higher and more to the side, over one, or occasionally both, of the seminal vesicles. There may also be pain suprapubically, in the back, or down the inner side of the thighs.

DRAINAGE (GRADE 2.4)
Use an exaggerated lithotomy position, and make an oblique lateral perineal incision. Dissect bluntly until you feel the swollen vesicle. Push a haemostat into it, drain it, and close the wound lightly round a drain.

6.21 Penoscrotal abscess

(a) Penile infection (balanitis)

Infection of opposing surfaces of the prepuce and glans may be the result of inadequate hygiene, incomplete retraction of the foreskin, underlying ulceration with chancroid, syphilis or carcinoma, or unusual sexual practice.

SPECIAL TESTS
Test for diabetes and HIV. Biopsy a suspicious ulcer.

TREATMENT
If proper cleaning with chlorhexidine (or similar) fails, either because of the severity of the infection or because there is phimosis or underlying ulceration, use an antibiotic such as cloxacillin, and arrange circumcision when the inflammation has settled.

If there is phimosis and urinary retention, perform a circumcision (27.29). *A dorsal slit is not really adequate.*

If gangrenous patches develop, this is phagedaena and the patient becomes septicaemic. Use IV chloramphenicol and cloxacillin, and under ketamine, debride necrotic tissues widely. This will involve removing the foreskin, and may mean removing skin from the penile shaft also. If sepsis is extensive, insert a urethral catheter in order to show you where the urethra is and avoid damaging it during debridement.

(b) Scrotal abscess

If pain and swelling develop with explosive rapidity in the scrotum and the base of the penis, **with hypotension**, this is acute necrotizing infection known as

(c) Fournier's gangrene (6.23).

SPECIAL TESTS. Test for diabetes and HIV.

This occurs with HIV disease or in diabetics spontaneously but may follow surgery to the scrotum or penis, or extravasation of urine, especially if infected. It is caused by a synergistic combination of organisms, including anaerobes. (*Clostridium welchii* is sometimes responsible, and may form gas in the scrotum.) It spreads rapidly, because the necrosis affects all the fascial layers, *dartos* and *tunica vaginalis* together, and eats away much of the scrotum, penis or abdominal wall, and ends in Gram-ve septicaemia and death.

TREATMENT; DEBRIDEMENT (GRADE 2.2)
Treat with IV gentamicin, or a cefalosporin, and metronidazole. Resuscitate with IV Ringer's lactate or saline. Apply wet dressings and arrange debridement immediately. (Hydrogen peroxide is effective, but quite painful.) The sloughs will probably separate rapidly to expose the testes.

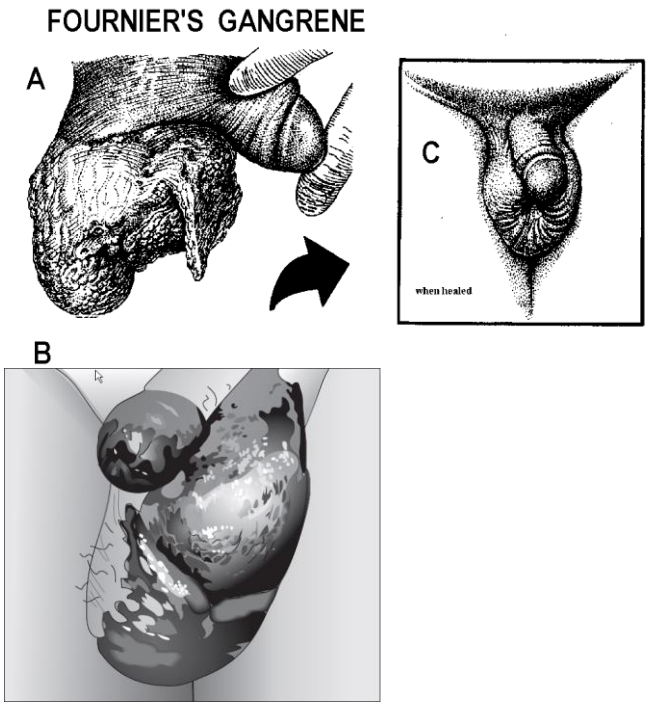


Fig. 6-16 FOURNIER'S GANGRENE OF THE SCROTUM.
A, it is usually much worse than this. B, when it affects the penile shaft as well. C, healed without skin grafting, after radical debridement. *After Bowesman C. Surgery and Clinical Pathology in the Tropics, Livingstone 1960 with kind permission.*

Excise all dead tissue as soon as possible, sacrificing some living tissue if necessary. The testes are spared, having their own blood supply, and it may be necessary to expose both testes and leave them dangling free. You may have to extend the debridement to the shaft of the penis and abdominal wall. *Unless you remove all dead tissue in severe cases, sepsis cannot be controlled and death is inevitable.* Examine the wound bd, and if you see further necrotic tissue, do another debridement.

When the infection has settled, attempt secondary suture of the remaining elastic scrotal skin over the testes, or if this is not possible, just allow the wound to granulate. If there is insufficient scrotal skin left, you may have to bury the testes in the medial part of the thighs.

(d) Epididymo-orchitis and testicular abscess

If chronic infection of the epididymis persists, suppuration may result and spread to the testis itself. Alternatively septic micro-emboli travel directly to the testis resulting in septic necrosis. This occurs with HIV disease. The infection is inside the scrotum rather than in the scrotal wall; the scrotal skin is normal until the sepsis points through. There is deep pain, which may be felt in the abdomen.

TREATMENT
Antibiotics (usually doxycycline) may already have been given. Explore the scrotum through a transverse incision; if the testis and/or epididymis are severely infected, perform an orchidectomy (27.26) and close the wound round a drain.

6.22 Cellulitis

Infection in the fatty tissues under the skin is extremely common, but is dangerous because it can spread easily, and there is no demarcation as with pus in an abscess. Cellulitis can occur anywhere and is especially dangerous in the face and orbit (6.6) or neck (Ludwig's angina, 6.11) but usually arises in a limb, commonly the lower leg, which is swollen, warm and tender; later it becomes red or shiny, frankly oedematous and increasingly painful. Erysipelas is similar, affecting the subcuticular lymphatics, resulting in pustular eruptions on the skin. There is a high fever, which can develop quickly into bacteraemia with rigors.

The cause is usually a small abrasion, puncture wound (especially by a thorn, metal-piercing, or conventional surgery), blister, ulcer, burn, or infected bursa but in diabetics and HIV disease, it can arise spontaneously. Cellulitis used to be caused almost exclusively by *streptococcus*, but now at least 50% of cases are from *staphylococcus*, and in HIV patients, may harbour Gram-ve organisms. *De novo* cellulitis, in the absence of diabetes, is a frequent presentation of HIV, which should be tested for. *Actinomycosis* produces a chronic cellulitis discharging yellowish granules in suitably anoxic conditions. In Southeast Asia, check for melioidosis (6.10).

SPECIAL TESTS
Test the blood or urine for sugar, and screen for HIV.

DIFFERENTIAL DIAGNOSIS
You may have difficulty differentiating cellulitis from a **deep vein thrombosis** (DVT): this is usually less shiny, not warm, and not so tender but the diagnosis of DVT is notoriously hard: you really need a Doppler to be sure as Homan's sign is useless.
There may even be cellulitis together with a DVT, which occurs especially in those >40yrs, after pelvic or prolonged surgery, with patients using an oral contraceptive, and those having had prolonged air or bus travel.
In **necrotizing fasciitis** (6.23), the skin is not shiny but dull and purplish.

TREATMENT
Start IV cloxacillin 1g stat, then 500mg qds preferably; if penicillin is ineffective, valuable time may be lost trying it out. However, the most important thing is to *elevate* the limb so that (for the leg) the *big toe is level with the nose* and (for the arm) the hand is strung up inside a sling on a drip stand, and insist on bed rest. Once the temperature has come down, give antibiotics orally, and when the swelling has reduced (the skin often becomes wrinkly as the oedema disappears) you need no longer continue elevating the limb.

DIFFICULTIES WITH CELLULITIS
If sepsis persists, do blood cultures and change to a different antibiotic, check that the patient has not been walking around, and look for any abscess formation or necrosis. *Make sure any foreign body has been removed.*
If swelling worsens with purplish discolouration and skin peeling, there is developed fasciitis (6.23) and this needs urgent widespread debridement.
If there is chest pain or dyspnoea, think of DVT: if this is more likely, start anticoagulants.
In children, cellulitis is often secondary to acute osteomyelitis (7.3), which needs drilling.
In the diabetic foot (8.17), sepsis often spreads rapidly and even more so with HIV disease, resulting in osteomyelitis and gangrene; radical debridement with amputation of suspect toes is necessary. Frequently you will have to perform a below- or above-knee amputation to clear the sepsis.

6.23 Necrotizing fasciitis

Mixed infection in the superficial and deep fascial tissues with aerobes and anaerobes can cause extremely rapid dissolution of collagen in connective tissue, gross oedema and so interruption of blood supply to the overlying skin, and fat, which necrose. Advance of infection however may be sudden, alarming and relentless, and its extent is greater than at first seems apparent, particularly if there is *mucormycosis* (fungal infestation), which can occur in extensive natural disasters such as volcanic eruptions (34.13).

It can occur anywhere: in the abdomen it is known as Meleney's gangrene, maybe as a result of contamination from a colostomy, or a septic umbilicus (33.5), or in the scrotum as Fournier's gangrene (6.21), maybe as a result of extravasation of septic urine. It may arise after any type of perineal sepsis, including FGM. However, it is often spontaneous, especially in HIV disease and diabetics. The limbs, neck, chest wall and breast may all be affected; in the mouth it leads to gross facial destruction (cancrum oris, 31.5).

There is marked swelling and tenderness with areas of blistering, patchy central necrosis and crepitus; the patient is much sicker than with cellulitis, and pain extends beyond the confines of visible inflammation. The skin is not shiny, but dull and purplish. The necrotic fascia is greyish in colour and has lost its sheen but there is also a telltale milky exudate separating the sick fascia from the fat. Septicaemia soon overtakes, and he becomes very toxic, dehydrated and anaemic.

With certain infections, however, and typically *mycobacterium ulcerans*, the necrosis is slower to develop and limited to subcutaneous fat and results in a well-defined tropical ulcer (34.9), with an undermined edge due to skin survival through development of collateral circulation.

SPECIAL TESTS
Test for diabetes and HIV. Cross-match blood if necrosis is extensive.

DEBRIDEMENT (GRADE 2.3)
Start IV gentamicin or chloramphenicol and metronidazole. Resuscitate with IV saline rapidly to correct dehydration which is almost universal. Add fluconazole if you suspect *mucormycosis.*

Do this in the septic theatre. Excise *all* the affected fascia; this is inevitably more widespread than the overlying skin, and debridement *must* be radical. If you leave dead tissue behind, the patient will die. *(Necrosis involving the breast may mean doing a mastectomy!)* You may not know how far the necrosis has spread, but you must continue till no more grey fascia is found! You may lose a considerable amount of blood, so transfuse especially if he is anaemic to start with. Irrigate the wound with hydrogen peroxide.

You will be surprised how drastically the condition improves if you have done an adequate debridement (and how miserably it deteriorates if you haven't). Extend the debridement if you find more necrosis. Inspect the wound bd, and skin graft the defect when it is clean. You can speed up this process dramatically by using suction dressings (11.13).

MAZHOU (36yrs) was brought to a small mission hospital *in extremis*. He had uraemic frost, he was hardly conscious with shallow breathing, and had necrotizing fasciitis extending from the base of the scrotum to the costal margins. Whilst intravenous saline was poured in, under oxygen alone all the necrotic fascia was cut away: it hardly bled, and gave off ammonia fumes! Towards the end of the procedure he started moving and needed nitrous oxide to finish the operation. The next day he was conscious and hungry. He then explained that the scrotal swelling began after someone forcibly removed a urethral catheter that had been inserted when he'd been admitted with cerebral malaria. He was faithfully married with 3 children, and later tested HIV-ve. The urethral stricture was later successfully dilated, and the extensive abdominal wound grafted.
LESSONS (1) Extensive surgery is possible *in extremis* with no or hardly any anaesthesia. (2) Radical debridement gives results. (3) A small blunder gave rise to a huge problem. (4) Urethral catheterization is invasive and potentially hazardous. (5) Not everyone who is moribund is HIV+ve.

6.24 Gas gangrene

This is an anaerobic infection of injured muscle caused by various species of *clostridia*. Suspect that it may occur if:
(1) There are extensively lacerated muscles, or a missile wound, especially if this involves the buttocks, thighs, or axillae, or the retroperitoneal muscles following an injury to the colon.
(2) The blood supply to these parts of the body has been interfered with.
(3) The wound is grossly or deeply contaminated with soil.
(4) There is prolonged dead conceptus in the uterus.

Gas gangrene is probably developing *if there has been satisfactory progress and then sudden deterioration*. Over 2-3h the patient becomes anxious, frightened, or euphoric. The face becomes pale or livid, often with circumoral pallor. The injured limb feels uncomfortable and heavy. Although there may be recovery from shock and no bleeding, the pulse rises. It quickly becomes feeble as the blood pressure falls. There may be vomiting. The wound may have a sickly-sweet smell of apples.

Don't let these features mislead you:
(1) *There may not always be the smell of death*, and even if there is, there may not be gas gangrene.
(2) *Gas in the tissues is a late sign*, and even if it is present, it does not always mean gas gangrene.

One of the muscles may be involved, or more often a group of them, or a whole limb, or part of it. Infection spreads up and down a muscle, and has less tendency to spread from one muscle to another. As infection progresses along a muscle, it changes from brick red to purplish black (6-17). At first the wound is relatively dry; later, you can express from its edges a thin exudate with droplets of fat and gas bubbles, which becomes increasingly offensive. Stain this and look for Gram+ve rods.

N.B. Try to prevent gas gangrene:
(1) Always perform a thorough wound toilet, especially in all extensive muscle wounds of the buttock, thigh, calf, axilla or retroperitoneal tissues. Use plenty of clean water, and remove dead tissues and foreign material. *Never close these wounds primarily.*
(2) Administer prophylactic antibiotics such as cloxacillin, gentamicin or chloramphenicol. Start immediately after the injury for a maximum of 24h. There is probably no absolute need for prophylactic antitoxin serum, which is probably hard to obtain, if you have performed a thorough wound debridement.

Once gas gangrene has developed, *don't delay exploring the wound because there is hypotension*. Radical excision and massive doses of penicillin are the only hope. You will be wise to excise too much muscle rather than too little.

GAS GANGRENE: AREAS AT PARTICULAR RISK

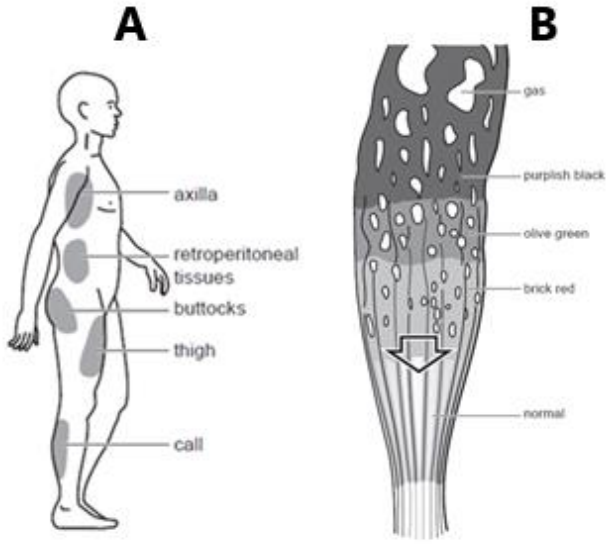


Fig. 6-17 MUSCLE CHANGES IN GAS GANGRENE.
A, areas which are at risk. B, as the infection advances down muscle, its colour changes from its normal purple, through brick red and olive green, to purplish black.

ANY MUSCLE WOUND IS A POTENTIAL SITE FOR GAS GANGRENE

DIFFERENTIAL DIAGNOSIS. Gas gangrene is *not the only cause of gas in the tissues*. Air sometimes escapes into the tissues from under the skin. In ischaemic gangrene (35.2), there is *no toxaemia*, unless the gangrenous tissue becomes secondarily infected. The diagnosis is usually clear. There are however 2 other conditions where the diagnosis is not so obvious. Both require drainage and penicillin or doxycycline but neither needs radical muscle excision.

Suggesting necrotizing fasciitis (6.23): Infection is limited to the subcutaneous tissues, but the patient is toxic and may be uraemic. Spread may be rapid and there may be much subcutaneous gas. Sometimes the whole abdominal wall is involved. When you remove the affected tissue, the muscle underneath appears healthy, and bleeds and contracts normally. Remove *all* the necrotic tissue, and drain the wound.

Suggesting anaerobic streptococcal myositis: Spreading redness and swelling originating in a stinking discharging wound with Gram+ve cocci and pus cells in its exudate. The muscles are boggy and pale at first, then bright red and later pale and friable. The characteristic toxaemia of gas gangrene does not develop. Make radical incisions through the deep fascia to relieve tension and provide drainage.

TREATMENT FOR GAS GANGRENE

NURSING. Isolate the patient from the other surgical patients. If possible, barrier nurse him.

ANTIBIOTICS. Treat with 10MU benzylpenicillin IV qds for 5 days. Or, use ciprofloxacin 400mg IV bd.
Culture the wound, do sensitivity tests, and if necessary change the antibiotics. Although *clostridia* are not sensitive to metronidazole, some other anaerobic bacteria are and may coexist in the wound, so use it.
There is no need to use ANTITOXIN.

RESUSCITATION. Infuse IV saline rapidly, and keep this running during the operation. You may need to transfuse blood if there is severe anaemia.

EXPLORATION (GRADE 1.3). *Do this in a septic theatre*. Open the wound, enlarge it if necessary, lengthwise in the limb, and cut the deep fascia throughout the whole length of the skin incision. Excise all infected muscle widely. Remove:
(1) Any black crumbling muscle.
(2) Any muscle which is swollen and pale and looks as if it has been boiled.
(3) Any muscle which does not contract when you pinch it.
(4) Muscle which does not bleed.
(5) Muscle which contains bubbles of gas.

If necessary, remove whole muscles from their origin to insertion, part of a large muscle, or a whole group of muscles. Remove any suspect muscle: if you leave any dead muscle behind, he will die. Excise also any dead tissue. Irrigate the wound with hydrogen peroxide. Close the wound later by secondary suture, with a skin graft or flap.

AMPUTATION. If the limb is disorganized by injury or infection, amputate it (35.3), especially if there are signs of severe toxaemia. Take a radiograph of it first to see how far the gas has reached. Amputate under a tourniquet. When you have amputated, the toxaemia should improve rapidly.
CAUTION! Close the stump by delayed primary suture, even if you think you are amputating through healthy tissue.

POST-OPERATIVE CARE. Septic shock and/or fat embolism may develop if it has not already done so. Expect, and treat as best you can, the dehydration, vomiting, delirium, jaundice, and anuria that may develop.

GAS GANGRENE

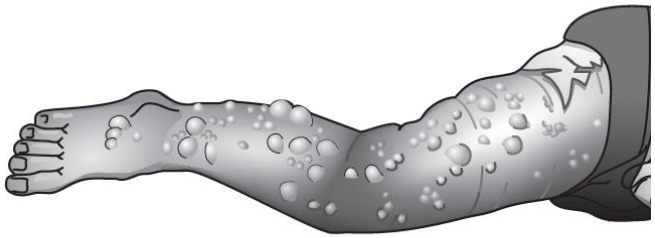


Fig. 6-18 SEVERE GAS GANGRENE.
This followed an intramuscular injection, but it could equally well have followed a severely contaminated wound.
Adapted from a photograph, Fry,D. with kind permission of Tropical Doctor.

7 Pus in the muscles, bones & joints

7.1 Pyomyositis

Pyomyositis describes abscesses forming inside striated muscle. It is common between 5-35yrs, especially with HIV disease and in those debilitated by poverty, diabetes, steroid therapy or cirrhosis. One or more muscles become exquisitely painful, tender, and swollen, and the skin overlying smooth and shining. A single muscle may be involved, or a group of them, or several in different parts of the body. The larger muscles, such as those of the thighs, buttocks, shoulders, back, and abdominal wall, are more often involved than the smaller ones, though this ratio seems to be reversed in HIV disease. Infection makes them hard and indurated, so that movement is painful. Later, the signs of inflammation may subside as the infected muscle is replaced by pus and becomes fluctuant. Infection of the muscle limits the movement of joints nearby. Serious complication with fever and rigors is common as bacteraemia ensues. Lymph node involvement is not conspicuous. Septicaemia associated with pyomyositis may be fatal and is often not diagnosed. The patient is very ill and drowsy, with a high fever and multiple tender areas over the muscles. He may have a history of a trivial skin laceration, a blister, or a small sore. The condition rapidly progresses, so that he becomes desperately ill with a swinging fever, weakness, prostration, dehydration and hypotension. Pyaemia associated with pyomyositis results in a sequence of abscesses in one muscle after another.

Staphylococci are responsible in 90% of cases but Gram-ve organisms may be found especially with HIV disease. Also, an abscess in muscle may arise secondary to hydatid disease (15.12), cysticercosis, actinomycosis, or a haematoma from any injury. If *streptococcus* is the cause, the prognosis is usually worse.

SITA (38yrs) presented with fever and a vague, mild pain in her left hip, which was made slightly worse by movement. No malaria parasites were found and no definite diagnosis was made. She was treated with gentamicin and cloxacillin and her fever improved. Ten days later she returned with a huge abscess in her left inguinal region. This was incised and she recovered completely. LESSON Pyomyositis may cause large abscesses in the deeper muscles with few localizing signs.

DIFFERENTIAL DIAGNOSIS includes osteomyelitis (7.2) and septic arthritis (7.16). The exact site of the tenderness and swelling will usually lead you to the correct diagnosis. There are several other possibilities which depend on the site of the abscess:

In the upper abdomen, pyonephrosis or a perinephric abscess (6.8), a liver abscess (15.10), a subphrenic abscess (10.2), or an acute abdomen (12.1).

In the lower abdomen, an appendix abscess (14.1), an iliac or psoas abscess (6.16), a strangulated groin hernia (18.6, 18.8), pelvic abscess (10.3) or PID (23.1).

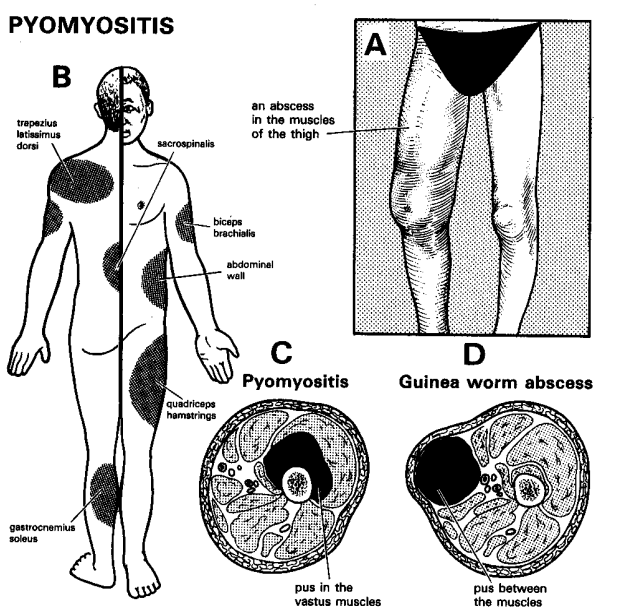


Fig. 7-1 PYOMYOSITIS.
A, shows an abscess in one of the muscles of the thigh. B, the common sites. C, D, the distinction between pus in the muscles (as in pyomyositis), and pus between them, as in an abscess round a dead Guinea worm (34.8)**.**
After Adeloye A, Davey's Companion to Surgery in Africa, Churchill Livingstone Edinburgh 2nd ed 1987 p143-4 Figs. 12.1,4 with kind permission.

In the thigh, acute osteomyelitis (7.3), guinea worm infection (34.8), a haematoma, or a sarcoma (34.15).
In the calf, deep vein thrombosis, cellulitis (6.22) or a sickle cell crisis with bone infarction.

DRAINAGE (GRADE 1.4). If you are not sure whether pus is present or not, aspirate it with a large bore needle. If you are not sure where to point your needle, use an ultrasound to guide you if possible. Make a small incision to begin with, if possible in the most dependent position, and open the abscess by Hilton's method (6-3). If it is large, extend the incision, so that you can insert your finger, break down any loculi and explore the whole cavity. *Don't use a curette*. You may find >1L pus: make sure your incision drains it adequately.

If the bone feels rough and craggy at the bottom of the abscess cavity, it may be involved; if so, this is osteomyelitis, *not* pyomyositis. Chronic osteomyelitis (7.5) may develop later.

If there are signs of spreading infection (cellulitis), treat with IV cloxacillin as well as draining the abscess(es) and taking a culture of the pus.

If there is hypotension with septicaemia, correct hydration with large volumes of saline or Ringer's lactate, and if there is severe anaemia, transfuse blood whilst you drain the abscesses. Use IV cloxacillin and chloramphenicol (2.7,8). Change these if different sensitivities are reported. *Don't forget to drain the abscesses nonetheless*. If there is already coma from septicaemia, *don't add to the problems by using an anaesthetic!*

If there is a succession of abscesses (pyaemia), drain them as they appear, culture the pus, and treat with an appropriate antibiotic as soon as you know the results of culture. Treat with cloxacillin or chloramphenicol meanwhile. Check the HIV status.

DIFFICULTIES WITH PYOMYOSITIS

If blood pours from the abscess, pack the cavity tightly with gauze for 24h. *Don't curette an abscess.* It may be an infected false aneurysm (35.9); make sure you have vascular clamps ready when you re-explore.

If there are very many or very severe lesions, you may have to make >10 incisions, with repeated staged visits to the theatre, to evacuate pus and remove dead muscle.

If there is overlying black necrotic skin, removing it may reveal a huge quantity of avascular greyish-pink, mushy suppurating muscle extending deeply underneath. Remove this, taking care: (1) *not to injure vital structures*, (2) *not to lose more blood than is inevitable*. Survival may depend on aggressive (but not too aggressive) surgery, intensive antibiotic treatment, and IV fluid replacement. If you have had to remove much muscle, there will inevitably be resulting weakness, deformity, and loss of function, but you will have saved the patient's life! If you are afraid of too much blood and muscle loss, do an amputation (35.3).

If there are fever and rigors after drainage, there is septicaemia, from either new abscesses or inadequate drainage.

If abscesses are near joints and liable to develop contractures (32.1), apply skin traction or a cast, as appropriate.

7.2 The pathology of osteomyelitis

Osteomyelitis is a particularly tragic preventable disease which often disables for life if it is treated late or inadequately. You can treat osteomyelitis satisfactorily only if you treat it early. Later treatment is difficult, expensive, and time-consuming. There are several kinds.

(a) Haematogenous osteomyelitis

The acute stage of haematogenous osteomyelitis is a systemic disease which may be life-threatening. It is associated with sickle cell disease (common in Brazil, Africa and India), and is an indicator of poverty, manifested by poor hygiene and a poor nutritional state. Typically it is an affliction of children between 4-14yrs and is more common in boys, probably because boys are more prone to trauma and boys are socially allowed to be dirtier. There is often a history of minor trauma, such as being kicked on the football field, and the most commonly affected bones are the tibia and the femur where the commonest sites of infection are the distal metaphyses of the femur and the proximal metaphyses of the tibia, where the blood flow is sluggish.

However, other sites of infection are also common: the proximal femur, the proximal humerus, the distal radius or ulna, the distal tibia, or the calcaneus. But any bone can be involved, and sometimes several of them at the same time, especially in neonates where the maxilla is often involved (7.14), and the origin of the sepsis may be umbilical, pneumonic or from gastroenteritis.

Metaphyses are endowed with a rich network of subperiosteal vessels and it appears that the primary lesion is a subperiosteal hematoma. People who walk barefoot and whose skin is exposed to all manner of scratches and thorn pricks and insect bites, people who suffer from pimples, eye and ear infections and who pick their noses, are prone to episodes of bacteraemia. Boluses of bacteria are quickly eliminated from the circulation but devitalized blood is an excellent culture medium and hence if bacteria settle in the subperiosteal hematoma, infection will ensue and a subperiosteal abscess will develop. Pus accumulates under pressure, breaks out and strips the periosteum off the shaft and deprives part of the bone of its blood supply, so that it dies and forms a sequestrum. Alternatively, pus erupts into the muscle and spreads along fascial planes, and so resembles pyomyositis (7.1).

Although acute haematogenous osteomyelitis can be caused by a whole array of micro-organisms, *staphylococci* are by far the most common bacteria implicated, and *salmonellae* are probably the second commonest microbes. *E. coli*, *Haemophylus influenza* and *Salmonella* are often found with sickle cell disease or other haemoglobinopathies.

With HIV disease, haematogenous osteomyelitis occurs in adults as well as children, often with *enterobacteria* but also with all manner of bacteria. There is little periosteal reaction, but osteopenia leading to bone destruction.

Before the age of 6 months, an epiphysis offers no barrier to the spread of infection, so that pus in a metaphysis rapidly spreads to a joint. After this age, the cartilage of an epiphyseal plate limits the spread of infection, so that a joint is infected only if an infected metaphysis extends inside a joint capsule, as in the hip or shoulder.

(b) Acute traumatic osteomyelitis

Here the organisms reach bone directly from an open fracture, particularly if the wound is contaminated in road vehicle trauma, war, or a gunshot incident. The bone in such wounds is *always* at risk especially if there is inadequate wound toilet, or immediate instead of delayed wound closure.

Similarly, infection can reach bone through internal fixation of fractures, and so you must seriously weigh the advantages of such procedures against their risks.

(c) Subacute localized pyogenic osteomyelitis

Here the infection develops insidiously from the metaphysis of a long bone, which cavitates and produces a surrounding reactive bone sclerosis, known as a Brodie's abscess (7-2A). Ultimately the marrow cavity is obliterated.

(d) Late HIV-related foreign-body osteomyelitis

Infection occurs in HIV+ve patients around metallic bone implants years after they have been inserted. This results in septicaemia systemically, and bone destruction locally. The larger the implant, the greater the problem. *Staphylococci* are usually responsible, but you may find many other organisms.

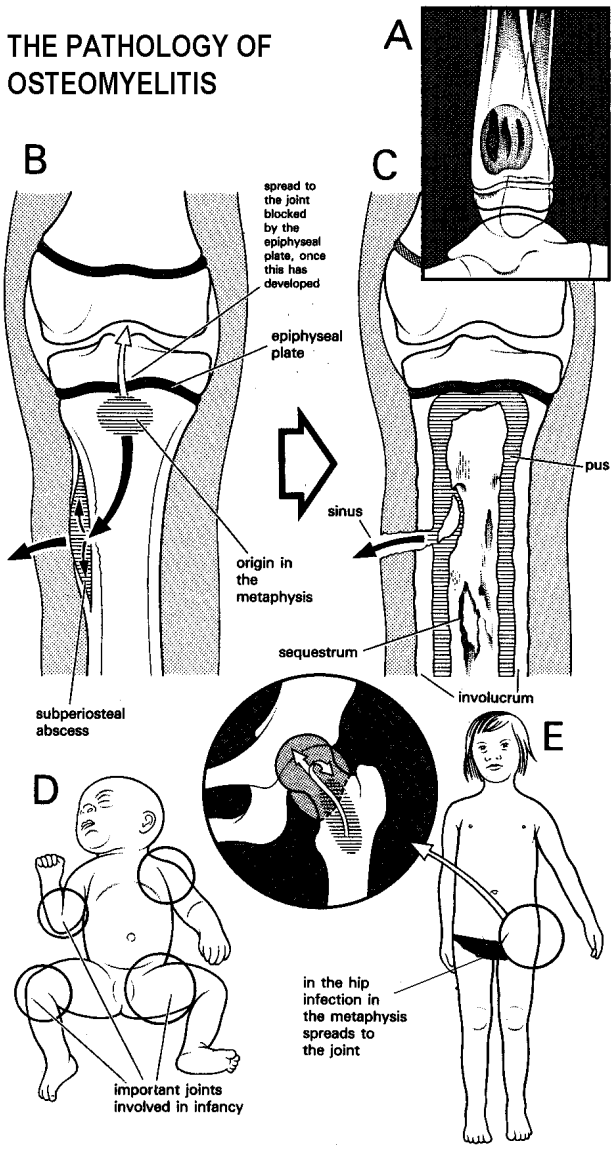


Fig. 7-2 PATHOLOGY OF OSTEOMYELITIS.
A, Brodie's abscess is an uncommon form of chronic osteomyelitis: the upper end of the tibia or the lower femur are the common sites. B, initial infection in osteomyelitis typically is in the metaphysis. After 6 months, the epiphyseal plates have developed sufficiently to prevent infection spreading to the joints, except in the hip. Before this age, infection spreads to the joints. C, chronic osteomyelitis with sequestra and a sinus. D, in a baby <6mths old, osteomyelitis is always associated with septic arthritis. E, osteomyelitis of the proximal femur is always associated with septic arthritis, regardless of the age of the patient, because the epiphyseal line is intracapsular.

(e) Chronic pyogenic osteomyelitis

The stripped periosteum of acute osteomyelitis responds by producing new bone. Where acute osteomyelitis is inadequately treated, the bone dies and is known as a sequestrum. It behaves then as a foreign body allowing infection to persist. The sequestrum becomes surrounded by new bone from the surviving periosteum and this new bone is known as the involucrum. The stability of the bone may depend on this involucrum. Persisting infection within the sequestrum may rupture through the involucrum, producing multiple sinuses.

(f) Other chronic bone infections

Certain other types of chronic bone infection however do occur, because of tuberculosis (32.3), actinomycosis, especially in the jaw (7.14), or mycetoma (34.11). In these there is a locally destructive process with little periosteal reaction, in contrast to the situation with syphilis and yaws.

7.3 Acute osteomyelitis

Acute haematogenous osteomyelitis is a surgical emergency. It is also the supreme example of the axiom, "Where there is pus, let it out." Your challenge is to let out the pus before it causes pressure necrosis of the bone, and to do so with the least possible delay. If you don't explore an infected bone early enough, or don't explore it at all, the patient may become severely disabled. Early operation is not difficult, but the sequestrectomy that may be necessary later will be very difficult.

Typically, a child from a poor family living under unhygienic conditions presents with fever and *an exquisitely painful tender bone near a joint* which he is unwilling to move. Or, the parent may bring him to you with fever, pain, and a limp. When you first see him, the tender area will probably not yet have started to swell. Soft tissue swelling is a late sign which shows that pus has already started to spread out of the bone.

Unfortunately, many children present late after they have already sought help elsewhere. Often, the history is atypical and may be misleading:
(1) There may be no history of an acute illness; the first sign may be a boil-like lesion which discharges spontaneously or is incised, and which is followed by a chronically discharging sinus.
(2) If an infant is very ill, he may have no fever and few general signs of infection.
(3) There may be signs of a severe general infection, but few local signs.
(4) There may be a history of a fall, suggesting a fracture. So think of osteomyelitis in any ill child who is not using one limb.

The only sure way to confirm or exclude osteomyelitis is to decompress the bone, urgently. Many doctors are only used to soft tissue surgery and don't like working on bone and look upon it as specialized orthopaedics. The main message of this chapter is that *you must decompress osteomyelitis early!*

Do your utmost to drain pus from an infected bone before it has stripped the periosteum off the shaft. After this has happened, the bone normally heals by forming a sequestrum and an involucrum, with all the disability that this causes. Early treatment needs early diagnosis, so everyone who provides primary medical care must be aware of osteomyelitis. Make sure that your staff in the clinics know about it, and immediately refer any child with fever and a painful limb. Because of the common practice of giving antibiotics and seeing if the patient improves, osteomyelitis is apt to be one of the worst-treated diseases in primary care. One reason why it is such an important disease in resource-poor settings is that patients are so often referred to hospital late, after they have been inadequately treated in peripheral units.

Any of the diseases in the list below can cause pain, fever, and inability to move a limb. Local redness and oedema are later signs. The important decision is not what the exact diagnosis is, but whether you should decompress bone or not. The site of the greatest tenderness (at the end of a metaphysis near a joint) is a useful point of differential diagnosis, and so is the young age of the patient. The tenderness is localized and is greatest on direct pressure and percussion.

MURARULAL (9yrs) was brought in by the mother with a one-day history of a limp. There was tenderness over the right fibula and he had a low-grade fever, but no other signs, and no radiographic changes. The diagnosis was uncertain, so the fibula was explored. It looked normal when it was exposed, but even so it was drilled. Pus came out under pressure. The wound was dressed and left open and he was given chloramphenicol. He rapidly improved and the wound healed spontaneously. A month later he had no limp and no discharge, but a radiograph showed periosteal elevation. A year later the radiograph was normal.

BUROO (8yrs) was admitted with a swelling over the upper end of her right tibia present for the last 4 days. A small abscess pointed. This was incised and drained. A week later a radiograph was taken and considered normal. After three months of antibiotic treatment, her wound was still discharging, and radiographs showed obvious chronic osteomyelitis.

LESSONS (1) If osteomyelitis is a possibility, drill the bone, especially the upper tibia. (2) Drill it even if it looks normal when you expose it. If Buroo's bone had been drilled early, she would have been spared many years of disability. (3) When you have found pus, leave the wound open.

HIGH FEVER AND A TENDER BONE MEAN OSTEOMYELITIS

DIAGNOSIS

The diagnosis is clinical, but you can easily confirm it by ultrasound scan: look for a subperiosteal fluid collection >7mm in size (38-13). The simplest and fastest method of confirming this subperiosteal abscess is aspiration, which, at the same time, will yield a specimen for you to send for culture.

Ordinary radiographs may not show any abnormality, especially when the disease is early and treatable! Blood tests, particularly the white blood cell count, are unhelpful, as are sedimentation rate and C-reactive protein, for they simply suggest the presence of inflammation.

If a child has a high fever and is acutely tender over a bone, this is osteomyelitis until you have proved otherwise. If the mother tells you that there was an injury up to 2wks before, this may indeed have been true in 50% of cases as increased blood supply to the area may have been the predisposing factor producing the infection. *Radiographs don't help in the early diagnosis of osteomyelitis*, but they will exclude a fracture.

If the tenderness is in the soft tissues, rather than over a bone, this is more likely to be cellulitis or pyomyositis than osteomyelitis.

If the lower leg is swollen, oedematous, tender and warm, but the tenderness is not particularly localized over a bone, should you explore it or not? Its exact site may help you to decide. If you are still in doubt, be safe and drill. You will probably operate on some cases of cellulitis unnecessarily, but if you don't operate, you will miss osteomyelitis.

If the point of maximal tenderness is over a joint, not over the adjacent bone, and all its movements are exquisitely painful, this is probably a primary septic arthritis. Aspirate the joint and, if necessary, drain it.

If there is fever and an acutely painful hip which is extremely painful to move, this is osteomyelitis of the neck of the femur with septic arthritis (they are in effect the same disease). Aspirate to confirm that pus is present (7.16). Drill the upper femur and its neck, and drain the hip (7.18).

If the muscles are swollen and tender, this is probably pyomyositis (7.1): feel the site of tenderness carefully.

If sickle cell disease is common, suspect bony infarction, which is common in this condition, may be causing the symptoms, especially in a child <2yrs, if:

(1) several of the bones are involved

(2) an unusual bone is involved, such as the skull, or the small bones of the hands or feet, particularly in an infant

(3) there is respiratory distress.

So check for sickle cells in the blood, and for anaemia.

N.B. Osteomyelitis can complicate avascular necrosis; both diseases may be present. *There is no certain way of distinguishing a sickle cell crisis from osteomyelitis except by decompression.* If there is sickle cell disease, a wait of 24h is reasonable, because the pain of an infarct improves rapidly with IV morphine. Signs in an SS patient are usually obvious clinically, but are not in SC patients (quite common in West Africa). *Don't transfuse so that the Hb level exceeds 100g/L.*

If lesions in the hands are causing diagnostic difficulties, remember that:

(1) Tuberculous dactylitis is much less painful than sickle cell dactylitis.

(2) Syphilis will probably show abundant new bone formation elsewhere.

If the disease is some weeks old, but there are no signs of new bone formation on the radiograph, suspect that this is tuberculosis, with or without HIV disease. This is most likely to be a diagnostic problem in the spine. Tuberculosis usually forms no new bone, whereas chronic pyogenic osteomyelitis is more likely to. Patients with HIV disease make very little involucrum.
If there is much swelling, but not much fever, suspect that this may be a sarcoma, which can mimic subacute osteomyelitis and may cause fever. Radiographs should distinguish one from the other. Confirm it by biopsy.
If there is a subperiosteal swelling without fever, this may be due to scurvy or a bleeding disorder.
If there is fleeting pain in many joints, this is probably a rheumatic polyarthritis. Rheumatic fever and parvovirus infections are other acute and subacute causes.
If any other septic lesion, such as a carbuncle or middle ear disease, coexists, suspect this may be the source of the osteomyelitis.
If the diagnosis is still difficult, consider brucellosis, yaws, syphilis, and leprosy.

PRESENTATION
The presenting symptoms and signs are pain, inability (or refusal) to move the limb, fever and prostration. As long as the abscess is subperiosteal, there are hardly any local signs.

EXAMINATION. Elicit tenderness and hypersensitivity to vibration by holding a tuning fork against the bone, even distant from the affected area.
Look for a septic problem anywhere, but especially from a child's skin, chest or stool from which the infection may have spread. Culture any skin lesion, sputum and diarrhoea stool.

BLOOD CULTURES. If there is pyrexia, take a blood culture (if you can), and preferably 2 more at 2hrly intervals, before you start antibiotic treatment. If treatment has already started, cultures will probably be unhelpful.

RADIOGRAPHS. *Don't expect any signs in an early case.* You will only see bony changes >10 days in an older child, or >5 days in an infant. Examine the edge of the bone with care: the earliest sign is the faintest second line of new bone about 1mm away from the shaft. You will see this more easily if you look at the film obliquely. Nonetheless it is useful to have a radiograph as a baseline.

IF YOU SUSPECT OSTEOMYELITIS, DECOMPRESS THE BONE AND LET OUT THE PUS!

NEEDLE ASPIRATION using a 16G needle may be useful in localizing pus. Unfortunately, if pus is present under the periosteum the disease is already advanced. Good results are obtained by decompressing bone earlier than this. Aspiration is useful for diagnosing septic arthritis, but not for treatment.

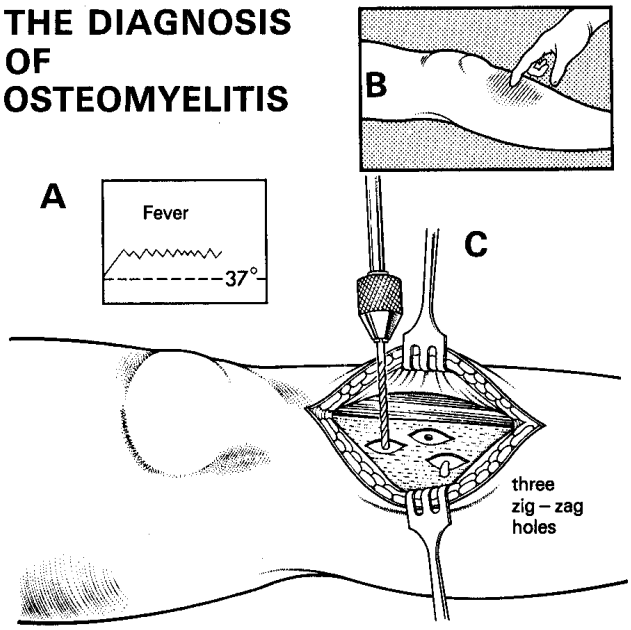


Fig. 7-3 DIAGNOSING OSTEOMYELITIS.
A,B, critical signs: fever and painful tender bone, especially close to an epiphysis. C, the only way to confirm or exclude osteomyelitis is to drill the bone. If a patient comes when pus is already discharging, it is too late for an easy cure.

CAUTION!
(1) Explore the bone (7.4), whether or not you find pus.
(2) *Failure to aspirate pus does NOT exclude osteomyelitis!*

GENERAL CARE AND IMMOBILIZATION. Correct dehydration. Ease pain with IV analgesics. Splint the limb in the position of function, or use skin traction for a leg.

ANTIBIOTICS. Start these immediately after you have taken a pus swab, and if possible a blood culture also. If you have been able to drain the lesion early and it is clinically quiescent, and there is no bone necrosis, continue for 2-3wks. Before you know the results of culture, or if culture is impossible, treat with IV chloramphenicol 10mg/kg qds, IV cloxacillin 10mg/kg qds, or azithromycin IV in sickle cell disease. Monitor the response by a drop in leucocytosis.
Treatment has 3 objectives:
(1) to treat and prevent pain,
(2) to treat the septicemia and the associated inflammatory syndrome, and
(3) to prevent the bone from dying.
You need to administer IV antibiotics and analgesia fast!
Whilst the periosteum is relatively inelastic and cannot accommodate much inflammatory exudation, the bone is altogether incapable to adjust to the pressure rising in the Haversian system. Therefore, as the subperiosteal lesion expands, the periosteum will be stripped from the diaphysis, and as the pressure in the Haversian system eventually exceeds capillary pressure, the bone will become ischaemic and will die. Consequently, the 3rd objective of treatment is addressed by decompressing the subperiosteal abscess.

At a very early stage, antibiotics alone may abort the process, but in the regions where the condition is common, presentation is usually late, and decompression is necessary.

If the bone looks normal, drilling holes through the cortex of the diaphysis into the medullary cavity may in early cases decompress the Haversian system.

The unfortunate circumstance in poor-resource settings is that in the overwhelming majority of cases the bone, or parts of it, are dead at the time of presentation.

If septicaemia persists, grave complications will follow: pneumonia, endocarditis, pericarditis, and 'metastatic' abscesses. Fortunately most patients recover from septicaemia and if the bone has not died, the local inflammation will subside.

If the bone has died, as is usually the case, pain and local signs will continue to be present. After 10-14 days, a radiograph will show the extent of the dead bone: this will be relatively denser than the living bone, for the living bone will have begun to lose mineral density whereas the dead bone will not.

N.B. Damage to the growth plate in childhood may lead to stunted growth, and limb shortening or deformity.

7.4 Exploring a bone for pus

If you suspect that there is osteomyelitis, the critical procedure is to decompress the painful tender bone.

DECOMPRESSION FOR OSTEOMYELITIS (GRADE 1.4)
TOURNIQUET. A bloodless field will make the operation much easier (3.4). Elevate the limb first. *This is essential in sickle cell disease. Don't use an exsanguinating bandage*, because this may spread the infection.

INCISION. Expose the bone on either side of the point of greatest tenderness. Try to incise over a bony surface which is covered with muscle, rather than one which is covered only with skin. Make the incision long enough, and start it at the epiphysis. Incise the oedematous subcutaneous tissues.

If you find pus in the muscles away from the bone, *don't automatically think that there is pyomyositis*. Culture the pus. Make sure you wash the tissues with plenty of water, and create adequate drainage.
If you don't find pus in the muscles, continue your incision down to the periosteum. Incise it longitudinally, and if pus immediately floods up from under the periosteum, culture the pus and make sure there is adequate drainage.
If you find no pus under the periosteum, drill a minimum of 3 holes into the bone in a lazy zig-zag line, starting about 1cm from the epiphyseal line and at least 1cm apart.
Make a separate small incision in the periosteum for each drill hole. Drill at right angles to the bone, not obliquely, because drilling will be easier.

If no pus or tissue fluid under pressure comes out, there is probably no osteomyelitis, provided you really have drilled the tender area. If pus flows from the first hole, send a specimen for culture. Drill 1-2 more holes 1cm apart in a lazy zig-zag line down the shaft of the bone until only blood or tissue fluid flows out of the hole from healthy bone.

CAUTION!
(1) *Don't elevate the periosteum*, because the bone under it will die.
(2) *Don't elevate too much muscle either*, because periosteum receives its blood supply from the muscles over it.
(3) *Don't incise the periosteum beyond the epiphyseal line*, or you may spread the infection to the epiphysis.
(4) *Don't remove any periosteum*, because the bone under the raw area will not regenerate.
(5) *Never drill a row of holes transversely across a bone,* because they weaken it.
(6) *N.B. A single drill hole may not drain an abscess sufficiently.*
(7) *Don't use suction drainage,* because this might suck excessive amounts of bone marrow straight out of the medullary cavity.

POST-OPERATIVELY, if there is any danger that the bone might break, apply a plaster gutter splint. In the lower femur or upper tibia, apply skin traction. If the limb is painful, elevate it.
If at 2wks, the lesion is clinically quiescent, and radiographs show no bone necrosis, stop antibiotics. Otherwise, continue for a maximum of 6wks. Follow up for 3 months; if the radiograph is normal, then you have succeeded. Unfortunately, even early decompression is not guaranteed to save the bone, though *you must try!*
CAUTION! If the bone is very osteoporotic, apply a cast before discharge to prevent a pathological fracture, especially if the leg is involved.

DIFFICULTIES WITH ACUTE OSTEOMYELITIS
If a child has radiographic changes on the first visit, chronic osteomyelitis will follow. Proceed as above: pus in the tissues or under the periosteum will need draining.

If the child is aged <6 months, osteomyelitis arising in the metaphysis is inevitably complicated by septic arthritis. Drain the joint also. Bone necrosis is less likely, because the arteries are not end arteries.

DON'T REMOVE A LARGE SEQUESTRUM UNTIL THERE IS A STRONG INVOLUCRUM

7.5 Chronic osteomyelitis

If there is dead bone (sequestrum), the condition is necessarily chronic. The sequestrum acts as a foreign body and maintains a chronic infection. However, here the general principle, that you must remove all dead tissue, needs delay in case the result destabilizes the limb.

If necrosis involves the entire width of the diaphysis as seen on a radiograph, in order to retain a limb that eventually can recover function, the sequestrum has to remain *in situ* so that it serves as a matrix for the newly forming bone (involucrum) that is made by the surviving periosteum. *Not only must the sequestrum be retained, it must be kept in position to avoid a pathological fracture.* You can achieve these objectives by applying a plaster cast or using an external fixator. You must leave holes in the plaster corresponding to any sinuses, so these may drain. *Contrary to common practice, antibiotics are not indicated at this stage*.

The timing of the removal of the sequestrum depends on the strength of the involucrum, but this itself may be weakened by removing the sequestrum!

Don't remove a sequestrum until a patient has formed enough involucrum to make a new shaft for the entire bone. Deciding when to operate is critical.

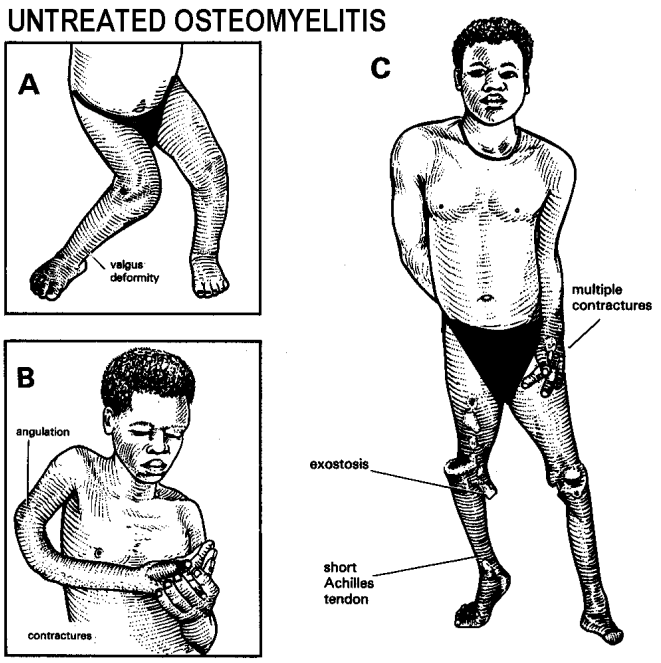


Fig. 7-4 UNTREATED OSTEOMYELITIS.
A, late osteomyelitis of the knee with a severe valgus deformity. B, destruction of the humerus causing angulation, combined with contractures of the elbow and wrist. C, osteomyelitis in several joints. This patient could run with simple boots after excising the exostosis, and lengthening both the Achilles tendons. So save a patient's limb if you possibly can: amputation (35.3) **is usually avoidable unless there is HIV disease.** *Kindly contributed by Ronald Huckstep.*

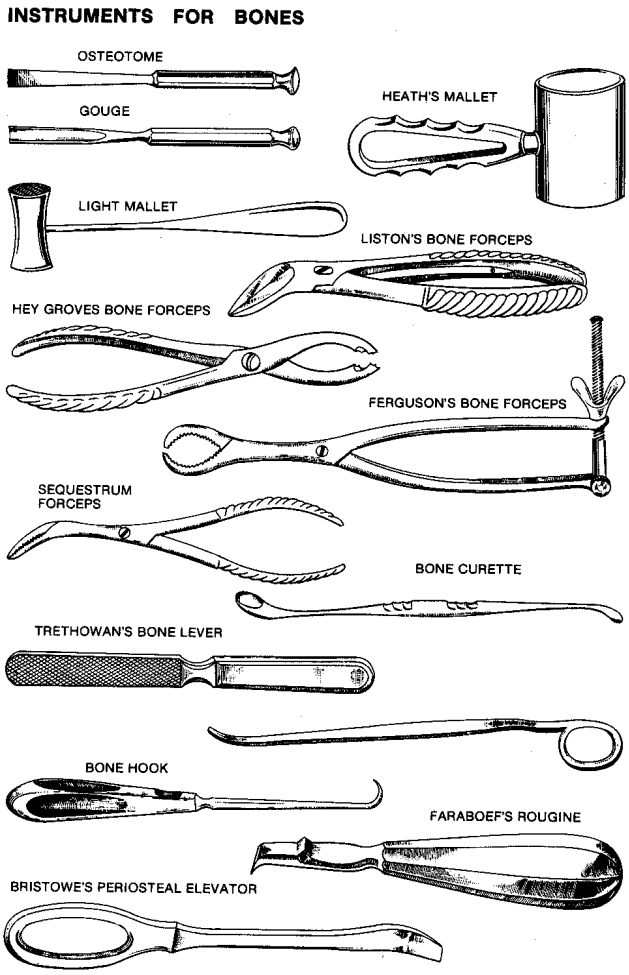


Fig. 7-5 INSTRUMENTS FOR CHRONIC OSTEOMYELITIS.

OSTEOTOME, Swedish model, solid forged stainless steel, (a) 6mm. (b) 10mm. Use these for cutting the bones of children. An adult's bones are too hard to be cut by an osteotome alone. Weaken them first with a line of drill holes.
BONE NIBBLER.
GOUGES, Swedish model, solid forged stainless steel, (a) 6mm, (b) 10mm. These curved bone chisels must be sharp. If necessary, get them sharpened on a grindstone. Use them for deepening a cavity in a bone.
MALLET, stainless steel, 350g. This is an adequate size of mallet, there is no need for a larger one.
BONE FILE or rasp.
FORCEPS, bone cutting, Liston, angled on flat, 200mm. These are general-purpose bone cutters. You can also use them instead of special rib cutters.
FORCEPS, bone-holding, Hey Groves, 210mm. This is for small bones, such as the radius.
FORCEPS, bone-holding, Lane's, 390mm. This is a heavier pair of forceps for larger bones such as the tibia.
FORCEPS, sequestrum, angled, 190mm. These are slender, angled forceps to remove sequestra.
CURETTE, or scoop, Volkmann, double ended, size C. Use this to curette infected bone when you operate for osteomyelitis.
LEVERS, bone, Trethowan, 220mm. Put these round a bone to expose it.
LEVERS, bone heavy, 275mm.
HOOK, bone, 220mm.
ROUGINE, Faraboef, with curved end, chisel edge. Use this to scrape the periosteum from a bone.
ELEVATOR, periosteal, large.

Surgery for chronic osteomyelitis is difficult, bloody, and dangerous. If you have to operate, do so only to relieve persistent pain or remove persistent sinuses, not merely to improve the radiographs.

If an area of bone is abnormally dense on the radiograph, showing that it is dying or dead, it may be absorbed slowly if it is attached to existing healthy bone. But if it is lying free as a sequestrum, it will act as a foreign body and will not be absorbed, so you will have to remove it. Occasionally, you can remove a small sequestrum through a sinus, but you usually need to cut a window in the involucrum. Once you have removed a sequestrum, no new involucrum will form. This is an important exception to the general rule that a foreign body should be removed immediately, especially in the presence of infection.

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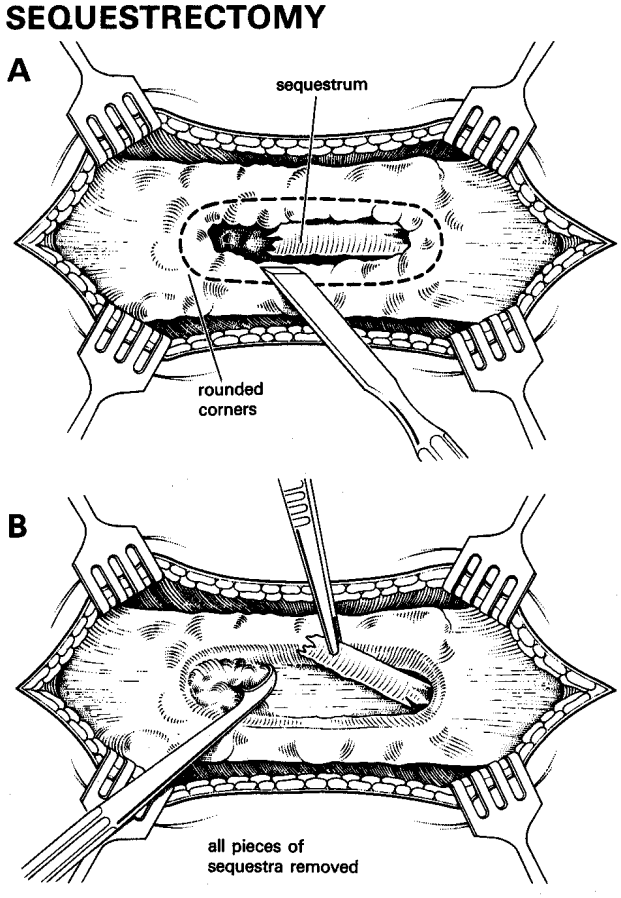


Fig. 7-6 SEQUESTRECTOMY.
A, sequestrum presenting through a *cloaca* (hole) in the bone. B, enlarge the *cloaca* and remove the sequestrum.
Kindly contributed by John Stewart.

Antibiotics will not produce a cure, except perhaps if the infection (in Southeast Asia) is due to melioidosis (6.10). So, explore, curette, and if possible saucerize the cavity (*i.e.* obliterating the cavity by making the hole flat). This will relieve the pain dramatically. If possible, leave it open to the outside, and let it granulate from the bottom. If not, leave it open to the soft tissues.

When you have removed a sequestrum, there may be a defect in the soft tissues or skin; if the wound granulates, you can place a skin graft over it. Otherwise a complex flap may be necessary.

Encourage a strong involucrum to form by exercising the limb so that the newly growing bone of the involucrum is gently stressed, without being angulated or shortened. For example, in the femur use a trunk-to-groin (hip spica) or groin-to-knee cast, add crutches and allow cautious weight-bearing. Occasionally there is localized sclerotic osteitis without an involucrum (Brodie's abscess). *Always send tissue for histology to exclude TB or cancer.*

SEQUESTRECTOMY (GRADE 2.5)

INDICATIONS. Consider removing any sequestrum which you cannot remove through a sinus. *Don't operate to remove a large sequestrum until:*

(1) The involucrum extends across the defect that will follow.

(2) The involucrum is made of rigid bone.

(3) The limb is capable of being supported, either by the remaining healthy shaft, or by a sufficiently strong involucrum.

CAUTION! If you remove the sequestrum too early, the involucrum will stop making new bone, and will collapse, so that there is no hope of a sound limb.

RADIOGRAPHS. Examine AP and lateral films carefully to see where the sequestra are. If ordinary films don't show enough detail inside the bone, take more with greater penetration. *Don't operate just for radiographic appearances!*

PREPARATION

ANTIBIOTICS. Culture the pus or liquid from a sinus, and start the appropriate antibiotic in high dose for 2-3 days before surgery.

METHYLENE BLUE may help to show up sequestra during an operation. Sterilize a 1% solution, and inject it into the sinus 24h beforehand. It will stain everything blue, except the sequestra, which will remain white.

EQUIPMENT. As for acute osteomyelitis (7.3), plus 6 and 10mm osteotomes and gouges; 10 and 15mm chisels; a 250g mallet, a Volkmann's scoop, a curved sequestrum forceps, and a bone nibbler. In the thigh you will need strong retractors, a strong assistant, and a good light. Use an ordinary electric drill (held in a sterile glove) with a rotation saw (which you can autoclave).

TOURNIQUET. Bleeding can be alarming, because infected tissues are very vascular, so always use a tourniquet (3.4), unless you are operating on the proximal femur or humerus, or there is sickle cell disease (7.4). The anatomy may be very distorted, and without a tourniquet, important structures will be very difficult to recognize. Tie any vessels you see as you operate. Have blood cross-matched, and infuse IV fluid.

INCISION. The choice of incision will depend on the anatomy of the sequestrum, the involucrum and the neuro-vascular structures of the limb. (Incise the tibia antero-medially, and the femur laterally.) Start by probing any sinuses to see where they extend. They often join up. Where possible, make one of the standard incisions described. These are given for the entire length of the bone.

Use ketamine for GA.

You will usually only need part of an incision. Very often it will include the draining sinuses. If possible, make the incision over one of the larger gaps in the involucrum. The tissues will be tough, so use a sharp scalpel.
Open the indurated periosteum in the length of the incision, and elevate it on each side. You will have to make a hole by chisel or drill and rongeur in the involucrum so that you can extract the sequestrum. Either: enlarge an existing gap in the involucrum with a gouge. Or: drill holes so as to outline a window (7-6). Then open it with an osteotome.

CAUTION!

(1) Scar tissue may have disturbed the normal position of the nerves and arteries.
(2) *Don't break the bone*. If you have carefully outlined the window with drill holes, this will be less likely.
Use a hammer and gouges or chisels to cut bits of bone from the involucrum until you get to the marrow cavity. Look for sequestra inside it.

Sequestra move separately from the surrounding involucrum. If they have been covered by tissues, they are ivory white and have a brittle texture which is different from ordinary bone. If they have been exposed to the air, they may be black or grey.
Sometimes it is necessary to break the sequestrum and remove it piecemeal. To prevent the bone splitting, use a drill with a rotation saw instead of a hammer and gouge to chip away the involucrum around each sequestrum so that you can remove it. To minimize weakening, make a window 1x10cm in the cortex longitudinally. Round or taper the ends of the window; these will be stronger and allow it to fill with soft tissue more easily. Pull out sequestra with sequestrectomy forceps. If necessary, remove more involucrum to free a sequestrum. There will be pus, but usually not much: send it for culture.
When you have removed all the sequestra you can find, explore the abscess cavity up and down quite widely with a probe. If necessary, extend the skin incision and enlarge the hole in the involucrum until you have explored the whole cavity. You may need a 2nd osteotomy in the diaphysis. Scrape the granulation tissue in its walls with a bone curette (Volkmann's spoon), until you reach bleeding healthy bone. If sinus tracts in the soft tissues are short, excise them. If they are long, curette them.
If bone overhangs the edge of the cavity, chisel it away. Lavage the cavity with warm water.

CAUTION! If the operation is to succeed, you must remove all sequestrated bone. The radiographs will suggest how much there is, but expect to find more. Allow muscle to fall into the cavity (7-10); if this is inadequate, mobilize a flap of muscle, preserving its blood supply, to fill the cavity.

CLOSURE. Complete meticulous haemostasis is essential. A suction drain may be beneficial to avoid accumulation of blood. Fix a sponge drain to the wound with a stitch, because a simple drain will block or get lost in the wound, and act as a foreign body.
Apply a pressure dressing for the first 48h, but watch the circulation distally.

After some weeks there will be a floor of healthy granulation tissue, which will either epithelialize spontaneously, or can be grafted. As you change the dressings you will find that fewer are needed as it closes. A large wound takes a long time to close.

CAUTION! Remove all the dressings you put into a wound. If any fragments remain, they will act as foreign bodies, and cause infection to persist. If you use pieces of gauze to pack a wound, knot them together, so that you can pull them all out at the same time.

POST-OPERATIVELY, the wound will ooze. Do all you can to improve nutrition. You will need quantities of sterile dressings. Change them regularly. Remove any dead tissue as necessary. *You may need to perform further formal sequestrectomies under GA to do this.* After you have removed all the dead tissue, the disease process comes to an end and rehabilitation can begin. Encourage use of the limb, walking with crutches without weight-bearing if the lesion was in the leg, and the use of the arm as much as possible. In severe cases this active movement will encourage the periosteum to produce a really robust involucrum, which will not happen if the limb remains completely immobile.
If the involucrum might fracture, apply a cast and window it. Or, in the leg, apply skin traction. If a large area of bone has been destroyed, careful splinting is essential.
Get radiographs at a convenient time post-operatively. This is only necessary to assess the strength of the leg for weight-bearing, or, if sinuses persist, to look for more sequestra.

DIFFICULTIES WITH CHRONIC OSTEOMYELITIS

If there is severe bleeding into the dressings, return to theatre, open the wound, tie off any bleeding vessels, repack it tightly, and apply a pressure bandage. Back in the ward raise the limb, and put a cradle over it, so that you can inspect it readily. *Don't leave a pressure dressing in place for >48h*, or it will promote infection.
If pus continues to discharge from the wound, it may be due to:
(1) Inadequate excision of fibrous tissue and curettage of the granulations.
(2) Leaving sequestra behind.
(3) Leaving a swab or piece of dressing in the wound.
(4) Not opening up the cavity in the bone widely enough.
If the leg has malunited in a deformed position, an osteotomy may be necessary.
If there is a pathological fracture, splint the limb in the correct position in a cast until it has healed soundly. While it is healing, pay special attention to the alignment of the knee and ankle. Keep the wound open, dress and toilet it regularly. Skin traction is suitable for the femur and upper tibia, especially <14yrs. Otherwise an external fixator is best.
If osteomyelitis has followed internal fixation with a plate, remove it. The only exception is an AO compression plate. If this is still maintaining compression, leave it, but if it is holding a gap open between the fractured ends, remove it.

In an infant, the bone will probably heal well, even after you have removed a large sequestrum. If an operation is needed, *don't hesitate to operate as soon as a satisfactory involucrum has formed*.
If there is sickle cell disease, new bone will form particularly slowly.

Amputation (35.3) is justified if:
(1) The infection is so extensive that antibiotics and surgery have been unable to arrest the disease. This is usually the case with HIV-related osteomyelitis.
(2) Life is in danger from infection.
(3) So much bone has to be removed that the leg is useless.
(4) There is constant pain.
(5) There is no chance of referring the patient for cancellous bone implantation to reconstruct the leg.

7.6 Osteomyelitis of the humerus

Osteomyelitis usually occurs at the ends of the humerus, more often at the upper than the lower end. You can expose and drill the bone through quite limited incisions; the upper end anteriorly and the lower end either anteriorly or posteriorly. If absolutely necessary, you can expose the humerus from end to end by approaching it from the antero-lateral side.
The main danger is that you may injure the radial nerve, as it winds round the humerus posteriorly. If you are working near it, find it first so that you can avoid it.
Proximally, enter the arm between the *pectoralis major* and the *deltoid*. Distally, enter it between the *brachioradialis* and the *biceps*. As you do so, retract the radial nerve laterally, and the musculo-cutaneous nerve medially with the *biceps*.

(a) Proximal end
Approach this in the deltopectoral groove. Find the cephalic vein, and try to displace it medially. If necessary, tie it proximally and distally. Reflect the deltoid laterally, and expose the humerus by using two pairs of bone levers. Both the heads of *biceps* and *coracobrachialis* lie medial to the insertion of the tendon of *pectoralis major*.

(b) Distal end, posterior approach
Make a midline incision in the posterior surface of the upper arm, and end it 3cm above the epicondyles, so as to avoid the olecranon pouch. *Don't extend the incision up into the middle third of the arm*, or you will injure the radial nerve. Divide the tendon of the triceps and the muscle under it to expose the humerus.

(c) Distal end, anterior approach
Open the arm between the *brachioradialis* laterally, and the *biceps* medially (7-7B). Separate these muscles by blunt dissection, find the radial nerve and leave it laterally. Incise the *brachialis* medial to the nerve and expose the humerus. Retract the muscles by placing two pairs of bone levers subperiosteally.

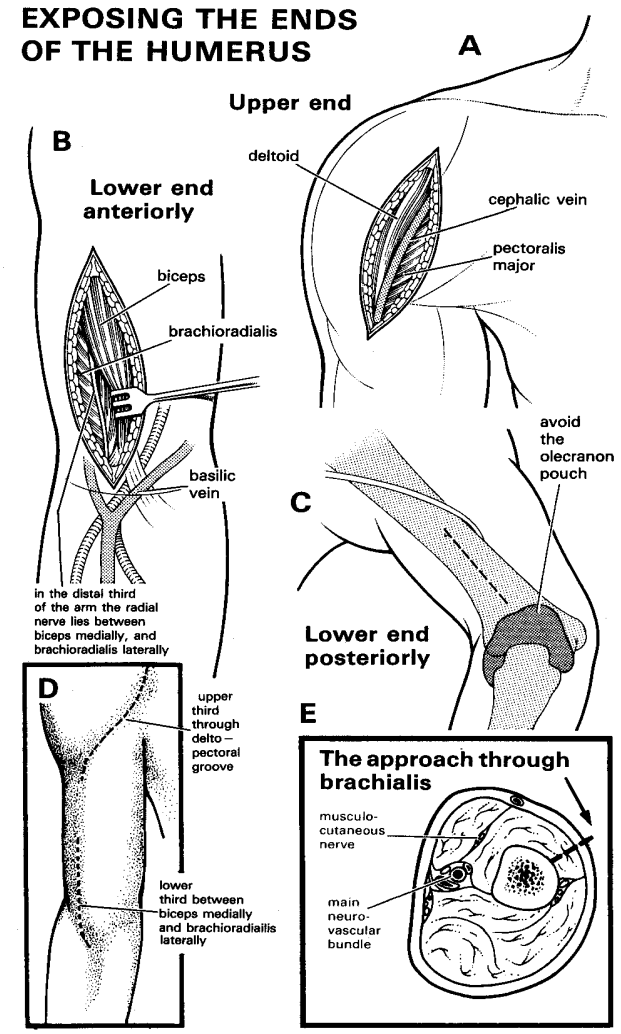


Fig. 7-7 OSTEOMYELITIS OF THE HUMERUS.
A, approach for the upper end. B, anterior approach to the lower end. C, posterior approach to the lower end. D, incisions to approach the ends of the bone. E, cross-section a little below the mid-point of the arm above the origin of *brachioradialis*, to show the approach to the middle of the shaft and the position of the radial nerve.

If necessary, you can split the *brachialis* to within 3cm of the epicondyles without entering the elbow joint. *Don't extend the incision beyond the flexor crease of the elbow*, because you may cut the radial artery.

(d) The shaft
Put a sandbag under the shoulder on the same side. Drape the whole arm. Extend the approach to the upper humerus distally, or the lower anterior approach proximally. Distally, divide the deep fascia to expose division between *biceps* and *brachialis*. The musculo-cutaneous nerve lies between these muscles. Displace it medially with the *biceps*. Separate the *biceps* and *brachialis* and find the radial nerve. Above the origin of the *brachialis*, it lies between *biceps* and *triceps* and winds posteriorly round the humerus in the radial groove.
Post-operatively, put the arm in a sling and encourage active movements within the confines of the sling, or apply a backslab.

7.7 Osteomyelitis of the radius

You can expose the distal ⅔ of the shaft of the radius by approaching it from its anterolateral side. The difficult part is its proximal ⅓, which is covered by the *supinator* muscle, through which the posterior interosseous nerve passes. So avoid operating here if you possibly can. Enter the forearm between the *brachioradialis* laterally (it has a characteristic flat broad tendon) and the *flexor carpi radialis* medially. The radial artery lies between these 2 groups of muscles. *Pronator teres* is inserted into the middle of the radius. You can approach the bone on either side of this muscle, and displace it medially or laterally. Distally, *pronator quadratus* covers the radius, so you will have to divide it.

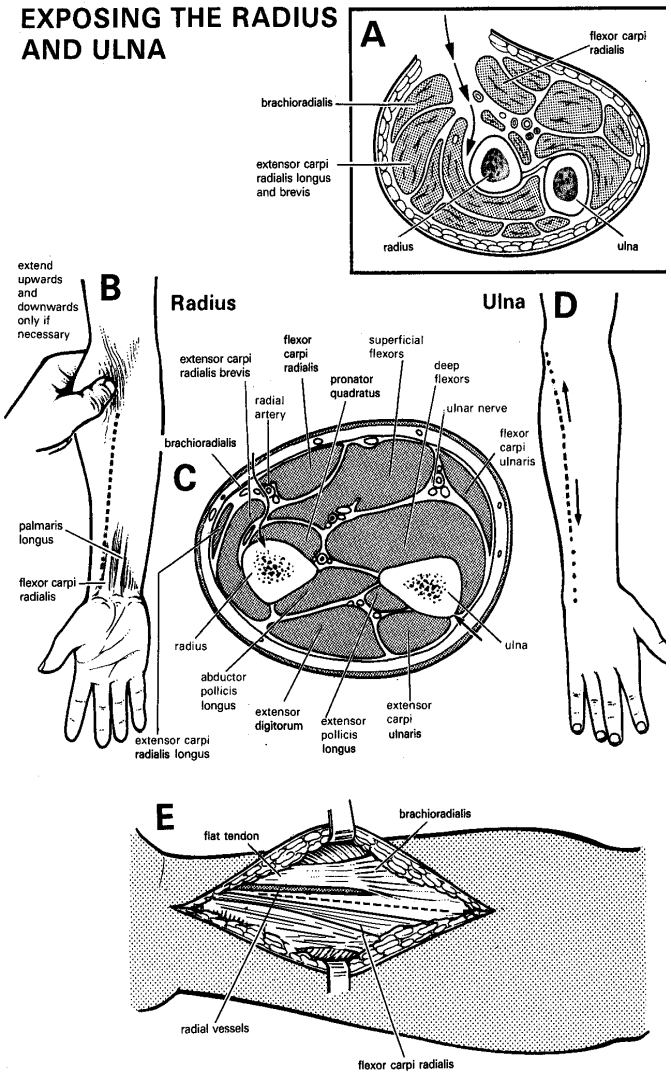


Fig. 7-8 EXPOSING THE RADIUS AND ULNA.
A, cross-section of the forearm at the level of the radial tuberosity. B, to expose the radius, enter the forearm between the *brachioradialis* and the two radial wrist extensors laterally, and the *flexor carpi radialis* medially. C, a transverse section through the middle of the arm. D, the ulna is subcutaneous, so you can approach it easily. E, *brachioradialis* has a long flat tendon, so you can recognize it easily. It and *flexor carpi radialis* flank the incision for the lower part of the radius. *Partly after Watson-Jones R, Fractures & Injuries, E&S Livingstone 1960, with kind permission.*

(a) Distal end

Lay the patient supine with the arm on a side table and the forearm supinated. Define the line of the incision by identifying the tendons of the *palmaris longus* and the *flexor carpi radialis* at the wrist. Incise just lateral to this muscle (7-8B).

You will probably only need to incise over the distal ⅓ of the bone. If necessary, you can continue the incision proximally to include its middle ⅓.

CAUTION! Don't extend the incision to the proximal ⅓, or you may injure structures on the front of the elbow.

Cut the deep fascia in the line of the skin incision. Tie any vessels you meet. Retract laterally the 3 muscles that lie along the lateral border of the forearm: *brachioradialis*, *extensor carpi radialis longus* and *brevis*. When you retract them, the superficial radial nerve will be included with them. This is sensory only.

Find the radial artery and vein, which lie between the lateral group of muscles and *flexor carpi radialis*. Retract them laterally. You will now have exposed the anterolateral surface of the distal ⅔ of the radius.

Post-operatively, apply plaster only if a fracture threatens or has occurred. If so, apply a tubular forearm cast leaving the wrist and elbow free. The remaining bone will prevent angulation. Encourage use of the arm.

7.8 Osteomyelitis of the ulna

The ulna has a subcutaneous border throughout its whole length, so it is easy to expose.

Make an incision anywhere from the tip of the olecranon to the ulnar styloid. Use the most appropriate part of the incision (7-8D), not all of it. Cut straight down on to the shaft of the bone and elevate the periosteum. This will carry the muscular origins of the *flexor carpi ulnaris* anteriorly, and those of the *extensor carpi ulnaris* posteriorly.

Post-operatively, apply plaster only if a fracture threatens or has occurred. If so, apply a tubular forearm cast leaving the wrist and elbow free. The remaining bone will prevent angulation. Encourage use of the arm.

7.9 Osteomyelitis of the femur

If osteomyelitis is acute, you need only drill the upper or lower end of the femur, for which you will only need a limited incision. If osteomyelitis is chronic, it may have involved the entire shaft of the bone. By a lateral approach, you can expose it from its greater trochanter to its lateral condyle. Cut straight through the *vastus lateralis* down to the bone. The head and neck of the femur are more difficult to reach. If osteomyelitis has involved the neck, which is partly inside the capsule of the hip joint, it will have also involved the head and the hip joint. This will need draining. The anterior approach is easiest for drilling the femoral neck (7.18).

Osteomyelitis of the femur commonly involves the hip joint, and occasionally the knee, but seldom both. When a child's knee is involved, the distal femoral epiphysis may slip. If this happens, the shaft of the femur usually slips anteriorly in front of the distal epiphysis, unlike in injury in which it slips posteriorly. Prevent further slipping by applying skin traction up to the mid thigh. You may need to manipulate it under GA.

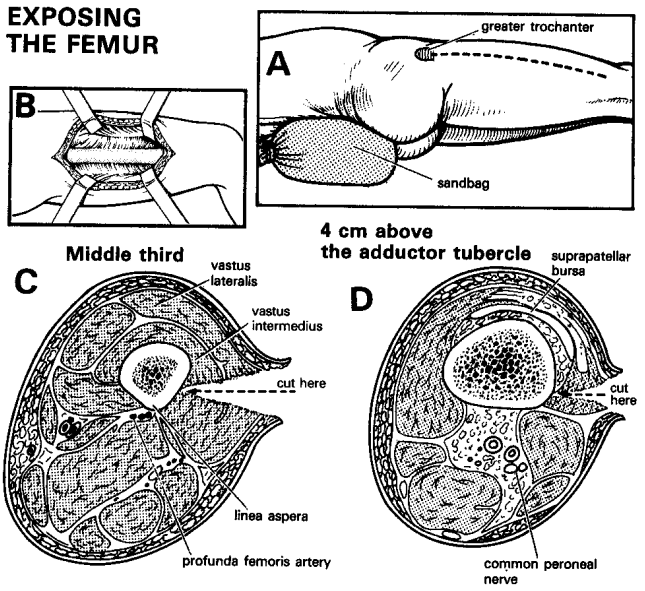


Fig. 7-9 EXPOSING THE FEMUR.
You can expose the femur by cutting straight down onto it along the lateral side of the thigh. A, prop up the buttock on a sandbag, and secure the patient on the operating table so he does not fall off! B, expose the middle ⅓ of the femur. C, cross-section of the middle of the thigh. D, cross-section about 4cm above the adductor tubercle.
Kindly contributed by John Stewart.

(a) Femoral shaft

Cross-match two units of blood; this can be a bloody operation, especially if you cut too far posteriorly. Use the supine position with a sandbag under the hip on the infected side. Use a tourniquet when you operate on the middle or distal thirds of the bone.

Cut along the relevant part of the incision (7-9A). This extends from just distal to the greater trochanter to just above the lateral femoral condyle. Cut through the skin, subcutaneous fat, and *fascia lata*. Then cut straight through the *vastus lateralis*, down to the lateral side of the shaft of the femur. There will be some bleeding, but much less than there would be if you cut posteriorly on to the *linea aspera*.

CAUTION!

(1) Take care to stay on the lateral surface of the femur.
(2) Avoid the *linea aspera*, and vessels near by.
(3) Remember that blood loss in a small child is proportionately more serious.

If there is bleeding from the vessels of the *linea aspera*, catch them with a haemostat, and transfix them with a ligature on a curved needle. Pass the needle round under the haemostat and the vessels at least twice. Pull the ligatures tight as you release the haemostat. They are usually too deep into the wound to tie on the tip of a haemostat. If you cannot reach a bleeding vessel, pack the wound tightly, raise the foot of the table and wait for the bleeding to stop.

If you are operating towards the distal end of the femur:
(1) *Don't enter the knee joint or the suprapatellar bursa*.
(2) Stay strictly on the lateral side of the knee.
(3) *Don't go posteriorly*: you may injure the lateral popliteal nerve.
(4) *Don't go medially* because you may injure the main vessels.

Post-operatively, apply skin traction. This will be easier than applying a medial plaster splint, which is the alternative. Later, use a hip spica or a plaster cylinder from the groin to the knee, add crutches, and encourage weight-bearing.

7.10 Osteomyelitis of the tibia

The tibia is one of the most common sites for osteomyelitis, which is fortunate, because it is one of the easier bones to approach. If the infection is early, decompress it through a short incision. If chronic infection exists, *don't operate before a firm involucrum has formed*, or you will leave a gap in the bone which will need extensive reconstructive surgery to repair. A gap is particularly likely in the tibia, because so much of it is subcutaneous.

DRILLING. Make a linear incision 1cm lateral to the anterior border of the patient's tibia (7-10).

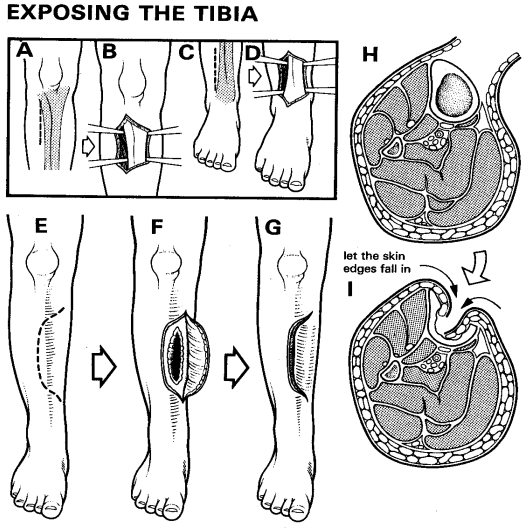


Fig.7-10 OSTEOMYELITIS OF THE TIBIA.
A-B, expose the upper end of a patient's tibia. Incise over muscle on the lateral side. C-D, expose the lower tibia; again incise over muscle on the lateral side. E-G, expose the shaft of the tibia. Incise mainly on the lateral side and reflect a flap medially. H-I, allow the edges of the flap to fall into the wound to close it post-operatively.

SEQUESTRECTOMY. Make the main part of the incision over the muscles rather than the bone. Make the longitudinal part of the incision 1cm lateral to its anterior border. Proximally, *don't extend it higher than the tibial tubercle*. If possible, avoid taking it across the tibia where this is infected, because the scar from the incision will stick to the bone and become painful later. If necessary, curve its upper and lower ends to cross the anterior border of the bone.

Reflect the skin with the periosteum. They will probably be so closely bound together that you will be unable to separate them. Hold the skin flap lightly with skin hooks. Incise the periosteum midway between the anterior and posteromedial borders of the bone.

If the position of sinus tracks is medial, you can make a medial flap in the same way, with most of the length of the incision over the muscle on the medial side of the tibia.

After you have removed the sequestrum:

(1) If the tissues are not too tight, close the wound lightly and insert a drain in its lower part.

(2) If the tissues are tight, let the skin edges fall into the wound and leave it unsutured (7-10H,I). Healing will take longer like this. Apply a posterior slab or a long leg cast with the ankle in neutral, and the knee in 20º of flexion. Mark a window in it while it is still soft, cut out the window with a knife, or with a plaster saw 2 days later when it is hard. Dress the wound through this window.

If you have left a deep trough in the front of the tibia which is slow to granulate and epithelialize, graft it.

CAUTION!

(1) *Don't go directly anteriorly through the subcutaneous surface of the tibia*.

(2) Make sure your assistant retracts the skin flaps gently, because they can easily necrose.

Apply a long leg cast with a walking heel, then encourage early weight-bearing with as normal a gait as possible.

DIFFICULTIES WITH OSTEOMYELITIS OF THE TIBIA

If there is a very large skin defect in the tibia which is slow to heal, consider making relieving incisions about 15cm long down the medial and lateral sides of the calf, and pushing the tissues forward to cover part of the gap. Hold them in place with sutures or strapping. Graft the gap made by the relieving incisions.

If a large part of the tibia has been destroyed, and inadequate involucrum has formed, try to get the fibula to hypertrophy. Apply a below-knee calliper. Later, an operation in which a length of the fibula is moved across to form a new tibia is needed. This is done in two steps, moving one end at a time. The transposed piece of the fibula can hypertrophy greatly.

If: (1) a sequestrum was removed before a firm involucrum had formed, or (2) the periosteum in the middle ⅓ of the shaft of the tibia is destroyed, use a Sarmiento cast, to support the leg and prevent the foot going into inversion until such a time as you can get a fibula transplantation done.

If a child's tibia is completely destroyed, the fibula may hypertrophy, and push the foot into *varus*; this needs expert correction.

7.11 Osteomyelitis of the fibula

Osteomyelitis of the fibula is uncommon. If the tibia is not involved, you can remove a sequestrum from the fibula as soon as is convenient, without waiting for an involucrum to form, because the tibia will support the leg. You can expose any part of the fibula by approaching it between the peroneal muscles anteriorly and the *soleus* posteriorly. The posterior tibial nerve and vessels are well out of harm's way; but *be careful not to injure the peroneal artery and veins which are close to the postero-medial angle of the shaft of the fibula.* If the head of the fibula is involved (rare), *be very careful not to injure the common peroneal nerve.*

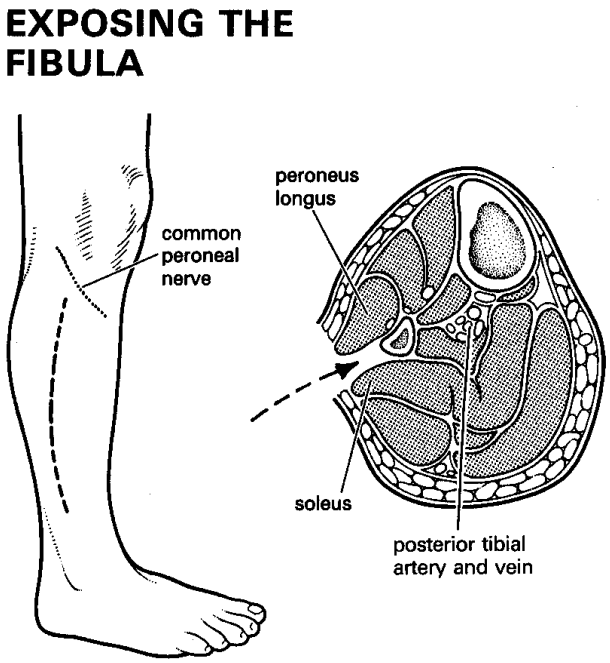


Fig. 7-11 OSTEOMYELITIS OF THE FIBULA.
Approach the fibula between the peroneal muscles anteriorly, and the *soleus* posteriorly.

INCISION. Use the lateral position with the affected leg uppermost, and the knee slightly flexed. Use the appropriate part of an incision which starts 5cm below the head of the fibula, and curves gently posteriorly down towards the lateral malleolus. Reflect short skin flaps anteriorly and posteriorly. Avoid the head and neck of the fibula, because the common peroneal nerve winds round it. If you have to remove sequestra from the head, try to pull them down from below.

If you are working on the middle ⅓ of the fibula, incise the periosteum vertically, and separate muscle from bone subperiosteally.

CAUTION! The peroneal vessels are close to the medial side of the fibula, *so strip the muscles carefully.*

EXCISION OF THE FIBULA (GRADE 2.5)
If necessary, and if the child is >10yrs, remove the entire shaft of the fibula, except for its lower 5cm. Use a Gigli saw, *not an osteotome, or bone-cutting forceps,* which will splinter it. *Be very careful to avoid the common peroneal nerve* winding round its upper end.

7.12 Osteomyelitis of the calcaneus and talus

The calcaneus is a completely cancellous bone which never forms an involucrum and seldom an isolated sequestrum. Pus soon perforates its periosteum without destroying much of its cortex. The most practical operation, and some would say the only one, is to remove the whole of the calcaneus to obtain an ugly but surprisingly useful foot.

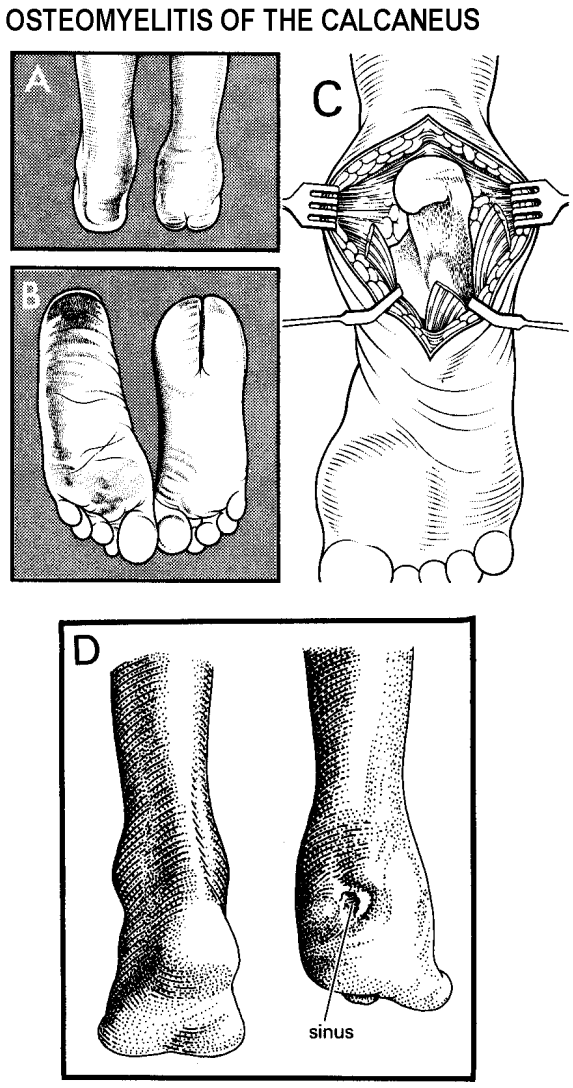


Fig. 7-12 OSTEOMYELITIS OF THE CALCANEUS.
Split the heel for the easiest approach to the calcaneus; this brings no disability. A-B, after the operation. C, expose the calcaneus. D, osteomyelitis of the right calcaneus with a sinus.
After Crenshaw AH, Campbell JW Operative Orthopaedics, CV Mosby, 5th ed 1971 Fig. 10-18 with kind permission.

If infection is limited to the pin track, opening up and scraping out the granulation tissue from around the pin track may occasionally be all that is needed.
You can approach the calcaneus from either side in order to drain a soft tissue abscess or to remove a window from the cortex during the acute stage of osteomyelitis.

(a) Calcaneus

Use the prone position with a support under the foot. Make a longitudinal incision exactly in the middle of the heel. Start it in the midline level with the base of the 5th metatarsal. Extend the incision proximally to split the distal end of the Achilles tendon for c.3cm.
Incise the plantar aponeurosis in a plane between the *flexor digitorum brevis* and *abductor digiti minimi.* Shell out the bone. You cannot remove it from inside its periosteum, so strip this away from the soft tissues of the heel and remove the bone completely, either as a single piece or in several smaller ones.
CAUTION! Start in the midline, stay close to bone and reflect everything you meet medially and laterally. In this way you will avoid important structures, especially the plantar nerves entering from the medial side of the foot.

POST-OPERATIVELY, allow the wound edges to collapse together, but don't suture them. Apply much gauze. Hold the ankle in a neutral position with a gutter plaster splint held with a crepe bandage. As the wound heals, start walking with crutches; later, progress to full weight-bearing. The edges of the scar will turn deeply inwards and split the heel into two cushions. If its surface is uneven, suggest wearing shoe pads.

(b) Talus

Presentation is with a painful ankle. Radiographs show an irregular dense talus. Sequestra are unusual. If you apply a below-knee cast and treat with an antibiotic for 3wks, the infection will probably settle without surgery, but degenerative arthritis may follow.

7.13 Osteitis of the cranium

Flat bones like those of the skull differ from long ones:
(1) They have little marrow between their diploë, so that when they are infected the condition is an osteitis, rather than an osteomyelitis.
(2) Unlike long bones, flat bones seldom sequestrate, and *don't form an involucrum*.
(*N.B.* Osteitis of a rib is usually due to TB.)

When sequestra do form in the skull, it is usually because a burn has destroyed the blood supply to the outer diploë.

Osteitis of the skull presents with headache, combined with tenderness and swelling over the lesion which may be particularly marked.

It may be secondary to:
(1) A deep burn.
(2) An open skull fracture.
(3) Frontal sinusitis (29.8).
(4) An extradural abscess (6.5).
(5) Septic thrombophlebitis of the scalp.
(6) Pyaemia causing metastatic lesions in the skull.

When you plan the incision, consider the arteries of the scalp, and incise between them. For example, don't make a transverse incision in the temple which will divide the temporal artery. *Split skin grafts will not take on bare skull*, but they will take on granulations. So, if necessary, remove dead bone, apply saline dressings for a few days, and wait for granulations to form.

CAUTION! If a sequestrum is firmly anchored, use an osteotome and light taps from a heavy hammer; *don't open the dura or injure the brain.*

If osteitis follows FRONTAL SINUSITIS (29.8), define the extent of the frontal sinus with radiographs. Shave the anterior 3cm of the scalp. Make a long incision above the hairline from ear to ear, and reflect the skin of the forehead downwards as a flap, based on the supraorbital vessels.

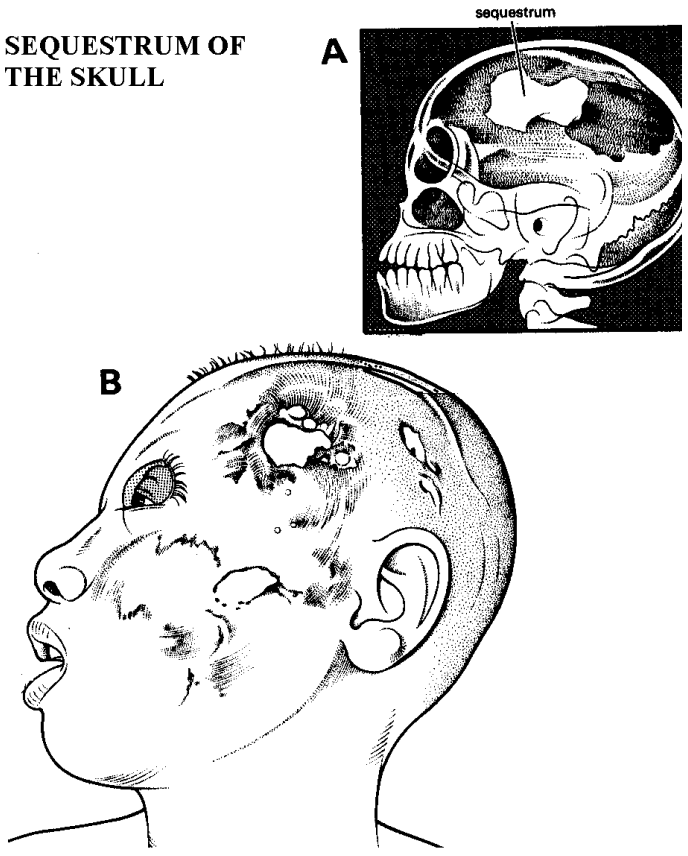


Fig. 7-13 SEQUESTRUM IN THE SKULL.
There is a dense white sequestrum in the skull, which has extended forwards. Burns of the scalp are however the commonest cause of necrosis of the skull. Another cause is septic thrombophlebitis of the scalp, which causes it to necrose and expose the bone underneath.
Kindly contributed by Gerald Hankins.

Remove the anterior wall of the frontal sinus; try to curette away all its lining, so that no more fluid will form. If possible, try to establish drainage through the nose. Insert drains through stab incisions above the outer end of each eyebrow. Lead them horizontally from the frontal region of the sinus through these incisions. Or, insert them below the inner eyebrows. Close the flap.

7.14 Osteomyelitis of the jaws

Osteomyelitis can affect either of the jaws, usually the lower one, and can be secondary to:
(1) An infected tooth socket in an adult, especially the mandible (6.9, 31.3). Suspect it if there is pain, swelling, tenderness, trismus, and fever after he has had an infected tooth removed (sometimes months before), or an alveolar abscess drained.
If the osteomyelitis becomes chronic, there may be sinuses over the lower face, or over the inferior border of the mandible (31-9). The offending teeth are usually loose, and you may see pus discharging around them.
(2) An open fracture, especially comminuted, of the lower jaw.
(3) Cancrum oris (31.5).
(4) Sickle cell disease.
(5) Actinomycosis (31.6).

(a) Acute osteomyelitis

If osteomyelitis is due to an infected tooth, extract the tooth (31.3).
If it is due to an open fracture or haematogenous, it is probably subacute and can be satisfactorily treated by antibiotics.

(b) Chronic osteomyelitis

RADIOGRAPHS. PA and oblique views may rarely show a sequestrum, or a patchy osteoporosis accompanied by new bone formation (dense thickened bone). No significant radiographic changes with multiple skin sinuses discharging 'sulphur granules' suggest actinomycosis (31.6).

TREATMENT. Treat with antibiotics (cloxacillin or chloramphenicol) for up to 2wks. Improve the oral hygiene. Remove any loose teeth. If a sequestrum is present, remove it. There is no need to wait for an involucrum to form unless the sequestrum is very large.

SEQUESTRECTOMY

(1) **Maxilla**. As the dead bone separates, it loosens. Wait for nutrition to improve. If the sequestrum is small and loose, remove it under sedation only. If it is larger, remove it under ketamine *in toto* or in pieces. If necessary, chip away a little living bone. Curette the residual defect. If the cavity bleeds, pack it for 5mins.

(2) **Mandible**. To avoid an unsightly scar, incise 1cm below the inferior border of the ramus of the mandible. Cut through healthy skin and subcutaneous tissue near the sequestrum. Avoid, or clamp and tie, the facial artery and vein, as they cross the ramus of the mandible 3cm (in an adult) anterior to its angle. Chisel away the outer bone covering the sequestrum and curette the cavity. Close the wound loosely, leaving a corrugated drain through one end, or through a separate stab wound.

CAUTION! Don't operate on a malnourished child until the general condition is acceptable.

7.15 Osteitis of the spine, pelvis and ribs

The spine can rarely be affected by suppurative osteitis: the patient is usually a very ill child with fever and severe back pain, usually in the lumbar region. There may be some inflammatory oedema over the spine, which is very tender, and may be arched backwards by muscle spasm, as if he had tetanus or meningitis.

Ultrasound or radiographs may show a paravertebral abscess, usually with normal bones. There may be paraplegia as the result of inflammatory oedema involving the cord. If there is to be any chance of survival, the pus must be drained by removing the transverse processes of some of the vertebrae and part of some of the ribs. If there are no spasms, recovery will probably occur in 3-6 months. But if there are extensor or, worse, flexor spasms, the paraplegia is likely to be permanent.

Osteitis may be chronic in an older child or adult. There is pain, but little or no fever, and no arching of the back.

Tuberculosis of the spine (32.4) is the commonest type of **spondylodiscitis** (infection of the disc space). This occurs more commonly in HIV disease. Ambulatory treatment with standard anti-tubercular therapy is effective if patients can walk: there is no advantage of an initial period of bed rest, application of a spinal POP jacket, or adding streptomycin to the regime. A costo-transversectomy (32.5) for the drainage of a cold abscess is only indicated when neurological signs ensue.

In sickle cell disease, *salmonella* is frequently the cause, and *staphylococcus* less so. The only early radiographic sign may be disc-space narrowing; treatment with IV antibiotics is necessary for 6wks, associated with spinal immobilization. Drainage is required if there is no response to antibiotics, neurological signs ensue, or there is an epidural abscess.

THE SPINE

If there is marked osteoporosis but minimal or no osteosclerosis, suspect tuberculosis (32.4).

If the bodies of the vertebrae are abnormal, but not the intervertebral discs, suspect malignancy.

If the disc and the adjoining bone are diseased, especially if this is maximal anteriorly, suspect infection. The diseased bone softens, and the vertebral bodies become wedge-shaped.

In a child, consider Burkitt's lymphoma (17.6).

THE PELVIS

Osteitis of the pubis may occasionally follow symphysiotomy (21.7).

If it involves the innominate bone, try antibiotics for up to 6wks. Sequestra are unusual.

THE RIBS

Osteitis of the ribs is rare, and almost always due to tuberculosis, usually only confirmed on rib resection.

7.16 Septic arthritis

An infected joint is another condition in which failure to drain pus early is a real disaster: severe chronic and probably painful disability results. If you don't drain the infected joint early, it will be destroyed and may ultimately ankylose. In a child, the epiphyses near it may displace or dislocate. As soon as you have made the diagnosis, drainage is urgent: *this is not an operation to leave until the next day!* If you allow pus to accumulate under pressure in the hip, it may impair the blood supply to the head of the femur within 8h, so that it necroses. Pus can also damage a joint, even if the blood supply is not impaired.

Bacteria can reach a joint:

(1) Before the age of 6 months, from osteomyelitis in the metaphyses of any long bone. After this age, the epiphyseal plates prevent such spread.

(2) At any age, in the hip, because the proximal metaphysis of the femur is partly within the capsule of the hip joint. This makes septic arthritis of the hip and osteomyelitis of the neck of the femur virtually the same disease. The hip may also be infected in a child as a result of femoral artery or vein puncture.

(3) Through the blood from a distant septic focus, or IV injection of drugs. This is haematogenous septic arthritis, which involves the knee, hip, shoulder, and ankle in this order of frequency. It is more frequent in HIV disease.

(4) From sexually acquired infections: gonococcal arthritis affects usually knees and ankles.

(5) Through a penetrating wound of a joint, especially of the fingers or knee, particularly after an animal bite, or previous surgery especially if a prosthesis has been inserted.

The first sign of septic arthritis is immobility. One of the joints, commonly the hip or knee, becomes so painful that moving it even a little in any direction causes great pain. Sometimes, several joints are involved at the same time. There is usually pyrexia. The combination of fever and a painful immobile limb is caused by either osteomyelitis or septic arthritis, until you have proved otherwise. Later, if the infected joint is near the surface, you will be able to feel that it is warm and swollen with fluid. Unfortunately, the shoulder and the hip are so deep that you cannot easily detect fluid, so that the only local sign is acutely painful limitation of movement.

Septic arthritis does not always run a typical course, and so is not often easy to diagnose. Here are some of the difficulties:
(1) In the very old or very young, there may be few general signs of infection, and the effusion may not even appear to be inflammatory.
(2) In the spine, the sacroiliac joints, and the hips, pain may be the only presenting symptom.
(3) The pus may be too thick to aspirate.
(4) Only 50% of patients have a fever or a leucocytosis, especially if HIV+ve.
(5) You can easily confuse tuberculous with subacute suppurative arthritis. To distinguish them, rely on the radiograph and your findings on aspiration (pus or caseous tissue). If you are still in doubt, treat for both diseases. Review the progress at 3 and 6wks, when suppurative arthritis should show much improvement, whereas it is still too early for tuberculosis to show much change.

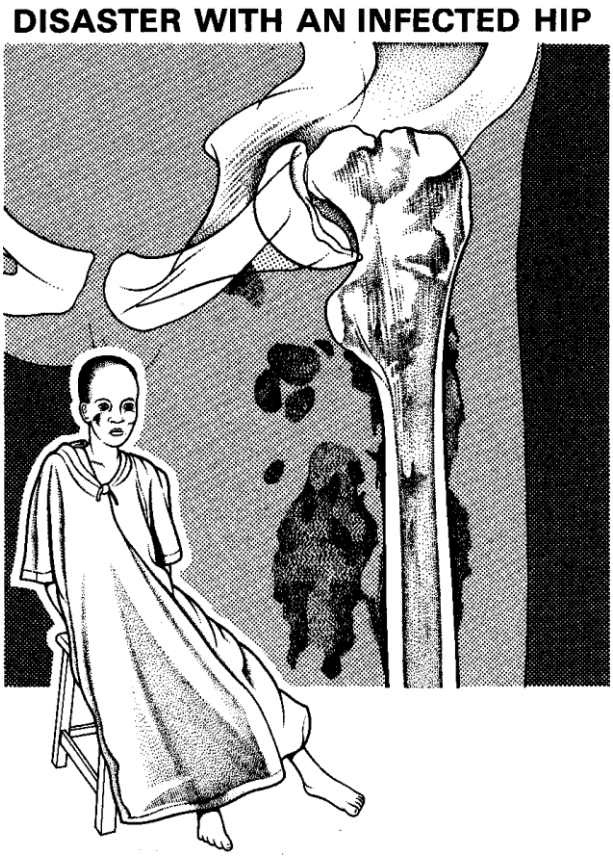


Fig. 7-14 DISASTER WITH AN INFECTED HIP.
Radiograph and classic position seated. Infection has displaced the epiphysis of the femur, and moved its shaft upwards. The infection in the thigh is producing gas.

The diagnosis is particularly difficult in babies:

AHMED (1yr) was brought by the mother saying he had fever and was drawing up the left hip in pain. This in itself was unusual, because, if a baby does this, he usually draws up both of them. He was found to have suppurative arthritis of the right hip, which was too painful to move. It was aspirated, antibiotics were started within 24h, and he recovered.
LESSON The diagnosis was made early and treatment started immediately.

Septic arthritis is more common in the disadvantaged and malnourished and also in infancy and old age. It is common in HIV disease, as well as diabetes mellitus, chronic renal failure and in joints previously damaged by trauma or inflammatory disease.

Staphylococcus aureus is the dominant organism, but if the patient has HIV or sickle cell disease, you may find *E. coli* or *salmonella* in the joint. *Haemophilus influenzae* is the most frequent organism in newborns, but is seldom seen in older patients. Other organisms include *streptococci*, *brucella* and *gonococci*. Congenital syphilis presents as swelling of both knees without much fever, in childhood. Actinomycosis and mycetoma (34.11) may also invade joints from outside.

Several things can happen to a severely damaged joint:
(1) It can dislocate.
(2) An epiphysis can slip, either immediately, or several weeks later (7-14).
(3) It can become fixed in a painless stable bony ankylosis in the position of function.
(4) It can develop a painful unstable fibrous ankylosis, which can be a serious disability.

HASINA (17yrs) was admitted with pain in her left hip and inability to walk for 3 days. She was given physiotherapy, nursed on a fracture bed for 3wks, and discharged on crutches. Some weeks later she was readmitted, pyrexial, and with a swelling of her right thigh extending from her knee to her iliac crest. 3L yellow-green pus were aspirated (7-14).
MARIAMU (12yrs) was admitted with osteomyelitis of her tibia. This was settling nicely when she developed pain in her left hip and became pyrexial. The radiographs of her hip were normal, septic arthritis was diagnosed, and she was given large doses of the latest broad-spectrum antibiotic. Her pain improved slowly but her fever continued. Later, radiographs showed destruction of the head of her femur. Traction was applied. Sinuses developed, and she was never able to walk again. Two years later her pain was so severe that she had to have her hip disarticulated. All this happened in a 'good' hospital.
LESSONS (1) The early diagnosis of septic arthritis of Hasina's hip was not made, although the history and signs were obvious. (2) Rest in bed on traction would have prevented her epiphysis slipping. At best she will have a painful hip, either for life, or until her hip has ankylosed spontaneously, or been fused surgically. (3) Explore a hip on the *suspicion* of septic arthritis.

ASPIRATE ALL SPONTANEOUS JOINT EFFUSIONS AND DRAIN ALL INFECTED JOINTS

ASPIRATION. Use pethidine IV; thoroughly sterilize the skin site you plan to use for the aspiration. Carefully choose the site of puncture and push a large (1.2mm) needle down into the joint (7-15). The critical investigation is to *aspirate the joint as soon as you suspect infection*. Frank pus in the syringe, *or even slightly cloudy synovial fluid*, confirms the diagnosis. You may get a false -ve, but apart from contaminants in the culture, you will never get a false +ve result. *Aspiration alone is not enough; it only tells you that pus is present:* you must thoroughly irrigate the joint till the effluent is clear. However, aim to aspirate as much of the pus as you can. Aspirating the more superficial joints is usually easy (7.17).

If you fail to aspirate a joint that you think is infected, you must incise and drain it, *i.e.* perform an arthrotomy. The results of not doing so are so serious that the dangers of attempting it are well worthwhile. Likewise, if the pus is too thick to aspirate properly, perform an arthrotomy to wash out the joint.

SPECIAL TESTS. Culture the synovial fluid (30% +ve result) and blood (14%). Screen for HIV.

RADIOGRAPHS. Signs are:
(1) Widening of the joint space.
(2) The signs of early osteitis (7.3). You may see the first signs of new bone formation as early as the 5th day in an infant, but it will not appear before the 10th day in an older child, and may take longer.

ANTIBIOTICS. Try to isolate the organism, otherwise cloxacillin or chloramphenicol are most suitable. Under 5yrs, salmonella is most common. In acute cases treat for 2-3wks; in chronic cases for up to 6wks. When infection is well established, antibiotics seldom help. Treated early, septic arthritis may recover fully. In Asia, if the infection is due to melioidosis, use ceftazidime IV.

If, when you drain an infected joint and wash out the pus, its joint surfaces are smooth, there is a good chance of having a normal or nearly normal joint. The prognosis is worse if cartilage has been lost, if the joint surfaces are rough, if the bone is soft, or if a radiograph shows severe joint destruction. There is still hope of a movable joint, especially in the young; a child's epiphysis may seem to be destroyed on a radiograph, and yet regenerate considerably.

EXPLORATION ARTHROTOMY (GRADE 2.1)
Open the infected joint. Use a tourniquet where possible, and if the hand is involved, watch out for the nerves. Irrigate the interior of the joint forcefully using a syringe and warm water. Do this until the fluid comes back clear. Feel the surfaces of the joint. Leave the wound open. The linear incision you have just made will become elliptical, and you will see the cartilage underneath. If the joint is superficial, it needs no drain. If it is deep, as in the hip and shoulder, insert a rubber drain.

If the joint surfaces feel smooth, the prognosis is good. After 10 days of rest, start gradual active movements.

If the joint surfaces feel rough but some cartilage still covers the bones, there may still be useful function in the joint.

If all its cartilage has been destroyed, the prognosis is bad. The best hope is a stable ankylosis in the position of function (7-16). If the hip or knee is involved, apply temporary skin traction.

If, later, there is a persistently painful joint with limited movement, an arthrodesis is indicated. Fusing a joint is difficult in a child, and is rarely necessary; if it is done too early, there will be growth problems, so delay this as long as possible.

7.17 Methods and positions for septic joints (except the hip)

Joints need to be in particular positions for particular purposes, so be sure to get it right. These positions seldom coincide with one another, and the position of function is absolutely critical.

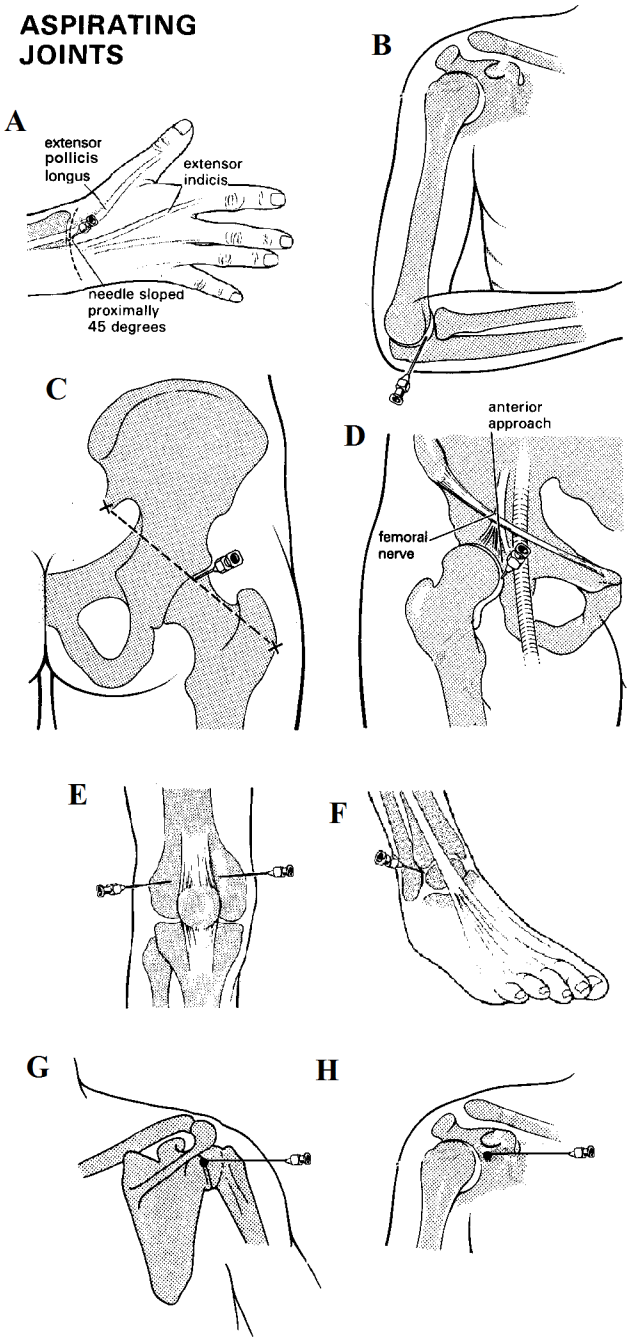


Fig. 7-15 ASPIRATING A JOINT may confirm the diagnosis, if pus is thin enough to come out of the needle. It is not effective treatment alone unless you wash the joint thoroughly.
A, wrist. B, elbow. C, posterior approach to the hip. D, anterior approach to the hip. E, knee. F, ankle. G, posterior, & H, anterior approaches to the shoulder (*right-sided joints shown*).
A-F, after Dudley, HAF (ed) Hamilton Bailey's Emergency Surgery. Wright 11th ed 1986, with kind permission. G kindly contributed by Jack Lange.

The position of function is the best position for a joint to be in if it is going to be fixed, or if its movement is going to be severely limited. It is also called the position for ankylosis.

The position of rest is the most comfortable position for a joint to lie. Put it into this position if it has to be rested for any reason, but is in no danger of ankylosing.

The neutral position of a joint is that from which its movement is measured. It is for anatomical description only.

The position of safety is for the hand only. It is the position in which the collateral ligaments of the finger joints are stretched, and in which fingers which are temporarily not going to be moved are least likely to become stiff.

Any kind of ankylosis, stable or unstable, is a dreadful disability if the joint becomes fixed in the wrong position, so make sure that, if it is going to ankylose, it does so in the most useful position. The position of function varies from joint to joint, and may depend on what the patient wants to do with it. You never know for sure when a joint is going to ankylose, so put it into the position of function for every case of septic arthritis. For example, splint the knee just short of full extension; splint the right (or dominant) elbow flexed. Make quite sure this position is maintained before discharge! *Don't leave this task to a physiotherapist in the hope that it will be achieved later!*

POSITIONS OF FUNCTION

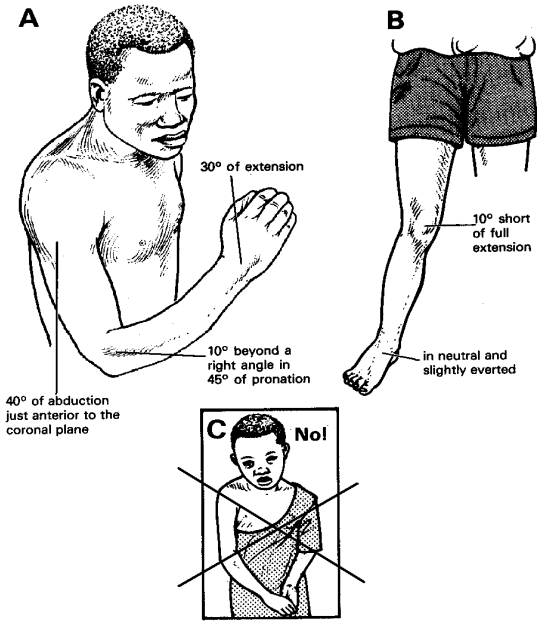


Fig. 7-16 POSITIONS OF FUNCTION.
If a joint is going to ankylose, the position in which it does so is critical. A, notice that the shoulder is abducted, the right elbow is flexed and in mid-pronation, the left elbow is extended (for toilet purposes) and B, the knee is just short of full extension, and the ankle is in neutral and slightly everted. C, this girl had an infected burn of her right elbow. The joint became infected. Tragically, it was allowed to ankylose in nearly full extension, so that she cannot eat with it or write! *Kindly contributed by John Stewart.*

THE POSITION OF A JOINT IS ALL IMPORTANT!

(a) The shoulder

ASPIRATION

Posterior route: sit the patient in a chair to face its back, ask him to touch the opposite shoulder with the arm that is to be aspirated, so as to adduct and internally rotate the shoulder. Feel for the head of the humerus. Keeping the needle horizontal, push it 30º medially into the joint space, from a point just under the postero-inferior border of the acromion (7-15G).

Anterior route: this is easier but more hazardous. Feel for the coracoid process just below the clavicle in the space between the *pectoralis major* and *deltoid* muscle. Push the needle into the joint slightly below and medial to the tip of the coracoid process. Slope it laterally 30º and push it backwards, until it enters the loose pouch under the lower part of the shoulder joint (7-15H).

EXPLORATION ARTHROTOMY (GRADE 2.4)

Approach the shoulder joint as if you were operating on the upper humerus for osteomyelitis (7-7), and separate the *deltoid* from the *pectoralis major* in the deltopectoral groove. Open the joint and irrigate with warm sterile water. Keep the wound open with a drain into the joint.

POSITION OF REST. Put the arm in a sling.

POSITION OF FUNCTION. Put the shoulder into a PoP spica in 45º of abduction, with the elbow just anterior to the coronal plane, in 70º of medial rotation so that the hand can reach the mouth.

(b) The elbow

ASPIRATION

Bend the elbow to 90º. Feel for the head of the radius, the olecranon and the lateral epicondyle of the humerus. Using these points of a triangle, push the needle through its centre into the posterolateral aspect of the joint.

EXPLORATION ARTHROTOMY (GRADE 2.4)

Make a 3cm longitudinal incision posteriorly in the sulcus between the olecranon and the head of the radius. Go through the skin and fascia, insert a haemostat, and open the joint. Irrigate it with warm sterile water. Keep the joint open with a drain.

CAUTION! Stay close to the olecranon, and remember that the posterior interosseous nerve winds round the neck of the radius 3cm distal to its head.

POSITION OF REST. Keep the arm in a sling in 90º of flexion.

POSITIONS OF FUNCTION depend on whether one or both joints are going to ankylose.

If the dominant elbow is going to ankylose, consider the patient's needs. For example, Muslims and many other peoples write and eat with their right hands and use their left hands for toilet purposes. If so, the right elbow should be more flexed than the left.

The dominant elbow will probably be most useful if it is flexed 10º beyond a right angle, with the forearm pronated 45º so that feeding, scratching the nose, and writing are possible. Put it into this position by fitting a collar and cuff.

If both the elbows are going to ankylose, arrange their positions so that the dominant arm can reach the mouth. Let the non-dominant elbow fuse in 10º short of full extension, so the hand can reach the anus.

(c) The wrist

ASPIRATION
Feel for the radial styloid; it will show you the line of the joint. Feel for the tendons of *extensor pollicis longus* on the radial side of the 'anatomical snuffbox'. Aspirate on its ulnar aspect, at the level of the wrist joint. Push the needle between *extensor pollicis longus* and the index tendon of *extensor digitorum* into the joint inclining it proximally 45º (7-15A).

EXPLORATION ARTHROTOMY (GRADE 2.4)
Flex and extend the wrist, as you feel for the exact line of the joint. Feel for the hollow between the tendons of *extensor pollicis longus* and the index tendon of *extensor digitorum*. Make a 3cm transverse incision, taking care not to cut the cutaneous branch of the radial nerve which runs in the web space of the thumb. Retract the skin edges and expose the joint through a longitudinal incision between the two tendons. Irrigate the joint with warm sterile water.

POSITIONS OF REST AND FUNCTION
Keep the wrist in 30º of extension with a volar plaster slab.

(d) The hand

THE POSITION OF SAFETY is peculiar to the hand and is the position which will minimize stiffness after an injury. Keep the mcp joints nearly fully flexed, the pip and dip joints fully extended. Keep the thumb well forward of the palm in opposition to the fingers, with its pulp about 4cm from them. To maintain this position use aluminium finger splints, plaster slabs, or a boxing glove dressing, as appropriate.

(e) The knee

ASPIRATION
Extend the knee. Push the needle into the suprapatellar pouch 2½cm above the upper border of the patella, from either the medial or the lateral side.

EXPLORATION ARTHROTOMY (GRADE 2.4)
With the knee extended, make a 5cm incision 2cm behind the medial edge of the patella and its tendon. Go through the *quadriceps* expansion, longitudinally, and put a curved haemostat into the suprapatellar pouch, under the surface of the patella. Put your finger into the joint and use it to remove the pus. Take a piece of joint capsule for biopsy. Irrigate the joint with warm sterile water. Leave the wound open, or sew up the upper part, and leave a corrugated drain in place. Dress the wound and apply skin traction, or a plaster backslab. Without one or the other, a painful flexion contracture is likely. Leave the drain in for 4-7 days.

POSITION OF REST
Apply skin traction to the lower leg to prevent flexion. Or apply a plaster backslab held on with a crepe bandage.

If there is already a flexion contracture following septic arthritis, put the knee in extension traction until it has been corrected. Then apply a cylindrical cast and encourage weight-bearing. With luck, a painless bony ankylosis will develop. If this does not happen, a compression arthrodesis of the knee will be necessary.

POSITION OF FUNCTION
Make sure the knee ankyloses in 10º of flexion, so the foot can just clear the ground on walking. Do the same when both knees are ankylosed.

(f) The ankle

ASPIRATION
Find the line of the joint by moving the ankle. Insert the needle into its anterior aspect just medial to the lateral malleolus. Push it backwards and slightly downwards, so that it enters the space in the angle between the tibia and the talus.

EXPLORATION ARTHROTOMY (GRADE 2.4)
Start the incision on the anterolateral aspect of the ankle, 5cm above the joint, and continue it downwards 1cm in front of the lateral malleolus to the base of the 4th metatarsal, lateral to the extensor tendons of the toes. Divide the superior and inferior extensor retinaculum as far as is necessary, so as to expose the capsule of the ankle joint. Then divide this and open the joint. (This incision will expose both the ankle and the tarsal joints.)

POSITION OF REST
Keep the ankle in neutral, without any flexion, extension, inversion, or eversion. Apply a plaster gutter splint.

POSITION OF FUNCTION
Keep the ankle neutral and slightly everted. Inversion will produce painful callus under the head of the 5th metatarsal on walking.

ANKYLOSIS IN THE WRONG POSITION IS A *REAL* DISASTER!

7.18 Septic arthritis of the hip

An acutely tender hip in varying degrees of flexion, together with fever, suggests infection. An important sign is spasm of the hip muscles. Test for this by rolling the thigh (7-17). If this is acutely painful, suspect that the hip is infected. **If there is septic arthritis or osteomyelitis**, tapping the greater trochanter lightly with your clenched fist will be painful.

If there is deep inguinal adenitis (6.16) **or pyomyositis** (7.1), it will not. In septic arthritis or osteomyelitis, the epiphysis of the femur may become indistinct, or even absent on a radiograph, but it often reappears. *This is not an indication for its removal!*

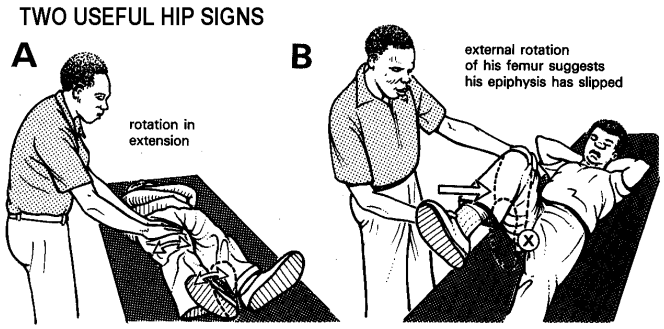


Fig. 7-17 SIGNS IN SEPTIC ARTHRITIS OF THE HIP.
A, lie the patient flat, place your hand on the thighs and try to roll the leg to and fro. A normal hip rolls easily; if it is infected, this will be acutely painful. B, if you flex a normal hip, it will flex without rotation. If it rotates externally into position 'X' as you flex it, the upper femoral epiphysis may have slipped. This can happen spontaneously in teenagers; it also happens in late septic arthritis.
Kindly contributed by John Stewart.

There are 3 operations you may need to perform, but only the 1st is common. Be prepared to:
(1) Drain pus in septic arthritis.
(2) Remove the head of the femur, when this has been destroyed as the result of infection.
(3) Perform Girdlestone's operation in chronic septic arthritis to remove the head and neck of the femur (7.19).
Sepsis may also follow after an arthroplasty or hemi-arthroplasty. Draining the pus in these cases is just as important; removing the prosthesis is difficult and may not be necessary: it is anyway something for an expert!

If you don't treat septic arthritis of the hip early, any of these things may happen:
(1) A flexion contracture may develop, which will be a great disability if you let it become permanent. Prevent and treat this in 2 ways.
(a) Apply extension (skin) traction to the lower leg. This is very effective prevention, so do it routinely.
(b) If a contracture has started to develop, extend the leg by using the prone position if this is tolerated. Few patients, especially children, will do this for long if their bed faces a wall! So make sure the bed faces the centre of the ward.
(2) The upper femoral epiphysis may slip off the shaft of the femur, and become a dead sequestrum in the hip joint (7-14). Later in the course of the disease there is a useful test to find out if it is slipping.
Bend the knee to 90º and then flex the hip (7-17B). If the leg turns to external rotation as you do this, the head of the femur may have slipped. Confirm this by taking a 'frog-leg view' radiograph. If a sequestrum has formed, open the hip joint and remove it.
(3) The hip joint may be destroyed. When this happens, there are 2 choices:
(a) Fuse the hip in the position of function by applying a spica for 3 months or more.
(b) Remove the remains of the partly destroyed head and neck of the femur by Girdlestone's operation (7.19).
This will result in a much more comfortable joint with some movement.
(4) The infection may extend into the acetabulum and involve the bones of the pelvis. When this has happened, there is little you can do, except drain the pus. The osteitis usually settles.

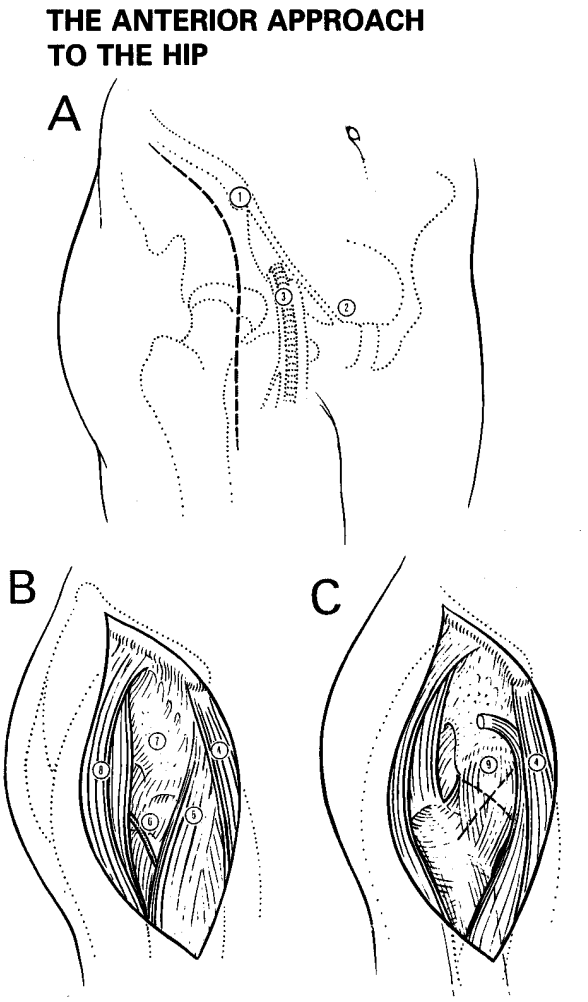


Fig. 7-18 ANTERIOR APPROACH TO THE HIP.
A, incision. B, retract the muscles. C, prepare to incise the capsule. (1) anterior superior iliac spine. (2) pubic tubercle. (3) femoral vein, artery and nerve from medial to lateral in this order. (4) *sartorius*. (5) *rectus femoris*. (6) ascending branch of the lateral circummflex vessels. (7) exposed surface of the ilium. (8) *gluteus medius* and *tensor fascia lata*. (9) incision in the capsule.

ASPIRATION
The hip lies immediately behind the mid-inguinal point. Use a thick lumbar puncture needle. If you can, do this under ultrasound guidance. If the anterior approach fails, try the posterior one.

Anteriorly, feel for the femoral artery 2.5cm below the inguinal ligament midway between the anterior iliac spine and the pubic tubercle. Insert the needle 1.5cm lateral to the artery (and thus lateral to the femoral nerve). If you cannot feel the femoral artery, insert the needle 2.5cm below and 2.5cm lateral to the mid-inguinal point.

Push the needle in, inclining it 15º medially and 15º superiorly. This will aim it at the joint directly behind the mid-inguinal point. Push it through the capsule into the joint. Aspirate. If you don't find pus, advance it into the cartilage. To prove that the needle is in the cartilage, rotate the thigh internally a little. This should move the adaptor of the needle medially. Withdraw it slightly to remove it from the cartilage, and aspirate. If necessary, alter its position and try again, if need be several times.

Posteriorly, use the prone position. Feel for the posterior inferior iliac spine and the centre of the greater trochanter. Insert your needle midway between these two points into the hip joint.

EXPLORATION ARTHROTOMY (GRADE 2.5)
Approach the hip anteriorly or posteriorly. If you can safely anaesthetize a prone patient, the posterior approach is easier because it allows better drainage, but the anterior approach is safer in children.

ANTERIOR APPROACH

POSITION. Use the supine position, but with a tilt to the opposite side by putting a sandbag under the affected hip.

INCISION. Cut from the mid-point of the iliac crest to the anterior-superior iliac spine. Extend the incision distally down the leg for 10-12cm. Divide the superficial and deep fascia. Use a periosteal elevator to separate the *gluteus medius* and *tensor fascia lata* from the iliac crest. Continue the dissection distally between the *tensor fascia lata* posterolaterally, and the *sartorius* and *rectus femoris* anteromedially. Divide the ascending branch of the lateral circumflex vessels between ligatures.

Insert 2 bone levers on each side round the upper shaft of the femur and retract the muscles. You will now see the thickened, oedematous, boggy capsule of the hip joint. Check that it is the joint by aspirating. Now open the joint with a cruciate incision. Take a biopsy. Ask a theatre assistant to grasp the patient's ankle and externally rotate the hip. You will see the head of the femur moving inside the acetabulum. If you want better access to the joint, insert levers round the neck of the femur. If you suspect osteomyelitis, drill at least 4 holes into the neck and upper shaft of the femur. Irrigate the joint with warm sterile water. Insert a suction drain from the joint to the surface, and leave it in for 5-7 days. *Don't suture the capsule*. Bring the muscles together lightly with a few '0' absorbable sutures. Close the fascia over the iliac crest. Close the skin with 2/0 monofilament.

POST-OPERATIVELY, apply 2-5kg of skin traction up to the mid thigh, with the leg in 1-15º of abduction and minimal flexion. Raise the foot of the bed.

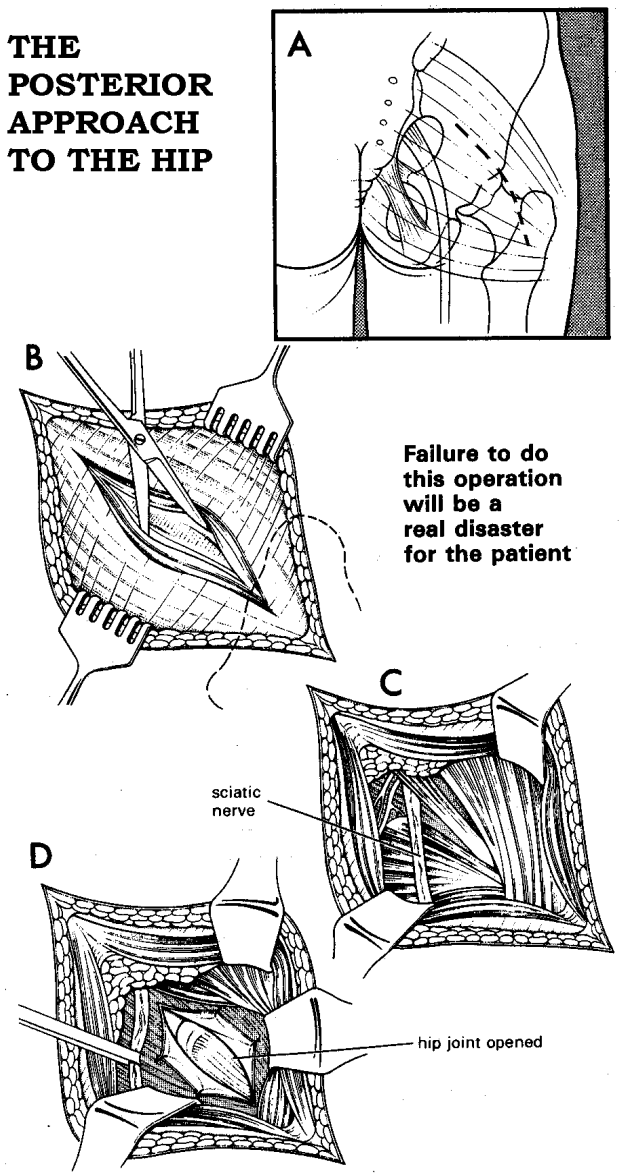


Fig. 7-19 POSTERIOR APPROACH TO THE HIP.
A, incise the *gluteus maximus*. B, separate its fibres. C, *be careful not to injure the sciatic nerve*. D, incise the hip joint.

POSTERIOR APPROACH

POSITION. Either use the prone position (needing GA and intubation) with a sandbag under the affected hip, or use the lateral position (needing GA alone or ketamine) with the affected hip uppermost (7-20).

INCISION. Find the tip of the great trochanter. Cut from its anterior margin obliquely up towards a point on the iliac crest 6cm in front of the posterior superior iliac spine, and down vertically for 5cm. Cut through the skin and superficial fascia. Separate the fibres of the *gluteus maximus* using your index finger and the end of a curved haemostat, until you meet the capsule of the hip joint. Open the incision with retractors.

LATERAL POSITION FOR POSTERIOR APPROACH TO THE HIP

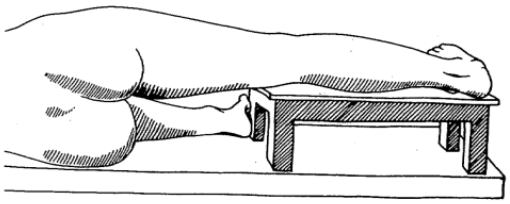


Fig. 7-20 LATERAL POSITION FOR POSTERIOR APPROACH TO THE HIP.
The affected limb rests horizontally upon a simple platform. The unaffected limb either lies beneath the platform, or, preferably, is flexed at the hip and knee in order to stabilize the pelvis. Place a pillow on the table to protect the leg from its hard edges!
After Crawford Adams J, Standard Ortthopaedic Operations. Churchill Livingstone 1976 p.25, Fig 10.

If you find pus in the muscles of the buttock, before you reach the hip, this is pyomyositis. If you go further and open a normal hip through an abscess in the muscles, you will probably infect it, *so stop!*
Ask an assistant to take hold of the ankle and rotate the hip internally, so as to increase the space between the trochanter and the acetabulum. Make a small incision in the distended capsule of the hip joint and widen it with a haemostat. Irrigate the joint with warm sterile water.
CAUTION!
(1) The sciatic nerve leaves the buttock halfway between the greater trochanter and the ischial tuberosity. There is no need to incise or dissect this far medially.
(2) The capsule of the hip joint is only 2cm wide between the posterior aspect of the greater trochanter and the posterior margin of the acetabulum.

If the upper femoral epiphysis has slipped and is forming a sequestrum, remove it. You will need to modify the incision by carrying it down to the greater trochanter. Remove the loose head of the femur with large bone forceps, and as much of its neck as is necrotic.

POST-OPERATIVELY, reduce the tendency of the hip to slide proximally by using traction for 6wks, while fibrous tissue forms to limit movement. The result will be an unstable hip, needing a crutch for support, but it should be pain-free and walking will be possible.

POSITION OF REST FOR THE HIP
If you are sure that the painful hip is only temporary, rest it in moderate flexion and 15º of abduction. In this position the legs are comfortably spread apart. Hold the hip in this position with skin traction. To produce abduction, bring the cord holding the weight to the end of the bar at the foot of the bed. If necessary, make sure it stays there by moulding a plaster pulley on the bar. Or, have a detachable bar with notches at suitable places, which you can tie to the foot of the bed. Or, put both the legs into abduction.

POSITION OF FUNCTION
Put the hip in a minimum amount of flexion, preferably none, 5º of abduction, and no rotation. *However, don't apply a spica with the hip in the position of function*, especially in a child. Otherwise, when you remove it, that spasm has rotated the pelvis anteriorly, and there is too much flexion. Instead, immobilize the hip in a spica in complete extension and 15º of abduction. When you remove the spica, you will find that it has gone into 15º of flexion, which is where you want it to be.

RELIEF OF SPASM. In a child, use diazepam, and apply up to $^1/_7{}^{th}$ of the body weight of extension (skin) traction. This will relieve the spasm of the muscles, and will prevent development of a flexion contracture.

7.19 Girdlestone's operation
(Hip excision arthroplasty)

Most procedures for infected hips are needed by children. An excision arthroplasty may help an adult whose hip has been partly destroyed by infection, avascular necrosis or a painful non-united femoral neck fracture. Walking will be less painful if what is left of the head and neck of the femur is excised, so as to allow the upper end of the femur to bear on scar tissue on the underside of the ilium. A false joint will develop, the leg will be short and a stick and a shoe-raise will be necessary but there will probably be very little pain.

Girdlestone's operation is a salvage procedure to relieve pain when an arthrodesis or, exceptionally, a prosthesis is impractical. It is inelegant and old-fashioned, and is not as good as an arthrodesis or a hip prosthesis, *but better than nothing.*

INDICATIONS
Walking painfully as the result of:
(1) Previous septic or tuberculous arthritis, which is now inactive.
(2) Aseptic necrosis of the head of the femur.
(3) A longstanding non-united fracture of the femoral neck.
(4) An infected hip prosthesis.
(5) A joint extensively damaged by a gunshot wound.

PREPARATION. Cross-match 2 units of blood. Use the lateral position with the affected hip uppermost (7-20).

INCISION (GRADE 3.5)
Reach the hip joint by the posterior approach (7.18) extending the incision down the leg for 10cm. Retract the sciatic nerve well medially. Incise the capsule of the patient's hip joint widely to expose the head and neck of the femur and the remainder of the greater trochanter.

You will find that removing the head is easier if you excise part of the upper rim of the acetabulum. Dislocate the hip by lateral or medial rotation of the femur. Curette all necrotic and infected bone from inside. Cut the femoral neck right down to its base from greater to lesser trochanter, and smooth it by chiselling away all sharp edges (7-21).

EXCISION ARTHROPLASTY OF HIP (GIRDLESTONE OPERATION)

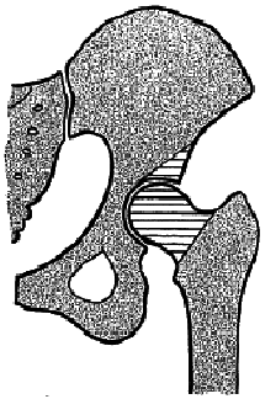


Fig. 7-21 EXCISION ARTHROPLASTY OF HIP (GIRDLESTONE OPERATION).
Remove this amount of bone *(shaded)*.
After Crawford Adams J, Standard Ortthopaedic Operations. Churchill Livingstone 1976 p.234, Fig 161.

To help reduce lateral rotation deformity, you can detach the *psoas* tendon from its insertion on the lesser trochanter and bring it round the front of the femoral shaft. Suture it to the tissues on the posterolateral side of the femur, so that it will act as a *medial* rather than lateral rotator of the thigh.

Fill the space created by removal of bone by a wad of *gluteus medius* muscle and sew back the edges of the incised *gluteus maximus*. If there is little active infection, insert a suction drain. If you find the bone seriously infected, leave the wound partly open with 1-2 rubber drains.

POST-OPERATIVELY, to prevent shortening, apply 3-10kg of skeletal traction using a pin through the tibia with the hip in 20-30º of flexion for 4-6wks. This will not be necessary if the hip is already fibrotic. In the elderly, if you are worried about a prolonged period in bed (because of pneumonia, bedsores etc), encourage walking with a stick after this time.

CAUTION! Try to prevent proximal displacement of the femur. This will prematurely seal off the area and defeat the purpose of the operation, which is to allow free drainage when there is active infection.

DIFFICULTIES WITH EXCISION HIP ARTHROPLASTY

If the head of the femur is not necrotic, or the hip is ankylosed, *don't proceed with the operation!*

If there is a prosthesis or cement *in situ*, remove these with a hammer, chisel and osteotome: this may present formidable difficulty, but you must get all the infected cement out.

8 Pus in the hands & feet

8.1 The infected hand

A badly infected hand can be a real disaster. Some infections arise spontaneously, others follow quite minor injuries, or even a seemingly trivial scratch. They are particularly common in diabetics, HIV disease and leprosy (32.18). The best prevention is an early and thorough toilet of all hand wounds, which is quite a minor procedure. The great danger of late or inadequate treatment is a stiff finger, which is a great disability and may ultimately need amputation (35.4).

Antibiotics may be effective in an early case, and may prevent a serious lesion spreading. *A careful wound toilet and early drainage is much more important.*

There are many spaces in the hand where pus can collect; each type of abscess has its own signs and incisions. *These spaces are not rigidly defined;* some run into one another, and more than one may be infected at the same time (8-1), so don't be dismayed by their apparent complexity. The common places for pus to collect are in the pulp spaces of the fingers (8.5), and in the web spaces (8.7). Even after pus has formed, recovery should be complete if treatment is correct, provided that tendon sheaths are not involved.

PUS IN THE HAND IS COMMON AND SERIOUS!

One difficulty is knowing when to incise an infected hand. Pus is so tightly trapped in the spaces of the hand that you cannot use fluctuation as a sign that it is present. A good rule to remember is that, if pain in the hand prevented sleep the previous night, or there is a suspicion of a foreign body, it needs incising.

When you operate:
(1) *Don't cut the digital nerves*: remember that they run on the radial and ulnar aspects of the fingers just anterior to the tips of the finger creases (8-6D).
(2) *Don't cut through a superficial abscess into the flexor sheaths underneath*, or you may infect them. These are in the greatest danger where they are nearest to the surface, under the flexor creases of the fingers. So *don't incise the palmar surface of a finger proximal to its distal flexion crease*, unless you are deliberately draining an infected tendon sheath.
(3) When you drain pus, be sure to remove the granulation tissue that surrounds it, so that the wall of the abscess is clean.
(4) Use a bloodless field whenever you can, so that you can see the anatomy clearly.

DON'T WAIT FOR FLUCTUATION
INCISE THE HAND USING A TOURNIQUET

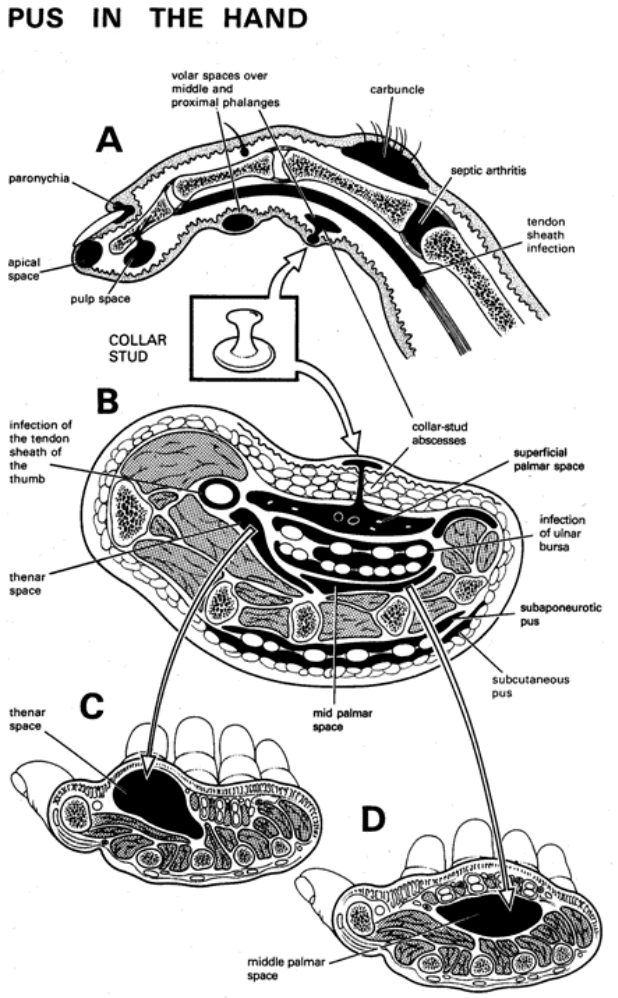


Fig. 8-1 THE MANY PLACES WHERE PUS CAN COLLECT IN THE HAND.
After Rintoul RF (ed) Farquharson's Textbook of Operative Surgery, Churchill Livingstone 7th ed 1986 Fig. 302; Milford, L, Hand Infections, in Edmondsen AS, Crenshaw AH (ed), Campbell's Operative Orthopaedics, CV Mosby Fig. 3-357. Both with kind permission.

WHERE IS THE PUS?
Feel carefully for the point of greatest tenderness by probing with a matchstick.

If the whole hand is swollen, the pus is probably in the mid-palmar space, or in a flexor tendon sheath, especially if movement of the little and ring fingers is impossible.

If the greatest swelling is over the web of the thumb, there is probably pus in the thenar space, especially if the index finger is held flexed, and its movement or the thumb's is impossible.

If: (1) the whole finger is swollen and tender,
(2) there is no obvious sign of the pus pointing, and
(3) any movement of the finger is exquisitely painful, there is probably a tendon sheath infection.

If all the fingers, especially the 5th, are held semi-flexed and rigid, suspect that the tendon sheaths in the ulnar bursa are infected.

Check if the terminal phalanx is infected (8.5).

If there is lymphangitis, lymphadenitis, or fever, the infection is spreading. If pus is present, incise the hand under antibiotic cover, and continue after the temperature and pulse have become normal.

CAUTION! Pus is much more likely to be present on the palmar surface than on the dorsum, so *don't be misled by swelling on the back of the hand*. The commonest cause of a swollen dorsum is a web space infection.

SPECIAL TESTS. Screen for diabetes and HIV.

TREATMENT

RAISE THE HAND for comfort and to promote healing. In less severe infections, raise the arm in a St. John's sling. In more severe cases, such as a tendon sheath infection, *make sure you rinse the tendon sheaths* (8.12), and admit the patient and raise the hand in a roller towel.

ANTIBIOTICS are necessary if the infection is spreading: treat with metronidazole and cloxacillin or chloramphenicol. *Don't forget to add an analgesic.*

INDICATIONS FOR INCISION

Don't try to treat an infected hand by aspiration only. Base your decision to incise on:

(1) The presence of acute local tenderness: this shows that pus is present and where it is pointing.

(2) The length of the history, particularly if symptoms are worse after 48h.

(3) The severity of the swelling.

(4) The nature of the pain, especially if throbbing pain prevents sleep.

ANAESTHESIA must be adequate. For any but the most minor infection, avoid LA close to the infection, because this will only spread it and increase the swelling.

If the infection is in the distal ⅔ of the finger or thumb, you should use a distal palmar block *without* adrenaline. For all other hand infections, use an axillary block, or an IV forearm block, or ketamine, or GA.

A TOURNIQUET is essential in all but the most superficial infections, because a bloodless field makes the operation easier (3.4). *Don't exsanguinate the arm with an Esmarch bandage*, because it may spread the infection.

If the pus is in the distal segment, wrap a rubber catheter twice round the base of the finger or thumb, and clamp it with a heavy haemostat.

If the pus is anywhere else, apply a pneumatic tourniquet (3.4).

INCISING, DESLOUGHING AND DRAINING THE HAND (GRADE 1.3)

Clean the skin with antiseptic. Incise where pus points, and take care you don't cut the digital nerves or spread superficial pus deeply (8-6). When you extend an incision, do so in a skin crease. If necessary, jump from one crease to another by making a Z-shaped incision. Remove skin that is already dead. If necessary, extend an incision to explore the whole abscess cavity, and remove deeper dead tissues.

If more than one space is infected, adapt your incision(s) accordingly. For example, if the mid-palmar space, several web spaces and the tendon sheaths are infected, you may need to make several incisions (8-5). As soon as you are through the skin, insert a haemostat, open it, and explore the abscess cavity (Hilton's method). Culture the pus.

If there are no vulnerable structures such as periosteum, nerves or tendon sheaths nearby, scrape away the lining of the abscess with curette or a swab. If there are vulnerable structures nearby, be more cautious, and only use a swab.

Drain the abscess by putting a piece of rubber glove into it. Or, leave a piece of petroleum jelly gauze between the wound edges.

CAUTION!

(1) *Don't cut the nerves* (8-6).

The digital nerves run near the anterolateral margins of the fingers. So cut either near the middle of the palmar surfaces of the fingers, or on their lateral surfaces fairly posteriorly at the apex of the finger creases. The muscular branch of the median nerve comes off the main trunk just distal to the tuberosity of the scaphoid and curves round into the thenar muscles.

(2) *Don't pack the wound tightly*.

CONTROL BLEEDING after removing the tourniquet by raising the arm and pressing firmly on the wound for 5mins without interruption.

POST-OPERATIVELY, be sure to elevate the hand, until pain and swelling subside: this is an important way of reducing stiffness. Rapid resolution of inflammatory oedema is more important than early movement in reducing stiffness. Wrap the wound with plenty of gauze, and use the dressings to splint it in the position of safety. Inspect the wound daily and wash with water bd.

If the infection was extensive, check for residual infection or necrotic tissue which may need further debridement.

CAUTION! Start active movements as soon as pain has subsided.

RAISE AN INFECTED HAND
STIFF FINGERS RESULT IN POOR FUNCTION

8.2 Subcutaneous hand infection

The skin and subcutaneous tissue can be infected anywhere in the hand. Pulp infections and paronychia are merely subcutaneous infections at the tip of a finger, the latter at the nail border. If there is pus under the keratinized layers of the epidermis, strip these off, and see if you can find the hole through which it has tracked from a deeper abscess underneath. An abscess near the surface may communicate with pus deep inside the hand through a narrow opening, forming a 'collar-stud abscess' (8-1). So, whenever you find a superficial abscess, look for the passage which might be joining it to a deeper abscess.

Carbuncles (6.4) may form in the hair follicles on the back of the fingers and hand. *Antibiotics will not cure this*, so desloughing is necessary.

DON'T BE MISLED BY A COLLAR-STUD ABSCESS

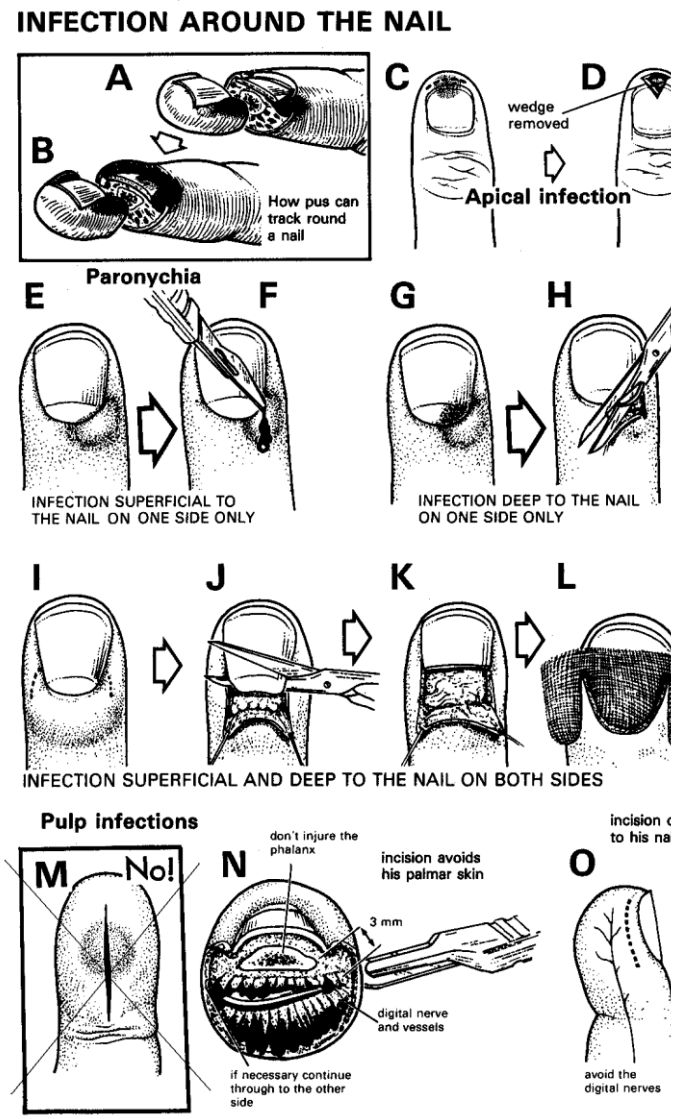


Fig. 8-2 INFECTION AROUND THE NAIL.
There can be pus apically (C-D), on the side of the nail, either superficial (E-F), or deep (G-H). It may track all round the nail (A-B) so that the proximal part of the nail needs excising (I-L). ***Avoid incising the ball of the finger*** **(M) unless pus is already pointing there. N-O, if infection is already present in several of the compartments of the pulp, make a lateral incision. Keep your incision away from the palmar skin, and** ***not more than 3mm from the edge of the nail.***
N.B. In N, don't cut the end of the finger off! **This is a schematic cross-section only!**
A,B, after Flatt AB, Functional Anatomy, Fig. 14.2 with kind permission.

8.3 Apical finger space infection

The apical space lies between the distal part of the nail and the bone of the distal phalanx. It may be infected when a splinter digs under the nail. The finger is painful, but there is little swelling. Tenderness is greatest at or just under the free edge of the nail. Cut a small 'V' out of the edge of the nail over the point of greatest tenderness (8-2C,D).

Remove the full thickness of the skin as a small wedge, and drain the pus.

8.4 Paronychia

Paronychia is an infection beside or proximal to the nail. Pus may track round it (8-2A-B), either superficial to the nail (8-2E-F), or deep to it (8-2G-H). Early antibiotic treatment may abort the infection, but you usually have to drain pus.

If the pus is superficial to the nail on one side only, incise it by angling the knife away from the nail to avoid cutting the nail bed (8-2E-F). If there is only a little swelling, you may be able to drain the pus simply by lifting the skin over the nail with a blunt needle: this doesn't even need LA.

If the pus lies under one corner of the nail, reflect a little flap and remove that corner only (8-2G-H).

If pus has tracked to the other side of the finger under the nail, make a second incision there, retract the flap, excise the proximal ⅓ of the nail, pack the wound open and drain it (8-2I-L).

If the infection fails to resolve, or the nail becomes indurated and red, suspect a fungal infection, and examine scrapings microscopically. If you find fungi, remove the nail and apply wet dressings, or a topical antifungal agent, such as gentian violet.

8.5 Finger pulp space infection

This is the commonest hand infection; pus more often gathers in the finger tips than anywhere else in the hand. The pulp of a finger is divided into many small fatty compartments by strands of fibrous tissue which run from the skin to the periosteum of the terminal phalanx. A sheet of fibrous tissue runs from the distal flexor crease to the periosteum, and so separates the pulp space from the rest of the finger. There is little room for swelling, so that infection causes a throbbing pain early. Pus from the pulp can track through to the skin outside, or through the periosteum, causing osteomyelitis of the distal phalanx. Its epiphysis is supplied by a separate artery, so this usually survives the infection. Tenderness is maximal over the ball of the finger tip.

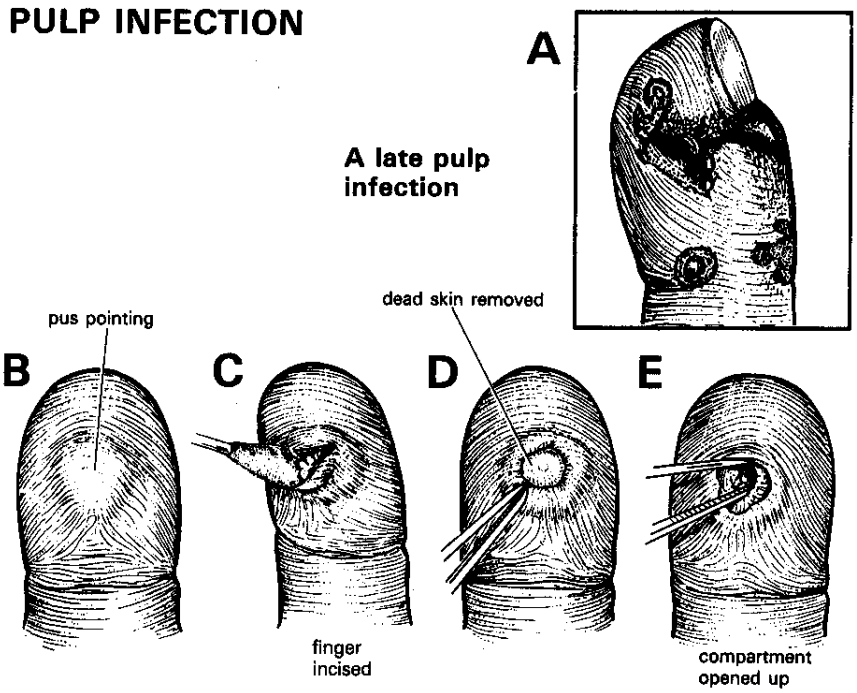


Fig. 8-3 PULP INFECTIONS. A, neglected pulp infection: much of the finger tip is already destroyed, and pus is starting to discharge spontaneously. B, if pus is already pointing, make a cross-shaped incision. C, swab away the pus. D, remove any dead skin. E, open up the infected pulp compartment. If it is not pointing and several spaces are infected, open up the finger tip from the side (8-2N).

If the abscess is in the distal pulp, and is already pointing to its centre, drain it by making a cross-shaped incision, or by removing a small circular or elliptical segment of skin over the abscess (8-3B-E). The incision will heal to leave a small punctate scar.

If the abscess is deep, is not pointing, and appears to extend into several compartments, make a J-shaped lateral longitudinal incision close to the bone, ≤3mm in a palmar direction from the free edge of the nail. Keep your knife away from the palmar skin (8-2N-O), and avoid the tip of the finger. Remove pus and slough, and lightly pack the wound with gauze. *Don't suture the incision*. Change the dressing after 2 days.

If the infection has been neglected, so that the whole terminal segment of the finger is swollen, continue the incision over the end of the finger and round to the other side. Divide the vertical septa and let the wound gape open. Dress it as above.

CAUTION!
(1) *Don't incise the tips of the fingers, or the palmar surfaces of the distal phalanges*, unless pus is already pointing there, because pressure on the scar may be painful.
(2) Any incision, other than those described, is likely to be a painful nuisance later, especially if you carry it towards the palmar surface.
(3) *Don't damage the periosteum*.
(4) Check for a collar-stud abscess (easy to see if you have used a tourniquet to provide a bloodless field).

DIFFICULTIES WITH FINGER PULP SPACE INFECTION
If the infected finger continues to discharge for >2wks, suspect osteomyelitis (8.16) or the presence of a foreign body. Obtain a radiograph. When this shows a foreign body, or a sequestrum that has separated, remove it.
In a child, the distal phalanx will regenerate under its periosteum. In an adult, the result will be an ugly curved nail and a short terminal phalanx.

8.6 Infection on the volar surface of the middle or proximal phalanx

Pus sometimes collects on the volar surfaces of the fingers, superficial to the tendon sheaths (8-1A). The spaces where it forms are separated from one another by the fibrous septa which run dorsally from the flexor creases of the fingers. The proximal space in each finger communicates with the web spaces in the palm. Pus may collect under the epidermis or under the deep fascia, and is less likely to remain localized than in a terminal phalanx.

The swollen, tender, indurated finger remains semi-flexed. Trying to straighten it is acutely painful. Explore the finger and rinse the infected space abundantly with sterile water, using a cannula. Drain pus from a volar space through a transverse incision over the point of greatest tenderness.

If the tendon sheath is infected, rinse it out thoroughly by making incisions c.2-3cm apart (8.12). *Take great care not to cut into the tendon underneath or to damage the digital vessels or nerves* (8-6G). Use a tourniquet to provide a bloodless field.

EXPLORE A TENDON SHEATH BUT DO NOT OPEN A JOINT UNLESS IT IS INFECTED

8.7 Web space infection

Three spaces, filled with loose fat, lie between the bases of the fingers in the distal part of the palm. They lie just proximal to the deep transverse ligaments, near the mcp joints. Pus more often gathers here than anywhere else in the hand, except in the pulp spaces of the finger tips. It gathers mostly near the palmar surface, but it may track:
(1) posteriorly towards the dorsum,
(2) along a lumbrical canal into the mid-palmar space,
(3) across the front of a finger into a neighbouring web space, or
(4) distally into the finger.
Pain and swelling may be so great that presentation is before much pus has formed. The back of the hand is swollen (8-5D).

If infection is severe, the fingers on either side of the web separate: a very useful sign. The point of maximum tenderness is on the palmar surface of the web, and may extend a short way into the palm. Although you may suspect a web space infection, it is usually difficult to exclude an infected tendon sheath.

Make a V-shaped incision between the fingers (8-5).
If pus is pointing into the palm, pass a probe proximally from the incision you have just made in the web space up into the palm. Its tip should underlie the place where the pus is pointing. Make a 2nd incision there. Scrape the walls of the abscess cavity free from granulation tissue. If necessary, divide some strands of the palmar fascia.

8.8 Superficial palmar space infection

When pus collects in the superficial palmar spaces of the hand, it does so under the palmar fascia. Sometimes, it tracks superficially and forms a collar-stud abscess under the superficial layers of the epidermis (8-1B).

If you can see pus under the epidermis, remove it and look for a track leading deeper into the hand.

If you cannot see any pus, make a small transverse incision over the point of maximum tenderness, in the line of the nearest skin crease. Probe the abscess cavity. If you find an opening leading to a deeper collection of pus, enlarge it. Scrape infected granulations from the wall of the cavity.

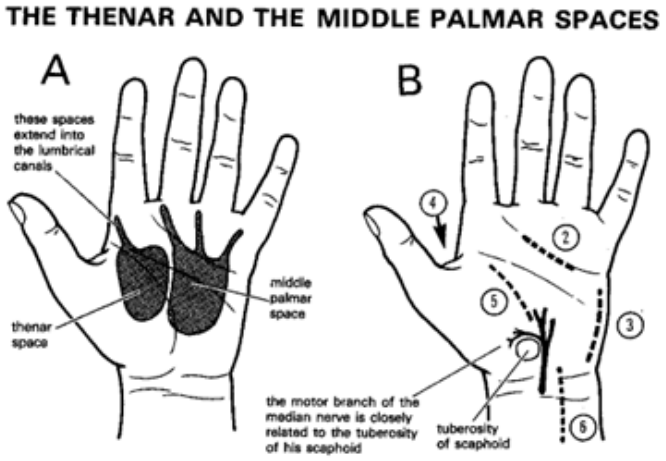

THE THENAR AND THE MIDDLE PALMAR SPACES
A
these spaces extend into the lumbrical canals
middle palmar space
thenar space
B
the motor branch of the median nerve is closely related to the tuberosity of his scaphoid
tuberosity of scaphoid

Fig. 8-4 THE THENAR (*RADIAL*) AND THE MID-PALMAR SPACES lie deep to the flexor tendons, between them and the fascia covering the metacarpals and *interossei*. They communicate with the lumbrical canals. Incise the mid-palmar space in the middle ⅓ of the distal (or proximal) palmar crease (incision 2), or along the ulnar border of the hand (incision 3). Incise the thenar space in the web between the thumb and the index finger (incision 4), or along the thenar crease in the palm (incision 5). *Beware of the motor branch of the median nerve!*

8.9 Mid-palmar space infection

This is the most important space in the hand, and is frequently infected in leprosy patients (32.18). It lies deep to the flexor tendons and lumbricals, and between them and the fascia covering the *interossei* and metacarpals. It is separated from the thenar space by a fibrous septum which extends from the middle metacarpal towards the palmar fascia. Infection reaches this space from a lumbrical canal, or from an infected tendon sheath.

The hand is so grossly swollen that it looks like a blown-up rubber glove. The normal hollow of the palm is obliterated, and the dorsum of the hand is swollen. Movement of the middle or ring fingers is impossible. The *interossei* are surrounded by pus and paralyzed, so that holding a piece of paper between the extended fingers is impossible.

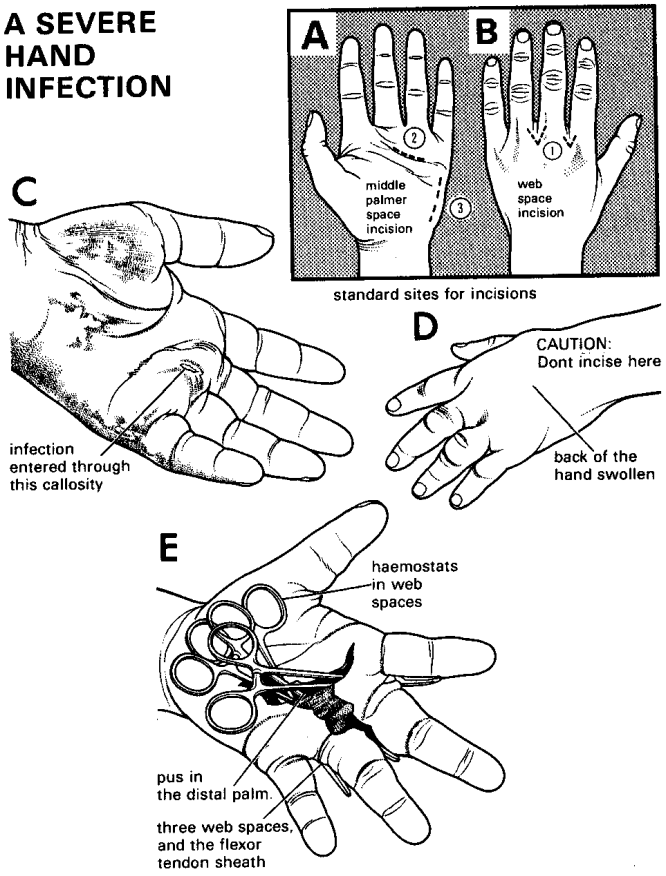


Fig. 8-5 SEVERE HAND INFECTION.
This started as a web infection which spread to the mid-palmar space. A, the standard site of incisions for a middle palmar space infection (incisions 2 and 3), and B, for web space infections (incision 1). In this patient these incisions had to be modified. C, callosity through which infection entered. D, although the back of the hand was swollen, swelling was due to secondary inflammatory oedema only. E, pus found in the distal palm, the 3 web spaces, and the flexor sheaths. Drain the spaces and excise necrotic tissue. *After Milford, L, Hand Infections, in Edmondsen AS, Crenshaw AH (ed), Campbell's Operative Orthopaedics, CV Mosby Fig. 3.355-6 with kind permission.*

The mid-palmar space communicates through the carpal tunnel with a space deep to the flexor tendons in the forearm (the space of Parona). If there is pus there, you may be able to detect fluctuation between it and the pus in the palm.

N.B. Always use a tourniquet.
Make a transverse incision (incision 2) in the middle ⅓ of the distal or proximal palmar creases or wherever fluctuation is maximal. Enter the middle palmar space on either side of the flexor tendon of the ring finger. Or, enter it through an incision along the ulnar border of the hand, passing between the 5th metacarpal and the hypothenar muscles (incision 3). As soon as you are through the skin, use blunt dissection (Hilton's method) in the line of the tendons and nerves (8.13).

CAUTION!
(1) *Don't make your initial incision deeper than the palmar fascia*. Push a blunt instrument through it to free the pus underneath. You can then see clearly to open up the space more by a combination of sharp and gentle blunt dissection.
(2) *Don't cut the digital nerves or vessels, the flexor tendons, or the lumbrical muscles*.

If there is pus in the space of Parona, drain it through a longitudinal incision (8-6A: incision 6) on one side of the *palmaris longus* tendon (absent in 5% of people), taking care *not to injure the median and ulnar nerves or the radial and ulnar vessels*. *Don't incise the dorsum of the hand* (8-5D).

8.10 Thenar space infection

The thenar space (8-1B,C) is sometimes infected because of a penetrating wound. It lies underneath the palmar fascia, and is bounded dorsally by the transverse head of the *adductor pollicis*. On its ulnar side a fibrous septum divides it from the mid-palmar space. The thenar eminence is grossly swollen, and the thumb is abducted.

Drain the thenar space over the point of greatest tenderness through a curved incision in the web between the thumb and index finger, parallel to the border of the first *dorsal interosseous* muscle, on the dorsal edge of the hand (8-6C: incision 4). Or, drain it through an incision along the thenar crease in the palm (8-6A: incision 5). Insert a haemostat deep into the abscess, and open it. You will usually find that it is walled off from the muscles of the thumb.

CAUTION! Remember the course of the sensory and motor branches of the median nerve which lie within the thenar muscles. These are in less danger from incision 4 (8-6C) than from incision 5 (8-6A).

THE COMMONEST CAUSE OF SWELLING ON THE DORSUM IS INFECTION IN THE PALM

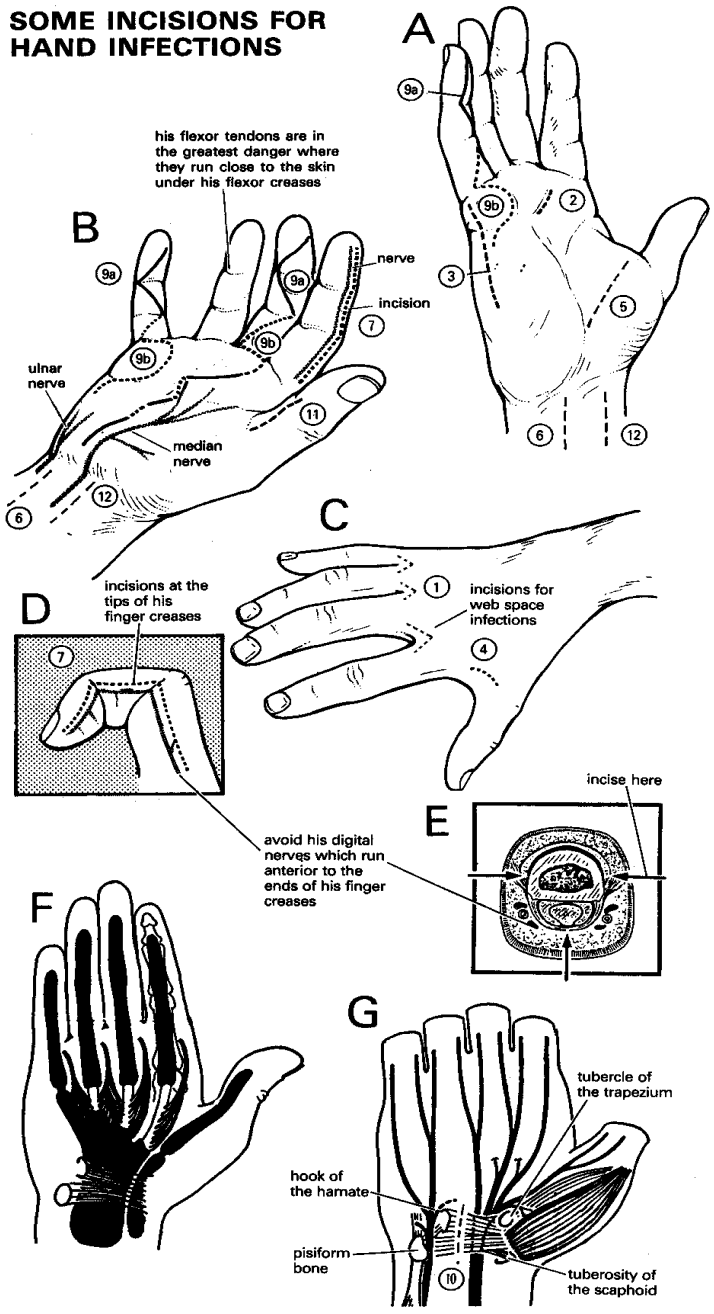


Fig. 8-6 INCISIONS FOR HAND INFECTIONS.
A-D, incisions for more serious hand infections: Incision 1 for web space infections. Alternative incisions 2,3 for a mid-palmar space infection. Alternative incisions 4,5 for a thenar space infection. Incision 6 for pus in the space of Parona (proximal to the flexor retinaculum and deep to the flexor tendons). Incision 7 for mild tendon sheath infection. Incision 9; 9a the 1st part, and 9b the 2nd part if necessary for more serious infections. Incisions 11 or 12 drain the radial bursa. E, cross-section of the finger. F, tendon sheaths, radial and ulnar bursae, lumbrical muscles, and flexor retinaculum. G, incision 10 for dividing the flexor retinaculum.
On the ulnar side of the retinaculum, the palpable landmarks are the pisiform and the hook of the hamate. On the radial side, you can feel the tubercle of the scaphoid and, more deeply, the tuberosity of the trapezium. The digital nerves are at the 'edges' of the palmar surfaces, so *don't incise there*. Either incise towards the middle of the palmar surfaces or laterally towards the dorsum as shown by the arrows. Incisions for the finger tips are shown in 8-2,3**. Some infections don't have fixed incisions (the volar surfaces of the proximal and middle phalanges, the superficial palmar space, and the dorsum of the hand).**
E-F, after Basmajian, JV, Grant's Method of Anatomy, 9th ed 1975. Williams & Wilkins, with kind permission.

8.11 Dorsal hand and finger infection

Infection almost anywhere in the hand makes the dorsum swell, but pus seldom collects there. On the rare occasions when it does, it is usually subcutaneous, and only occasionally in the subaponeurotic space under the extensor tendons (8-1B).

If localized tenderness persists for >48h, *don't wait for fluctuation*. Drain it through a longitudinal incision over the point of greatest tenderness.

8.12 Flexor tendon sheath infection

The sheaths of the flexor tendons in the hand lie nearest to the skin as they pass under the flexor creases of the fingers. It is here, and particularly over the distal flexor crease, that they are most often punctured and infected. They can also be infected by spread from a pulp infection. The sheaths of the little finger and thumb (and occasionally those of the other fingers also) extend proximally into the palm, and so provide a path through which infection can spread.

If an infected tendon sheath bursts, it does so into the mid-palmar space, through one of the lumbrical canals.

N.B. (1) An infected tendon may later stick to its sheath and make a finger stiff.

N.B. (2) If pressure inside a sheath exceeds that in its vessels, which can occur if drainage is delayed, the tendon will become ischaemic and slough.

If infection is localized or one area is maximally infected, *staphylococci* are the usual cause. Only one segment of the finger is swollen, so that distinguishing a localized tendon sheath infection of this kind from an infection of one of the middle palmar and thenar spaces can be difficult (8.9).

If infection is fulminating, *streptococci* are usually responsible, and the whole finger is swollen, sausage-shaped and acutely tender, without becoming red. The swelling extends into the distal palm. The finger remains partly flexed, except perhaps for a little movement at its mcp joint.

The danger when you open a tendon sheath is that you may cut the digital nerves. So study where these run in the cross-section of the finger (8-6G). Either approach a tendon laterally, well towards the dorsum, or from the palm. The danger area is the 'palmo-lateral' region. The other nerve which is in danger is the motor branch of the median nerve as it curves round the distal end of the flexor retinaculum and the tubercle of the trapezium.

Adjust your incisions to the severity of the infection. You can approach an infected tendon sheath:

(a) along the side of a finger towards the dorsum (8-6B: incision 7).

(b) through several transverse palmar incisions (8-7D: incision 8).

(c) by zig-zag cuts on the palm (8-6B: incision 9); these give the best exposure, but take longer to heal. Incisions 7 and 8 are for less severe infections.

Tendon sheath infections are common in leprosy (32.18), where loss of sensation allows neglect of an infection until it has destroyed the tendon sheaths themselves.

EXPOSING THE TENDON SHEATHS

Start by opening the soft tissue over the involved segment through a small lateral incision (8-7A: incision 7). Examine the synovial sheath. If there is any sign of infection (redness or thickening), open the sheath itself and look carefully at the fluid. If there is much fluid, it is probably infected; if it is even a little cloudy, it is certainly infected.

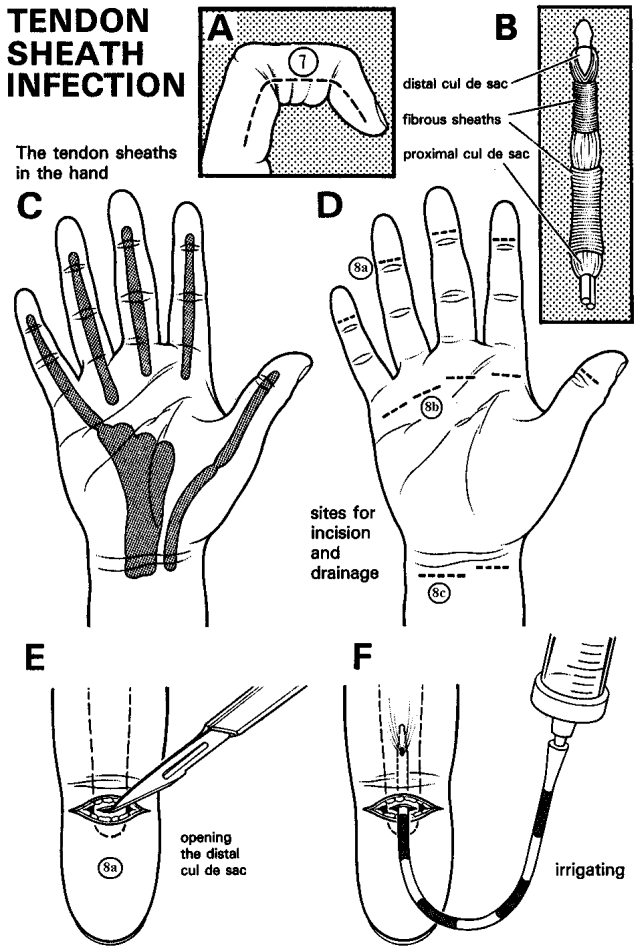


Fig. 8-7 INFECTIONS OF THE TENDON SHEATHS.
A, lateral incision for opening an infected tendon sheath (incision 7). B, anatomy of a tendon sheath, to show the fibrous pulleys opposite the shafts of the phalanges. C, surface markings of the tendon sheaths. D, transverse incisions for draining tendon sheaths (incisions 8a,b). E, open the distal *cul-de-sac* (incision 8a). F, irrigate the tendon sheath. *E, F, After Rintoul RF (ed) Farquharson's Textbook of Operative Surgery Churchill Livingstone 7th ed 1986 Fig. 317-8 with kind permission.*

If a sheath is infected, make several incisions over the finger(s) and distal palm (8-7D,E: incisions 8a,8b). Hold the sheath open with hooks and retractors. Using a stiff catheter, syringe the sheath with sterile water (8-7F).

If a sheath is infected in the palm (as is usual with the little finger and thumb), make a further incision (8-7D: incision 8c) at the wrist, and repeat the irrigation, inserting the catheter through the palmar incision.

If the tendon sheaths are grossly infected, operate urgently. Open the sheath by a zig-zag incision on the volar surface of the finger (8-6B: incision 9a,9b). Do this in 2 stages. First cut along the solid lines and then, if necessary, join up these incisions by cutting along the dotted lines. Cut the flaps in the palm larger than those in the fingers, and make them follow the skin creases where possible. Cut through the skin and open the tissues with scissors. Leave bridges of the sheath over the joints to act as pulleys to prevent the tendons prolapsing.

CAUTION! Don't take the incisions laterally where they may injure the neurovascular bundles.
Wash out the pus with water. *Don't close the incision*; the flaps will heal by granulation to leave a linear scar.

If a tendon has become a grey slough, extend the incision, withdraw the dead part into the wound, and excise it. Preserve its sheath and pulley. Allow the wound to heal. If the hand settles well, it may be possible to insert a tendon graft later. This will be worthwhile only if the joints of the fingers are mobile. So, as soon as the swelling is starting to settle, start intensive physiotherapy, both by the patient himself and by a physiotherapist: *this is important!* If the finger remains stiff, try to persuade that it should be amputated (35.4) as a stiff finger can be a severe handicap.

If a tendon and its sheath are extensively disorganized, amputate the finger. *If you don't do so:*
(1) infection may spread and cause further damage,
(2) when the finger heals, it will be stiff, and cause considerable disability by impairing the grip of the other fingers (35.4).

N.B. A stiff thumb is much better than no thumb, so don't amputate the thumb.

If the palm is seriously infected, divide the flexor retinaculum to free the tendons. Approach this either:
(a) through a longitudinal incision 1cm to the ulnar side of the scaphoid tubercle. Make a 5cm longitudinal incision over the retinaculum. Keep to the ulnar side of the median nerve and its ulnar branch (8-6G: incision 10), or
(b) as shown for the ulnar bursa (8-6A: incision 3).

AN UNNECESSARY INCISION IS BETTER THAN A LOST FINGER

8.13 Ulnar bursa infection

Infection of the ulnar bursa is the most serious hand infection, because it contains all the flexor tendons of the fingers. The whole hand is oedematous, the palm is moderately swollen, and there may be a fullness immediately above the flexor retinaculum. The flexed fingers resist extension, particularly the little finger, and least of all, the index. The radial and ulnar bursa sometimes communicate with one another. So if one of them has been infected, infection may follow in the other a day or two later.

Open the tendon sheath of the little finger with palmar flaps (8-6B: incisions 9a and if necessary 9b).
Incise the skin and deep fascia over the antero-medial side of the 5th metacarpal (8-6A: incision 3). Separate the *abductor* and *flexor digiti minimi* muscles from the bone. Retract them forwards and you will see the *opponens digiti minimi* muscle. Divide this close to its attachment to the flexor retinaculum.
Divide the flexor retinaculum deep to *opponens digiti minimi*: you will see the bulging ulnar bursa. Wash this out, as for a tendon sheath infection (8.12).
You can also drain the mid-palmar space through this incision (8.9).

8.14 Radial bursa infection

The radial bursa is a continuation of the tendon sheath of the *flexor pollicis longus*, so that any infection inevitably involves both of them. The distal phalanx of the thumb is flexed and rigid. Extension of the thumb is impossible but extension of the other fingers is possible. The hand is tender over the sheath of *flexor pollicis longus*, and you may be able to feel a swelling above the flexor retinaculum. If treatment is delayed, infection may spread to the ulnar bursa, or the tendon of *flexor pollicis longus* may slough.

Incise the radial bursa (8-6B: incision 11) along the proximal phalanx of the thumb. Open it at its distal end; pass a probe proximally towards the wrist, and make a 2nd incision over its proximal end (8-6B: incision 12). Insert a fine catheter down the sheath and irrigate it with water.

CAUTION! Don't incise along the radial border of the first metacarpal. Dissecting among the muscles there may impair the ability to bring the thumb across the palm.

8.15 Septic arthritis of the finger

The finger joints are easily infected from open wounds, or from nearby infections. A human bite into a joint is particularly dangerous. The infected joint is acutely tender, swollen and painful. An early sign is if twisting the joint is painful. Its ligaments, cartilage, and bone are soon involved, so that inevitably the result is a stiff joint. A stiff dip joint is little disability, but a stiff mcp or pip joint produces a severely disabled finger which is probably better amputated.

Treat with cloxacillin or chloramphenicol *and* metronidazole; but this is less important than drainage and an efficient surgical toilet.

Open the joint immediately, especially if there is a wound over it. If the edges of the wound are not obviously infected, excise their extreme margins. Examine the extensor tendon.

DON'T AMPUTATE THE THUMB
(except in extreme circumstances)

If you have not divided the extensor tendon, enter the dorsolateral aspect of the joint and retract it to the opposite side. Look inside the joint. Remove any debris and loose bits of cartilage or bone. Syringe it out with water. Leave the skin wound open for delayed primary closure.

If you had to divide the extensor expansion, repair it when you have drained the pus. Immobilize the joint in the position of function (7.17), in case it stiffens, *not the position of safety*.

If the finger (but *not the thumb*) is stiff, consider amputation (35.4) in an adult, but *not in a child!*

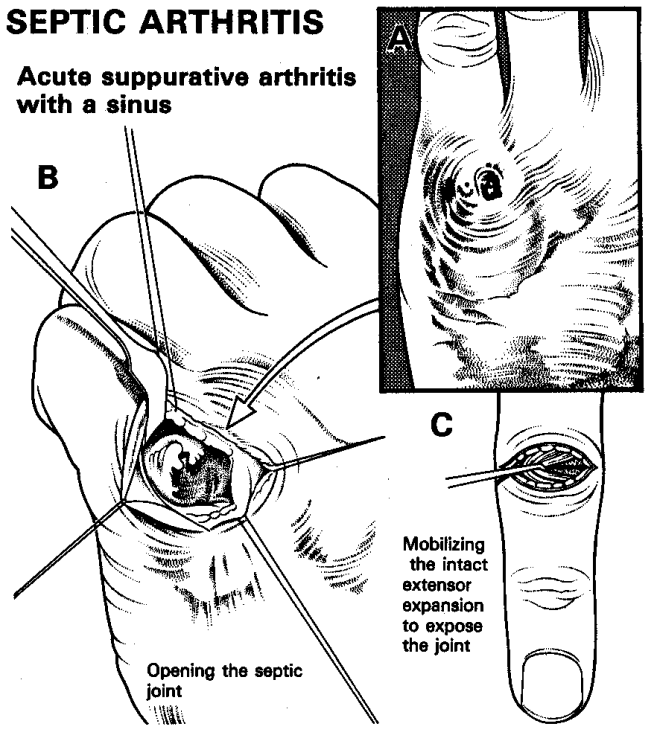


Fig. 8-8 SEPTIC ARTHRITIS.
A, acute suppurative arthritis presenting with a sinus. B, explore the lesion. C, mobilize an intact extensor tendon to expose a suppurating pip joint.
After Bailey DA, The Infected Hand, HK Lewis 1963 Figs 65-67, with kind permission.

8.16 Difficulties with hand infection

Hand infections, particularly if they are not well treated, can cause many problems.

If, a few hours after a minor scratch, the hand becomes hot and shiny, red lines spread up the arm, and there are rigors, tachycardia, and severe headaches, this is lymphangitis progressing to streptococcal bacteraemia and septicaemia. Treat with IV chloramphenicol or cloxacillin, and if an abscess or gangrene forms later, incise or deslough the hand.

If the hand has been infected as the result of a human or animal bite, perform an efficient wound toilet under a tourniquet, excise all tissue of doubtful viability, and leave the wound open. Treat with chloramphenicol or cloxacillin *and* metronidazole.
There is great danger of a serious infection, particularly with anaerobes. If you treat early, recovery is likely with a useful, mobile hand. If presentation is late (8-9), it will remain stiff, especially if a joint or a tendon sheath is involved. When the infection is controlled, amputate the stiff useless finger (35.4).

If swelling and tenderness spread above the wrist, pus has probably tracked proximally behind the flexor tendons up the arm into the space of Parona, as a result of a neglected palmar infection (8.9). Drain it (8-6A: incision 6).

If there are exposed joints or tendons after a hand infection, leave them open for c.1wk until the infection is controlled. Raise the hand in a roller towel, and start movements as soon as pain permits. When healthy granulations have appeared, try to get tissue cover by using an abdominal wall or groin flap.

If osteomyelitis develops, continue antibiotic treatment, immobilize the hand in the position of function. Get a radiograph 2wks later and remove sequestra through dorsal incisions as necessary. Osteomyelitis of the distal phalanx is common in untreated pulp infections (8.5), and can follow other hand infections. You may eventually have to amputate the infected finger.

Fig. 8-9 DISASTER WITH A HUMAN FINGER BITE.
The wound entered the dip joint, which became infected. The finger might have been saved by an efficient wound toilet soon after the injury. Amputation is advisable in this case (35.4).
After Bowesman, C. Surgery and Clinical Pathology in the Tropics Livingstone. 1960, permission requested.

If it involves a metacarpal (uncommon), treat this as if it were any other long bone. Approach it through a dorsal incision, and reflect the extensor tendons. Approach the middle and lateral phalanges through mid-lateral incisions.

If it involves a distal phalanx, this will usually present at the finger tip. Cut it off with a bone nibbler.

If discharge and pain persist, this is from:
(1) Inadequate drainage and desloughing.
(2) Osteomyelitis.
(3) Spread of a more superficial infection to a tendon sheath, or another fascial space.
(4) Sloughing of a tendon.
(5) A foreign body.

If an adult's finger continues to be painful and discharge because of osteomyelitis or established septic arthritis of an mcp or pip joint, consider amputation (35.4), because the proximal joints may become stiff too. A stiff dip joint is not much of a disability. Amputate at least through the joint proximal to the bone involved. *Don't merely remove part of the involved bone*, because the infection will spread. The thumb is an exception; spare as much bone as you can, and *don't amputate if you can avoid doing so*, because even a stiff stump of a thumb is better than no thumb at all.

CAUTION! A child is much more likely to regain some useful movement eventually, so *don't amputate* unless the finger remains stiff after infection has settled.

8.17 Pus in the foot

Foot infections are common, especially in communities where people don't wear shoes. Fine movements are not so important in the foot as they are in the hand, so that infection of the tendon sheaths of the foot is less of a disaster. *You must however drain septic arthritis and osteitis*, or persistent sinuses may follow.

Some foot infections are more complicated, *e.g.* osteomyelitis of the calcaneus and talus (7.12) and mycetoma (34.11). Diabetic and leprosy patients are particularly liable to foot infections (32.2), because of neuropathies.

SUPERFICIAL INFECTIONS
Manage subcutaneous infections (8.2), apical toe space infections (8.3), paronychia (8.4), pulp infections (8.5) and web space infections (8.7) as in the hand. They are all fairly common.

For all but the most superficial infections, use a tourniquet (3.4), unless the circulation has been impaired by ischaemic disease.

DEEP INFECTION OF THE PLANTAR SURFACE OF THE FOOT is usually due to an injury, such as a thorn, which has penetrated deeply.

If you suspect a foreign body, incise the abscess, search for the foreign body and clean out the cavity thoroughly. Leave the wound open sufficiently for it to heal up from below.

If infection is spreading on to the foot and up the leg, explore and drain the lesion, and treat with an antibiotic suitable for the *staphylococci* in your area. As in the hand, rapidly spreading infections are likely to be due to haemolytic *streptococci*.

INFECTIONS OF THE DORSUM OF THE FOOT present early, and you can usually drain them through a small incision using LA.

INFECTIONS OF THE TENDON SHEATHS are uncommon except in leprosy, and when there is a foreign body involving the tendon sheath. Incise over the infected part, drain it, and leave the wound open. In a late case you may need to remove necrotic tendon.

SEPTIC ARTHRITIS can involve any joint.
If a pip or dip joint is involved, open it widely through a longitudinal incision on the dorsal surface to one side of the extensor tendon. Clean it out and leave it open to drain.

If an mcp joint is involved, approach it either from the dorsal surface (open it from just one side of the extensor tendon), or from the plantar surface. Open the wound widely and let it drain. Wounds in the plantar surface heal well.

If other joints are involved, approach them from the side where the bone is nearest to the surface. Clean the joint out well and leave it open.

OSTEITIS. Treat with cloxacillin or chloramphenicol and remove necrotic bone as necessary in chronic cases.
If the phalanges are involved, drain the infection and it will probably settle. Osteitis commonly follows infection in the soft tissues, especially infections of the pulp of the distal phalanx.

If the metatarsals are involved (rare), there may be:
(1) Osteomyelitis following an injury. Approach the bone through a dorsal incision and reflect the extensor tendons. Drain the wound and remove necrotic tissue. Loss of 1 or 2 metatarsals is of little functional importance.
(2) Acute haematogenous osteomyelitis. In a child <10yrs, an antibiotic alone may be adequate. In a child >10yrs, the bone will also need drilling.
(3) Chronic haematogenous osteomyelitis. This presents with persistent pain and sinuses. Remove necrotic bone, without waiting for the formation of an involucrum.

CAUTION! **If the foot becomes infected without obvious reason**, search carefully for:
(1) A foreign body.
(2) Diabetes.
(3) HIV disease.
(4) Ischaemia.
(5) Leprosy (32.2).

Severe infection in these cases is best treated by below-knee (35.7), through-knee or above-knee amputation (35.6) as piece-meal debridement usually fails to control the sepsis and just prolongs the agony.

POST-OPERATIVELY, *stop weight-bearing*.
If there is a severe infection, apply a plaster gutter splint to hold the foot in neutral position. This will reduce pain and ensure that the foot is in the best position if it does become stiff.

9 Pus in the pleura, pericardium & lung

9.1 Pus in the pleural cavities: empyema

Pus usually reaches the pleural cavity from infection of the lung adjacent to it. This can be pneumonia, a lung abscess, or the pneumonitis that may follow an inhaled foreign body (usually in a child), or carcinoma of the bronchus (usually in a cigarette smoker or mine worker). Frequently, an empyema is tuberculous, especially in HIV disease; rarely it may follow rupture of a liver or subphrenic abscess through the diaphragm.

A common history is that a week or more before, as the patient was beginning to recover from a chest infection, improvement stopped. He now remains ill, anorexic and febrile, and is starting to lose weight, despite antibiotics. Many kinds of bacteria can be responsible, especially *streptococci*, *staphylococci*, *and E. coli*. Antibiotics are only effective in the earliest stages, and may mask the symptoms of an empyema later. The result is that empyemas can remain undetected for years and are often missed in a busy outpatient department. This is sad because you can treat them, so watch out for them, and ask your staff to do so too.

Pus in the pleural cavity, like pus anywhere else, must be removed. To begin with, it is thin, like serum; later it thickens and looks like scrambled egg. So adapt your method of removing it to its thickness. While it is still thin, aspirate it using a three-way tap or use closed drainage, as if you were draining blood from an injured chest. The surfaces of the pleura will not have stuck together at this stage, so you will have to use an underwater seal to prevent air getting into the pleural cavity and letting the lung collapse.

If the pus in the pleural cavity is left undrained, it will soon become too thick to flow down a long thin tube into a bottle. Once the empyema has reached this stage, you can improve things greatly by draining pus through an open drain. To do this, you need to remove a piece of a rib and open its bed. The surfaces of the pleura will be stuck so firmly that a pneumothorax will not ensue. In order to do this safely, *be sure to*:

(1) Remove the piece of rib from inside its periosteum, so as not to injure the vessels and nerve which run just below it.

(2) Place the inner end of the drainage tube at its most dependent site in the sitting position.

If pus in the pleural cavity remains even longer, it will be replaced by fibrous tissue which will be very difficult to remove: this is an extensive operation called decortication.

Children have special problems. In a child between 1-3yrs, an empyema may follow a post-measles pneumonia, or the rupture of a staphylococcal lung abscess into a pleural cavity.

The child is likely to be malnourished, anaemic, and anorexic, with a persistent cough, fever, dyspnoea, diarrhoea, and perhaps vomiting. He may be very sick indeed with a pyopneumothorax under tension. Check the HIV status, and drain the pus.

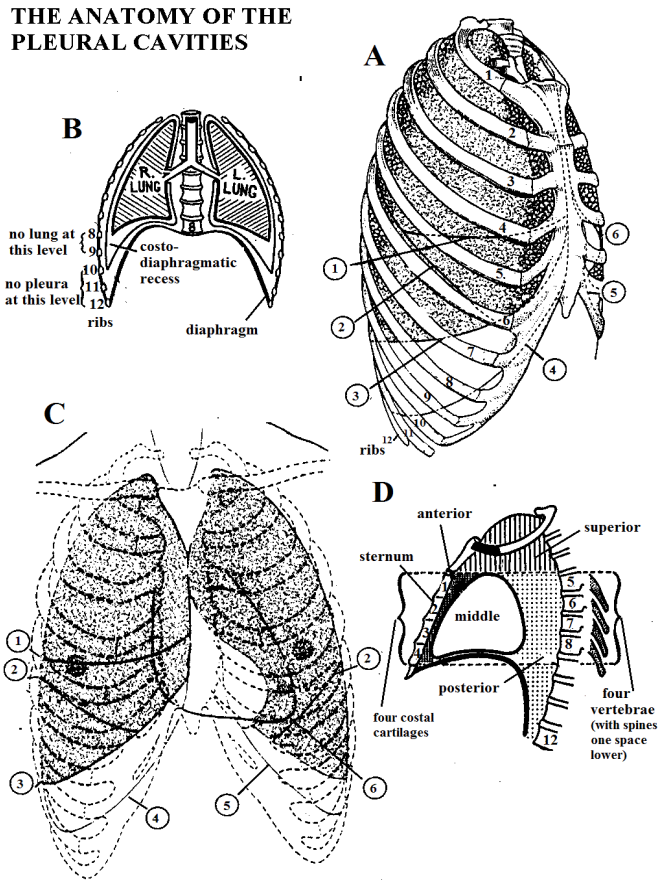


Fig. 9-1 THE ANATOMY OF THE PLEURAL CAVITIES.
A, relation of the pleurae and lungs to the chest wall. B, coronal section of the thorax (semischematic)**. C, ventral aspect of the thorax showing the surface projections of the heart and pleurae. D, subdivisions of the mediastinum, with surface markings.**
N.B. The anterior mediastinum contains essentially only the thymus.
(1) horizontal fissure. (2) oblique fissure. (3) inferior border of the right lung. (4) costodiaphragmatic reflexion of the right pleura. (5) costodiaphragmatic reflexion of the left pleura. 6, cardiac border.
N.B. In lung collapse, the level of the diaphragm rises.
A,C 9-23, from Gray's Anatomy, Churchill Livingstone. B, 6-6 & D, 6-9 from Basmajian JV. Grant's Method of Anatomy 9th ed 1975, with kind permission.

CLINICAL FEATURES. If an empyema involves the whole of the pleural cavity and contains ≥1L of pus, you should be able to diagnose it clinically. Look for limited movement of the chest on the affected side, shifting of the trachea and apex beat, dullness to percussion, reduced breath sounds and reduced vocal fremitus. Vocal resonance (the sound "99") may be high-pitched at the top of the empyema and absent over its lower part.

RADIOGRAPHS usually show a dense area at one lung base, often rising laterally towards the axilla. Take an *erect PA and a lateral* view to show the site and extent of the empyema.

N.B. A ruptured diaphragm or hiatus hernia with stomach or colon in the chest may look like a pyopneumothorax on a radiograph if there is no air visible!

ULTRASOUND is very useful and will indicate if there are septations in the pleural fluid.

ANTIBIOTICS. When an empyema is established, antibiotics are ineffective. Pus must be drained. If there is fever or malaise, treat with chloramphenicol until sensitivity tests show the need for change.

ASPIRATING A PLEURAL EFFUSION (GRADE 1.1)
INDICATIONS
(1) To confirm the diagnosis.
(2) To remove the bulk of the fluid in the early stages while it is still thin (*i.e.* needing only one pull to fill a 10ml syringe using a 21G needle).

EQUIPMENT. A Martin's aspirator, with a 3-way tap, a 20ml syringe, LA solution and a receiver. Or, improvise the equipment.

METHOD. Preferably use the sitting position, leaning over a bed table or a pile of pillows. You may need to provide oxygen. Aspirate near the lowest point of the empyema, as defined on the PA and lateral radiographs, or by ultrasound. To establish this, aspirate several sites if necessary, so as to find the lowest site that yields pus, but remember the surface markings of the pleura. Look these up if you are not sure, and mark them on the patient's skin. Commonly, the posterior axillary line is the correct vertical line in which to aspirate. Infiltrate LA solution into the skin and subcutaneous tissues over the chosen space, down to the pleura *(it does not matter if you enter the pleura: you will do so anyway!)* and also a space above and below. Insert the needle, pierce the pleura and aspirate gently; turn the tap and discharge the fluid into a receiver. If you don't have a 3-way tap, you can attach a giving set and use an empty vacolitre of IV fluid or attach a glove.

CAUTION! Very rapid decompression of a large pleural effusion can cause acute mediastinal shift and a vasovagal attack.

If the patient becomes distressed, clamp the drain immediately!

If the effusion recurs, repeat the aspiration but if pus does not stop forming, proceed to closed drainage.

CLOSED DRAINAGE FOR A PLEURAL EFFUSION (GRADE 1.4)

Many empyemas don't resolve on aspiration alone and closed drainage is necessary. Use an Abram's needle to get a pleural biopsy for tuberculosis or melioidosis. Insert an underwater seal drain (1cm diameter for an adult, 0·5cm diameter for a child), as for a haemothorax. Instil 250,000 units of streptokinase bd for 3 days to promote fibrinolysis. Leave the drain for at least 2wks until firm adhesions have formed between the surfaces of the pleura, which will prevent the lung collapsing when you take the tube out.

The instillation of 5-10g of lipiodol before repeat radiographs is a useful way of defining the lowest point of the empyema. Beware of damage to the liver or spleen by inserting a drain too low!

If radiographs show disappearance of the empyema and re-expansion of the lung, cut the suture securing the tube, and pull it out quickly while closing the hole with a purse-string suture. Remember to follow up for diagnosis of TB.

If there is no improvement, proceed to open drainage.

EMPYEMA THORACIS (PYOTHORAX)

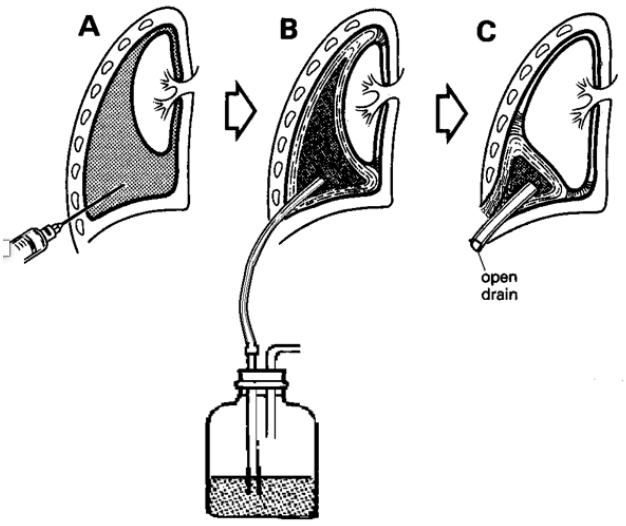


Fig. 9-2 STAGES IN THE DRAINAGE OF PUS IN THE PLEURA. A, drain a very recent pleural effusion with a syringe and needle. B, if pus recurs, use an underwater seal drain in a bottle (closed drainage). C, if pus becomes thick, resect a rib, and insert a short wide tube (open drainage). Shorten this tube as the empyema drains, and make sure it is in the bottom of the cavity.

OPEN DRAINAGE FOR AN EMPYEMA, RESECTING A RIB: PLEUROSTOMY (GRADE 2.5)

INDICATIONS. If pus thickens, so that aspiration needs 2 or more pulls to fill a 10ml syringe using a 21G needle, or where the pus has over ⅓ as sediment, or when closed drainage has failed, proceed to open drainage. The lung must have stuck to the ribs. Prove this by slowly withdrawing the tube of the underwater seal drain from the water. If the column of water does not run up towards the pleura, but stays in the tube, the pleura has stuck to the ribs, so that an underwater seal is unnecessary and open drainage can start.

If pus bulges on the chest wall (*empyema necessitatis*), almost certainly due to TB, open drainage is indicated.

RADIOGRAPHS. Examine PA and lateral chest films with the greatest care to see which rib to resect. If you cannot easily see the lowest point of an empyema, inject 10ml of oily contrast medium before you expose the films.

ANAESTHESIA. Use a combination of LA, intercostal blocks and sedation in theatre. Block the intercostal nerves at the site of your chosen incision, and also one rib above and one below it as far posteriorly as possible.

METHOD. Drain the empyema from its lowest point in the sitting position. Choose the lowest point of the empyema posteriorly. Often, the 9th rib in the posterior axillary line is the best, but it may be below this.

CAUTION! Don't make the opening too low, because the diaphragm will rise as the pus drains and block the opening. It should always be at least one space above the diaphragm. Use the sitting position, leaning forwards against the operating table. Before incising, confirm, by aspiration through more than one intercostal space, that you have chosen the correct rib to remove. Make a 9-15cm vertical incision, extending above and below the selected rib, so that you can more easily resect the rib on either side if necessary. Cut down to the rib, and incise the periosteum along its centre. Use a curved Farabeuf's rougine to strip the periosteum with its attached intercostal muscles from the outer surface of the rib. Clean its upper and lower borders. Then use Doyen's raspatory (or Farabeuf's rougine) to remove the periosteum from its inner surface. Strip its upper and lower borders (9-3).

CAUTION!

(1) The intercostal blocks should have anaesthetized the parietal pleura adequately; if they have not, repeat the intercostal blocks and wait. If you fail to administer adequate anaesthesia, extreme pain may cause a vasovagal attack.

(2) The intercostal vessels can bleed severely if you fail to identify them, so *be sure to avoid them by keeping inside the periosteum*.

Excise a 7-10cm length of rib with an osteotome, rib shears, or a large pair of bone cutters. In HIV disease, excise an adjacent length of rib as well to make an adequate-sized hole. Make an incision in the bed of this rib through into the pleural cavity. Open it with a haemostat, explore it with your finger, and remove what semisolid pus you can with sponge holders. This will probably induce coughing. Take a biopsy of the pleura for histology. Irrigate the cavity with copious amounts of warm water, *not hydrogen peroxide*.

CAUTION!

(1) **If when you explore the cavity with your finger, you find that you have not removed the rib at the bottom of the cavity**, remove the rib below. If you don't do this, the empyema will not resolve completely.

(2) Send pus for smear and culture; it may be tuberculous, which looks different from ordinary pus, is more watery and contains particles.

Fix a wide radio-opaque tube in the empyema cavity, leaving about 2cm above the skin surface. Fix it with a suture, a safety pin and adhesive strapping to avoid it disappearing into the chest; apply a large gauze and cotton wool dressing.

POST-OPERATIVELY, encourage vigorous breathing exercises by blowing into balloons or surgical gloves. Flush the cavity daily with warm sterile water or hydrogen peroxide. Encourage sleeping on the affected side to improve drainage. Monitor the size of the cavity by introducing contrast medium and taking radiographs.

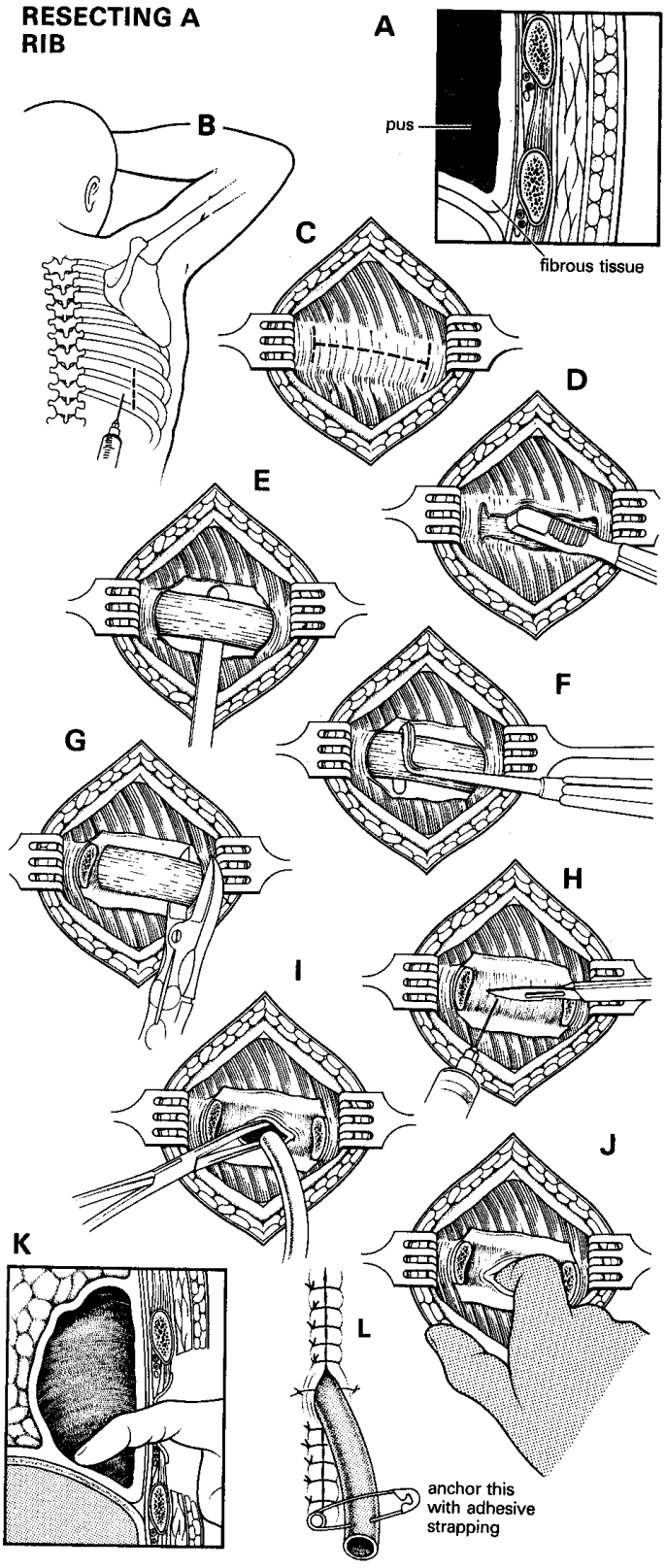


Fig. 9-3 RESECTING A RIB.
A, empyema covered with a thick layer of fibrous tissue. B, common site for draining an empyema: the 9th rib in the paravertebral line. Vary this as the occasion demands. C, incise the skin down to the periosteum. D, reflect the periosteum with Farabeuf's rougine. E, reflect the periosteum off the inner surface of the rib. F, complete the task with Doyen's raspatory. G, resect the rib. H, prepare to incise the periosteum in the bed of the rib. I, suck out the pus. J, K, put in a finger to break down the loculi. L, drainage tube in place.

Alternatively, measure how much sterile saline you can run into the remaining cavity.
When drainage stops or becomes <5ml/day, remove the tube. The residual sinus will heal, provided that there is no bronchopleural fistula. This can take 2 to 3 months.

CHILD WITH AN EMPYEMA. *You cannot drain a small child's pleural cavity adequately by inserting an intercostal drain between two ribs*, because the drain will be nipped by the ribs or obstructed by pus. So remove 1-2cm of rib, using ketamine (LA is inadequate in children), to make a hole which is big enough for a tube. Adequate drainage will eventually achieve a cure if:
(1) the lung is not immobilized with thick fibrin,
(2) there is no bronchopleural fistula, and
(3) the empyema is localized.
Start drainage with an underwater seal drainage bottle. This will limit activity, and may cause the drain to be pulled out, but the lung will expand.
When you are confident that the lung has stuck to the ribs (see above), cut the tube short, fit it with a pin and butterfly strapping, put a colostomy bag over it to collect the pus and allow exercise. Increased activity is the best physiotherapy.

DIFFICULTIES WITH AN EMPYEMA
If air comes out with the pus, there is a BRONCHOPLEURAL FISTULA which is unlikely to close spontaneously. You can confirm this if, on coughing, pleural irrigating fluid comes up. Make sure tuberculosis is treated, if present. Once there is no more pus draining, fill the drainage bottle with 500ml sterile water and empty this into the pleural space to clean it. Drain this and repeat the process till the fluid comes out clear.
Then perform a PLEURODESIS: mix 2-5g talcum powder (or tetracycline, if there is an allergy to talc) with 50ml saline to make an opaque milky fluid which can still flow, and introduce this into the pleural space through the chest drain using a bladder syringe. If the patient feels a pleuritic pain when you do this, the inflammatory reaction may well seal the fistula. If this procedure fails, you can repeat it one or two times more. Otherwise a decortication may be needed.

If the intercostal vessels bleed, encircle them with a needle and thread. Avoid tying the nerve because this is painful. If you have difficulty, transfix the vessels with a ligature, so that they are compressed against the stump of the rib which remains.

If the empyema fails to heal:
(1) The drainage tube may be too high or too far forward.
(2) You may have removed it too early.
(3) You may have put it in too late.
(4) There may be a foreign body, such as a piece of drainage tube, in the chest.
(5) There may be a fistula between the bronchi and the pleura.
(6) There may be tuberculosis, carcinoma, actinomycosis, or a ruptured liver abscess (15.10), which may be amoebic.
Further dependent drainage is all that is probably needed for (1), (2) or (3).
Instil 5-10ml of contrast medium, repeat the radiograph, and if necessary resect another rib.

9.2 Pus in the pericardium

Fluid sometimes accumulates in the pericardium. In sufficient quantity this may embarrass the action of the heart (cardiac tamponade) and may be fatal, so you should remove it urgently! The fluid may be blood after a cardiac injury or an effusion from many causes, either infected or sterile. Presentation with symptoms that immediately suggest a pericardial effusion is unlikely. Another initial diagnosis is likely before you observe some of the following signs:
(1) grossly distended neck veins,
(2) *pulsus paradoxus* (>10mm Hg fall in arterial pulse pressure on inspiration),
(3) a large cardiac shadow on a chest radiograph.

Elsewhere in the body, you drain pus to treat an infection. In the pericardium, you are mainly draining it to overcome its mechanical effects.

Severe illness with fever and breathlessness is accompanied by signs of a low cardiac output with a poor peripheral circulation; there is a small pulse volume, tachycardia, a low normal or subnormal blood pressure, and soft heart sounds. Early on you may hear a pericardial rub, but the accumulation of fluid soon separates the pericardial surfaces and stops the rub. There are the signs and symptoms of heart failure (enlarged liver, dependent oedema or ascites), and an increased area of cardiac dullness. The severity of the signs of cardiac tamponade is related more to the rate at which fluid accumulates in the pericardium than to the volume of fluid in it. The diagnosis may be obvious, or if fluid has accumulated slowly, it may be difficult.

There are problems:
(1) Any cause of cardiac failure may have distended the neck veins.
(2) Although *pulsus paradoxus* strongly suggests a pericardial effusion, not all patients show it.
(3) The radiographic finding of a large globular heart can also be due to gross cardiac enlargement without there being any fluid in the pericardium.
Ultrasound is much more reliable, and can also give you information about the thickness of the pericardium and the thickness of the fluid in the sac. It can also guide you during aspiration.

The great danger in putting a needle into the pericardial cavity to drain it is that:
(1) You can easily penetrate the right ventricle, cause bleeding, increase the fluid in the pericardial cavity, and produce an acute fatal tamponade.
(2) You may cause ventricular fibrillation with the tip of the needle.

Even so, in spite of these dangers, not aspirating the pericardium may be more deleterious than aspirating it, when there is tamponade.

N.B. The fluid from the pericardium may be heavily blood-stained and come out in a pulsatile manner, giving you the false impression you have entered the heart!

However with HIV, a pericardial effusion is so likely to be due to TB that *you should start treatment without aspiration*, particularly if ultrasound confirms a thickened pericardium.

RADIOGRAPHS. A *very large globular heart*, often with venous congestion. Depending on what is causing the pericarditis, you may see basal shadows in the lungs, or pneumonia obscuring the heart.

ULTRASOUND. You can easily recognize fluid around the heart. A thickened pericardium suggests a chronic process like TB, but not every case of pericardial effusion is tuberculous!

ECG. Tachycardia, usually sinus rhythm, a raised S-T segment (non-specific), an inverted T wave (late, non-specific), low voltage QRS complexes (highly suggestive).

TAPPING THE PERICARDIUM

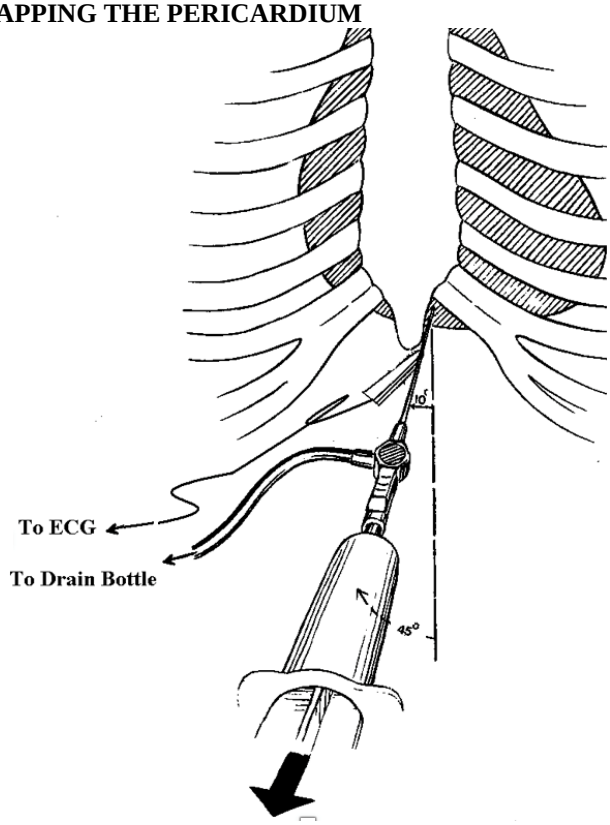


Fig. 9-4 ASPIRATING THE PERICARDIUM.
Insert the needle, attached to an ECG lead, in the epigastrium immediately to the left of the xiphisternum. With the patient propped up 45º, incline the needle horizontally and direct it 10º towards the left. In this way, if it does puncture the heart, it is more likely to meet the thicker left ventricle than the thinner right auricle.

DIFFERENTIAL DIAGNOSIS OF PERICARDIAL EFFUSION leading to tamponade is, in probable order of frequency:

Suggesting tuberculosis: a history of cough, bloody sputum, weight loss and chronic malaise, known HIV infection.

Suggesting malignancy: blood-stained fluid aspirated: *e.g.* Kaposi sarcoma.

Suggesting viral myocarditis: an influenza-like illness with generalized muscle pains. Early, you may hear a pericardial friction rub. Fluid may be blood-stained.

Suggesting a pyogenic bacterial cause: some other site of infection, such as pneumonia, meningitis, or measles with secondary staphylococcal infection. Often, there is some obvious site of infection, but not always.

Other causes of pericardial effusion that might cause tamponade include: uraemia, malignant deposits (only if they bleed seriously), collagen diseases, and the rupture of an amoebic abscess into the pericardium (rare).

These are some causes of a large heart *without* fluid in the pericardial cavity:

Suggesting rheumatic heart disease (common): valvular lesions; these are usually easily diagnosed by hearing heart murmurs.

Suggesting cardiomyopathy: an enlarged heart clinically and radiologically. The cardiac outline may be globular and closely simulate fluid in the pericardium; HIV status is often +ve.

Suggesting endomyocardial fibrosis (EMF): bilateral atrioventricular incompetence is usual with eosinophilia.

PREPARATION
Prepare as you would a theatre case using GA. Find two assistants, one to watch the ECG or the pulse, and ready to resuscitate if necessary, and another to fetch anything more that might be needed for resuscitation. Have the full resuscitation equipment available: laryngoscope, tracheal tubes, a sucker, oxygen, and an anaesthetic machine or an Ambu bag. Perform an ECG while you are aspirating, or, failing this, ask someone to feel the pulse continuously. Place the patient sitting comfortably at 45º; be sure to have IV access.

EQUIPMENT. A 16G (or 12G for thick pus) long cannula, a 3-way tap, and a 20 or 50ml syringe.

ASPIRATION (GRADE 2.3)
Infiltrate with lidocaine 2%. Attach the V-lead of the ECG to the cannula and insert this in the epigastrium immediately to the left of the xiphisternum. With the patient propped up at 45º, push the needle horizontally and direct it 10º towards the left (9-4). *(In this way, if it does prick the heart, it is more likely to meet the thicker left ventricle than the thinner right auricle.)*

If you can, aspirate under ultrasound guidance. Advance the needle slowly 1-2cm, aspirating frequently until fluid is withdrawn into the syringe. If the needle touches the heart, a sudden ST elevation will appear on the ECG monitor, and you will feel it knock against the needle.
Withdraw it a little and remove the needle, leaving the plastic cannula to drain the fluid. Repeat the ultrasound, if possible, to check if you have succeeded in draining all the fluid, or if it has re-accumulated.

CAUTION! **If there is a sudden deterioration with absence of a pulse:**
(1) Immediately remove the cannula.
(2) Clear the airway and ventilate with the Ambu bag.
(3) Start external cardiac massage at a rate of 30 beats to 1 ventilation.
(4) Administer 1mg adrenaline IV (and flush it through with saline) if there is no trace on ECG, or 50-100mg (2·5-5ml 2%) lidocaine IV if there is ventricular tachycardia (VT) or ventricular fibrillation (VF), or 0·3mg atropine IV if there is bradycardia. Continue cardiac massage. *(A defibrillator is useful, if available, for VT and VF.)*

If a normal heart trace does not return, administer further doses of drugs as required, and add 50ml 8·4% sodium bicarbonate. *Only when the situation is under control should you intubate and ventilate the patient mechanically.*

PERICARDIAL DRAINAGE (PERICARDIAL WINDOW) (GRADE 2.5)
If you aspirate pus, and it recurs, proceed to open drainage. Make a 4-5cm incision on the left side of the xiphisternum; incise the *linea alba* and proceed upwards in the extraperitoneal plane until you reach the pericardium. Put two stay sutures through the pericardium and lift this off the heart; then cautiously incise the pericardium, enlarge the hole and insert a Ch16 balloon catheter for thin pus and a Ch22 one for thick pus. Flush this with saline. Leave this draining for 2-6wks into a bag: no underwater seal is needed. If you leave the drain in long, it may erode the friable myocardium with disastrous results!

These patients have low cardiac output and usually need diuretics; dysrhythmias are common and often need IV lidocaine.

N.B. Recurrence of pyopericardium is common, especially if the pus is thick and looks like scrambled egg! Open drainage is simple and effective, but even then the pus may re-accumulate. In this situation a partial pericardiectomy through a left lateral thoracotomy, avoiding the phrenic nerve, is advisable, and is probably more effective, if you can arrange it.

9.3 Pus in the lung

Pus can collect in the substance of the lung as a result of:
(1) aspiration of vomit, especially after getting drunk or during anaesthesia,
(2) aspiration of pus, *e.g.* from a retropharyngeal abscess,
(3) inhalation of a foreign body, especially organic, such as a peanut,
(4) lateral spread from pneumonia, especially from *Staphylococcus aureus* or *Klebsiella pneumoniae* if multiple or *Streptococcus pneumoniae* if solitary in a lower lobe,
(5) lung contusion,
(6) bronchial obstruction by carcinoma,
(7) an infected pulmonary embolus,
(8) haematogenous spread especially in HIV.

Presentation is with coughing up copious amounts of foul sputum; there may be finger clubbing, and radiographs show a cavity with a fluid level. This may look like a tuberculous cavity if small, and a pyopneumothorax if large.

Pus, like everywhere else, must be removed, but *not* usually by incision. Postural drainage at physiotherapy is the most important treatment (11-24); use antibiotics to prevent spread of infection into the rest of the lungs. Drainage, however, may not be successful if the bronchus is blocked by a foreign body or carcinoma: it may be possible by bronchoscopy (29.14) to remove the former and biopsy the latter.

If there is a large lung cavity, especially peripherally placed, you can drain it through a rib resection (9-3) using a chest tube.

N.B. However, some lung abscesses, especially in children, need to be removed by pulmonary lobectomy.

10 Pus in the abdomen

10.1 Abdominal sepsis: peritonitis

Abdominal sepsis is a common and life-threatening complication following severe infection, necrosis, perforation or injury of abdominal viscera. Not infrequently, abdominal sepsis occurs after medical intervention: a laparotomy, an endoscopic procedure, peritoneal dialysis, a radiological investigation.

In the majority of cases the mainstay of treatment is the expeditious removal of bacteria and dead tissue; early recognition of the condition is therefore imperative. Abdominal sepsis occurs in the form of either generalized or localized peritonitis. One form can evolve into the other.

The common sites for localized peritonitis are (10-6):
(1) On the right side: subphrenic, subhepatic, ileocaecal (perityphlytic)
(2) On the left side: subphrenic, perisigmoid
(3) In the middle: intermesenteric, pelvic.
N.B. Localized accumulation of fluid in the peritoneum is usually referred to as an 'abscess'. This is a misnomer, because there is often no well-circumscribed wall containing the pus. Also, the nomenclature with regard to the anatomical sites of 'abscesses' is unsettled.

The area of the peritoneum is three times that of the skin, and is extremely porous to bacteria; thus septicaemia quickly ensues. So it is not surprising that the mortality from generalized peritonitis is at best 10%, even in good hospitals. Bacteria, both aerobic and anaerobic, can be released from the bowel when it perforates but also when it becomes inflamed or ischaemic. Bacteria can also come from an abscess, particularly the liver (15.10), or from the Fallopian tubes. When there is chemical contamination of the peritoneum from blood, stomach contents, bile, pancreatic juice, urine or cyst fluid, infection quickly follows. Traumatic or surgical intervention obviously allows a route for bacteria to contaminate the peritoneum; occasionally, especially in HIV+ve patients and cirrhotics, sepsis reaches the abdomen primarily from the bloodstream (primary peritonitis).

You can reduce the risk of death from peritonitis if you:
(1) Operate early, before the patient becomes very ill,
(2) Resuscitate adequately before operation, and
(3) Take the necessary precautions to minimize contamination of the abdominal cavity, when you perform a laparotomy. So handle the tissues gently, anastomose the bowel carefully, pack away potentially infected areas, control bleeding meticulously, and use appropriate prophylactic *pre-operative* antibiotics (2.9).

Peritonitis develops through several stages, which need different treatment:
(1) Disease in an organ before the overlying peritoneum is affected. For example, there may be the symptoms of peptic ulceration, appendicitis, cholecystitis or typhoid fever, but no involvement of the peritoneum. At this stage, you should be able to treat the underlying disease and prevent peritonitis.

(2) Localized peritonitis. With proper treatment, localized peritonitis may resolve. A mass may form, but the toxic effects of sepsis will be absent.

(3) '**Abscess' formation** around the organ responsible. Pus forms, but this is sealed off, not usually by a fibrous capsule from the rest of the abdominal cavity, but by loops of bowel and/or omentum which are stuck to one another by a fibrinous exudate. The mass is generally bigger than in stage 2 above, and is associated with toxic symptoms. It is still, if left untreated, likely to develop into spreading peritonitis (stage 4). Occasionally the abscess may be walled off entirely from the rest of the abdominal contents, or even be retroperitoneal (6.15). This abscess may however rupture and sepsis may then spread.

(4) Spreading peritonitis which may become generalized. Multiple collections of pus develop in the abdominal cavity, particularly in the pelvis and under the diaphragm. Later, all the abdominal organs become bathed in pus. If you operate and wash the pus out of the abdominal cavity, more collections may still form post-operatively.

Bowel which is surrounded by pus usually develops ileus (12.16): the bowel sounds become silent and the abdomen distends, as the bowel fills with gas and fluid. This fluid, and that which is lost into the abdominal cavity, depletes the circulating blood volume so the urine output falls and the pulse rate rises. As peritonitis advances, the peripheral circulation fails and shock results, but pain may diminish as ascites collects and dilutes the peritoneal irritation.

As always, but particularly with an acute abdomen, *a good history and a full examination are extremely important*: the commonest mistake is to leave out some of the essential parts of both. The history should suggest the diagnosis, and examination should merely confirm or refute it. Remember that your diagnostic success is proportional to the care you take getting the history, your keeping an open mind, and your thoroughness in your examination. When you decide to operate, *don't do so merely on the diagnosis of an 'acute abdomen'*, but on its most likely cause, with a list of possible alternatives, based on as much evidence as you can find. The early symptoms and signs will be more distinctive than the later signs, when generalized peritonitis has ensued. After the operation, if the diagnosis is a surprise, *think back to distinguish which features of the history were good clues*. *However, don't wait till you are certain of the diagnosis before you operate on a patient who needs surgery acutely!*

The common mistakes are:
(1) Not to ask the right questions properly and methodically.
(2) Not to examine the patient carefully and systematically, admit him and monitor him carefully and *to look at him again* if you are not clear of the diagnosis, or to see if the condition has worsened.
(3) Not to make and record a diagnosis and a differential diagnosis, especially if you are handing over to a colleague.
(4) To forget that many medical conditions, especially pneumonia (by causing diaphragmatic pain), can mimic an acute abdomen.
(5) Not to consider other, non-surgical conditions.
(6) To forget that age and sex can profoundly influence the probability of a particular diagnosis.
(7) Not to make adequate allowance for the late case whose history is obscured, whose mind is clouded, and whose signs are altered.
(8) To forget the 'silent interval' between the immediate chemical peritonitis and the delayed onset of bacterial peritonitis (13.3).
(9) To forget that signs you expect may be obscured in advanced peritonitis, septic shock, diabetes, HIV disease, aged patients and with steroid therapy.
(10) Finally, worst of all, not to go and see a patient with a suspected abdominal emergency immediately.
Base your diagnosis on as many items of information as possible. *The explanations given for a particular sign or symptom are suggestions only.*

HISTORY
Except in the very young, the very old, the demented, confused, psychotic and unconscious, PAIN is the cardinal symptom. Constitutional disturbances such as anorexia, nausea and vomiting, the inability to pass flatus in the presence of either constipation or diarrhoea and frequency of micturition are common. *In general, the patient with peritonitis is weak, thirsty, anorexic and nauseated.*

ONSET
(1) **"How did your pain start?"**

Waking at night	Probably serious
Start with an injury	Ruptured spleen
So severe that you collapsed or fainted	Perforated peptic ulcer Ruptured ectopic gestation Acute pancreatitis
Drinking much alcohol	Acute liver swelling Gastritis, pancreatitis

PAIN. Abdominal pain is usually the presenting symptom; its correct interpretation will lead you towards the cause. Expect it to have >1 component. *Severe pain lasting >6h is highly significant and demands you find out the cause!*
Look out for the *pattern* of pain:
(i) A sharp continuous pain due to inflammation of the parietal peritoneum.
(ii) An agonizing continuous pain due to ischaemia of the bowel.
(iii) A colicky pain is due to obstruction of either bowel, biliary tree or urinary tract: it comes in waves and spasms and often makes the patient move about restlessly.

Colicky pain may come before the pain of peritonitis, but is *not* the pain of inflammation.

Pain may also be referred from the diseased area to the other parts of the body that are derived from the same spinal segment. For example, pain from the gallbladder may be referred to below the right scapula; pus or blood under the diaphragm may present with pain in the shoulder.

There can be pain of more than one kind. For example, when the lumen of the appendix is obstructed, there is central abdominal pain of type (iii), but as soon as the peritoneum over it becomes inflamed, there is pain of type (i) in the right iliac fossa. If the appendix becomes gangrenous, there is ischaemic pain of type (ii).

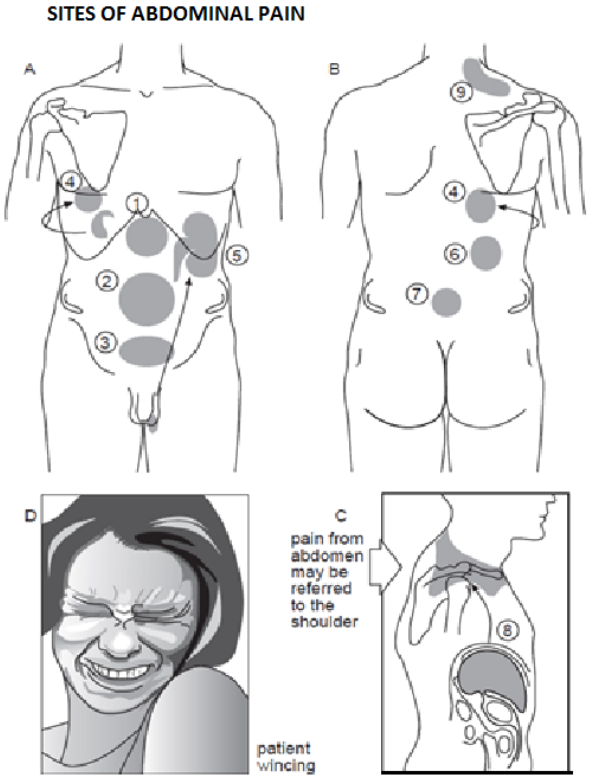


Fig. 10-1 THE SITES OF ABDOMINAL PAIN.
A, (1) lesions in the stomach, duodenum, gallbladder, and pancreas cause pain in the epigastrium. (2) lesions from the duodenum down to the middle of the transverse colon cause pain in the middle of the abdomen. (3) lesions from distal transverse to sigmoid colon cause pain in the lower abdomen. (4) gallbladder pain is primarily epigastric or in the right hypochondrium, but may be referred under the angle of the right scapula. (5) ureteric colic is frequently referred to the testicle on the same side. B, (6) kidney and pancreatic pain may be referred to the back. (7) uterine and rectal pain may be referred to the sacral area. C, (8) diaphragmatic pain is frequently referred to the shoulder. D, when you examine the abdomen for tenderness, *always* look at the patient's face: wincing is a very reliable sign of peritoneal irritation. *After Silen S. Cope's Early Diagnosis of the Acute Abdomen. OUP, 15th ed, 1979, Figs. 2,3 with kind permission.*

(2) **"Did the pain start suddenly?"**

Suddenly	Rupture of duodenal ulcer or bowel Torsion of testicle, bowel or ovarian cyst Mesenteric vessel occlusion Rupture aortic aneurysm

(3) **"Where is the pain and where did it start?"**

Epigastrium/umbilicus	Small bowel/appendix
Hypogastrium	Large bowel
All over abdomen	Perforated peptic ulcer Ruptured ectopic gestation Ruptured pyosalpinx
Loin & testicle *(retroperitoneal not causing peritonitis)*	Ureteric colic
Back	Ruptured aortic aneurysm Pancreatitis
Tip of the shoulder	Subphrenic/liver abscess Diaphragmatic pleurisy Ruptured spleen

(4) **"What is it like?"**

Throbbing/constant/burning	Inflammatory (*e.g.* appendicitis, salpingitis)
Burning/boring	(*e.g.* peptic ulcer, pancreatitis)
Intermittent with spasms	Colic

If it is colicky, how long do the spasms last, and is there complete relief between them?

(5) **"Has it moved?"**

Started in the umbilical region & moved to the right iliac fossa	Appendicitis
From the loin to the groin/testis on the same side	Ureteric colic

(6) **"What makes your pain better?"**

Lying absolutely still	Peritonitis
Walking bent forwards	Appendicitis, pancreatitis
Lying with knees flexed	Appendicitis, psoas abscess *(Inflammation in contact with the psoas muscle)*

(7) **"What makes it worse?"**

Coughing, sneezing, laughing, moving	Peritonitis

(8) **"Does it hurt to pass urine?"**

Dysuria	Urinary tract infection Pelvic abscess close to the bladder Inflamed appendix irritating the right ureter

VOMITING

Vomiting in the form of a single initial vomit is usual in most kinds of acute abdomen, so is little help to make a specific diagnosis. However, a few special features will give you some clues, especially with intestinal obstruction (12.2).

(1) **"Tell me about the vomiting."**

Started with pain but now less	Perforated peptic ulcer*
Severe and persistent	Strangulated small bowel
At the height of the pain	Acute pancreatitis Bowel, biliary, ureteric colic Torsion testis/ovarian cyst

** N.B. persistent vomiting is rare in such patients*

(2) **"What is the association between the pain and the vomiting?"**

Vomiting *before* the pain	Gastroenteritis
Vomiting sudden and soon after the pain	Strangulation or obstruction of proximal small bowel Cholecystitis
Vomiting c.4h after the pain	Distal small bowel obstruction Appendicitis Peptic ulcer
Pain but no vomiting	Salpingitis Tubo-ovarian abscess Haemoperitoneum Gastric volvulus Oesophageal perforation *(the cause is outside the bowel)* Large bowel obstruction

PREVIOUS HISTORY

(1) **"Have you ever had pain like this before?"**

Like the present but less severe	Intussusception Obstruction Appendicitis Torsion
When hungry relieved by food	Duodenal ulcer
Epigastric irregularly related to meals	Cholecystitis
Related to NSAID or steroid use	Gastritis Peptic ulcer

BOWELS

(1) **"Have you noticed any change in your bowel habit?"**

Usually regular, constipation for several days	Large bowel obstruction
Hypogastric pain & diarrhoea with mucus, then hypogastric tenderness & constipation	Pelvic abscess
Diarrhoea, colic, fever	Gastroenteritis
Bloody stools with mucus	Intussusception
Frequent bloody stools	Colitis
Worms	*Ascaris* obstruction

(2) **"When did you last pass a motion, and what was it like?"** Two or more stools may be passed after the onset of a complete small bowel obstruction. In complete low large bowel obstruction, no flatus or stools are passed.

MENSTRUAL PERIODS

(1) **"When was your last period? Was it before or after the normal time? Was the loss more or less than usual? Has there been any slight loss since your last period?"**

Last period late or scanty	Ectopic gestation
1-3 missed menstrual periods, followed by a small dark loss	Threatened miscarriage
Last period painful, dysmenorrhoea not usual	Salpingitis

CAUTION! Always ask the questions above with care. Asking "Are your periods normal?" is not enough.

OTHER SYMPTOMS

Poor appetite, weight loss, fever, general deterioration in health, change in girth	TB, HIV, malignancy
Severe illness with fever	Typhoid

Don't forget to ask about possible trauma. Vigorous massage of the abdomen may cause bowel injury!

GENERAL EXAMINATION FOR PERITONITIS

Abdominal tenderness, rebound tenderness and distension are the cardinal signs. There may be shortness of breath (which you may note simply by a difficulty in speaking), tachypnoea and cyanosis.

Tachycardia is common, as is sweating, whereby the extremities may be warm or cold. Fever is not always present, and infants as well as the aged may have hypothermia. There may be jaundice. Abdominal sepsis, almost invariably, if untreated, leads to septic shock.

(1) **The general condition** may be surprisingly normal especially early on in the disease. Later on, prostration and apprehension supervene. Look at the state of nutrition. If the patient is limp, lethargic, and slow to respond, suspect toxaemia, septicaemia, or shock. If he is restless, suspect cerebral hypoxia, due to hypovolaemia.

Look for a dry tongue and lack of skin turgor (dehydration), pale conjunctivae (anaemia), mouth signs of HIV disease (5.6), and a sickly sweet breath smell (ketones). Look at the unpigmented skin for blotchy purplish discolouration (shock).

(2) **The face** may be characteristic in advanced disease.

Nose tip & earlobe and hands cold	Hypovolaemia Peripheral circulatory failure
Mildly grey	Perforated peptic ulcer Acute pancreatitis Strangulated bowel
Deathly pale with gasping respiration	Severe bleeding (*e.g.* ectopic gestation)
Gaze dull and face ashen	Severe toxaemia
Eyes sunken, tongue and lips dry, skin elasticity reduced	Dehydration Intestinal obstruction

(The Hippocratic facies is a combination of all of these; any one of these may deteriorate and look worse!)

(3) **The pulse** may be normal early on. An increase in the pulse rate is important in deciding if the abdominal condition is serious, especially with an abdominal injury. Tachycardia is usual in peritonitis, and early in strangulation of the bowel. The pulse of typhoid fever is no longer slow after the ileum has perforated.

(4) **The patient's attitude in bed** is characteristic: lying still and only changing position in bed with pain and difficulty, keeping the hips and knees flexed, fearing to cough, sneeze or move. If he is constantly moving around, straight one minute and doubled up the next, he has colic and *not peritonitis*.

(5) **The respiration** will show minimal movement of the abdomen; later there is shallow and grunting respiration, and in shock it is rapid and shallow. If, in a child, the rate is twice normal, and the *alae nasi* are flaring, pneumonia is likely. *Don't forget to listen to the chest*.

(6) **The temperature** is raised; if it is not, the inflammation is early or there is pure intestinal obstruction. Severe fever from the onset may occur in typhoid, basal pneumonia, or pyelonephritis.

THE ABDOMEN. Ask the patient to point to where the pain started, to where it is now, and to where it is worst.

(1) **Look at the abdomen.** Is its contour normal? Look for distension due to gas or fluid: test for shifting dullness. Initially in peritonitis the abdomen is tympanitic, later it will fill with ascitic fluid.

Does the abdomen move freely on breathing? Ask the patient to inhale and exhale fully and cough: inability to do this properly indicates peritoneal irritation. Peritonitis anywhere may splint all or part of the abdomen, and stops the normal movement that accompanies breathing.

Reduced movement in the lower abdomen suggests PID or appendicitis.

Visible peristalsis means there is obstruction not paralytic ileus, and so no peritonitis.

Expose the whole abdomen including genitalia and look at the groins: there may be an obstructed irreducible hernia. Are there any old operation scars?

N.B. Don't miss the Pfannenstiel scar in an obese woman or the subumbilical laparoscopy scar!

(2) **Feel the abdomen.**

The great Hamilton Bailey (one of Britain's foremost surgical teachers, 2.10) used to examine the abdomen by kneeling by the patient's bedside: copy him!

First relax the abdomen by flexing the hips. If necessary, ask an assistant to support the flexed knees. If extending the hips causes abdominal pain, this is a reliable sign of peritonitis or psoas abscess. Tap the abdomen with your fingers: if this causes pain, you don't need to hurt the patient further by pressing harder!

Lay your hand flat on the abdomen, and keep your fingers fully extended as you feel for tenderness. Your hand must be warm, gentle, patient, and sensitive. You may win a child's confidence by examining him using his own hand or with your hand on top of the sheet, or secreted under the bedclothes, or even with him sitting on his mother's lap. Use light palpation first to test for muscle rigidity and spasm (guarding), and localize the tenderness. Then, if necessary use deep palpation.

Wincing (10-1D) on pressure of the abdomen is a very reliable sign of peritonitis and *you will miss it if you don't look at the patient's face!* If peritonitis is advanced, *there is no need to test for rebound tenderness*; it is cruel and unhelpful. If the abdomen is rigid like a board, this is proof of generalized peritonitis, especially that due to a perforated peptic ulcer. But if peritonitis is localized, rebound tenderness is a good indication as to which parts of the peritoneum are involved and which are not, but by itself, *it is not a very reliable sign.* Often light percussion is better.

Avoid the painful area, and start abdominal palpation as far from it as you can. (*Don't worry if the patient tells you it is the wrong place!*) Move towards it slowly. Find where the area of greatest tenderness is. It will be easier to find if there is no generalized guarding, and is a useful clue to the organ involved, *e.g.* the right iliac fossa (appendicitis), or the right hypochondrium (cholecystitis, liver abscess).

N.B. A doughy feel suggests chronic tuberculous peritonitis, especially if ascites is present.

CAUTION! A patient may show very little rigidity if:
(i) He is HIV+ve and has a neuropathy, or is very old.
(ii) The peritonitis is advanced and ascitic fluid dilutes the peritoneal irritation: *so as he gets more ill, the tenderness diminishes!*
(iii) He is anyway very toxaemic and ill.
(iv) She is a woman who is pregnant, or whose abdominal muscles are stretched after delivery.
(v) He has been given narcotic analgesia, especially post-operatively, or is paraplegic with a sensory level at the 10th thoracic vertebra (T10) or above.
(vi) He is taking steroid medication.
(vii) He is diabetic.
(viii) The perforation occurred about 6h before, so that the immediate rigidity has had time to go, and secondary bacterial peritonitis has not yet had time to develop.
(ix) He is very fat and flabby, and the muscles are thin and weak.
(x) Pathology is confined to the pelvis, retroperitoneum, or high under the diaphragm.

N.B The cause of peritonitis does not relate to the intensity of the pain.

Look for superficial induration and tenderness in the abdominal wall (pyomyositis, necrotizing fasciitis).

(3) **Can you feel any masses?** In the right iliac fossa this may be a tuberculoma, an appendix mass, amoebiasis, a mass of *ascaris* worms, a lymphoma; in the right hypochondrium it may be an empyema of the gallbladder or a liver abscess; in the loin a pyonephrosis; in the pelvis a full bladder, adnexal mass or enlarged uterus. Faeces in the bowel is compressible, but may appear as a fairly solid mass. A tender mass adjacent to the midline (haematoma from rupture of the epigastric artery) will not disappear when the patient tenses the abdominal wall (Fothergill's sign).
N.B. In very severe pain, a moderate dose of opioid may:
(i) reduce the pain,
(ii) make for more comfort and improve getting a good history, and
(iii) prevent immediate guarding of the abdomen whenever you touch it, so that the physical signs become more localized and the cause clearer.

(4) **Listen to the abdomen** for decreased or absent bowel sounds: you may need to wait for 3mins! High-pitched tinkling bowel sounds, coinciding with worsening of abdominal pain, are a sign of obstruction *not peritonitis*.
'Diminished' bowel sounds mean nothing specific!

(5) **The *iliopsoas* test** is only indicated if the patient is not very ill, and does not have generalized peritonitis. Get him to lie on the opposite side to where the pain is, and extend the thigh on the affected side to its fullest extent. If this is painful, there is some inflammatory lesion near the psoas muscle (appendix abscess, iliac abscess, pyomyositis of the iliopsoas). *This test is not useful if the anterior abdominal wall is rigid.*
(6) **The *obturator* test.** If rotating the flexed thigh so as to stretch this muscle causes pain, there is pus or perhaps a haematocoele (in a woman) in contact with the surface of the *obturator internus*.
(7) **The *thoracic percussion* test.** Percuss gently with your fist over the lower chest wall. On the right, a sharp pain indicates an inflammation around the diaphragm or liver (often present in acute hepatitis); on the left, inflammation around the diaphragm, spleen, or stomach.
(8) **Percuss for liver dullness** in the right nipple line from the 5th rib to below the costal margin. If liver dullness is absent, there is probably free gas in the abdominal cavity.
(9) **Don't forget the hernial orifices**. Feel both femoral and inguinal openings, the umbilicus, and any old incisions.

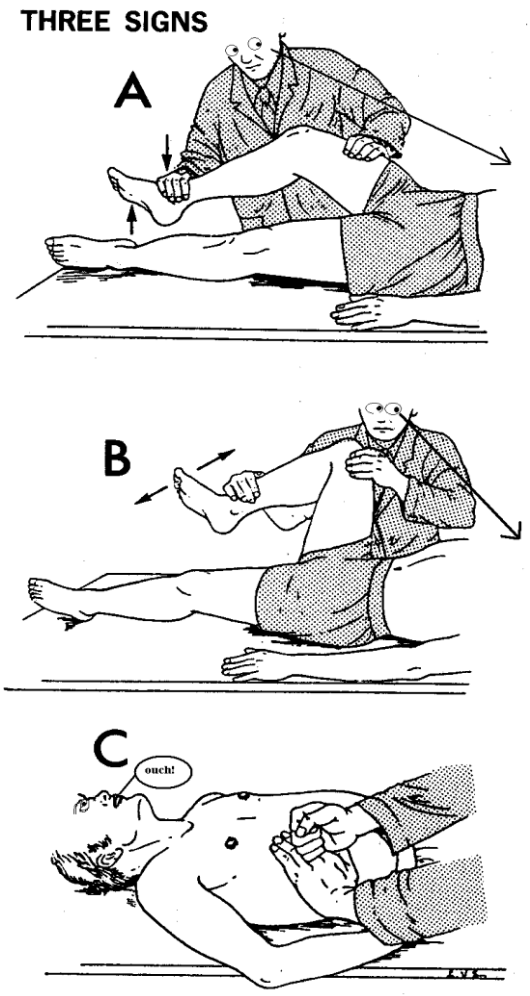


Fig. 10-2 THREE SIGNS.
A, iliopsoas test. Ask the patient to flex the hip against the resistance of your hand. If he feels pain, there is inflammation in relation to the psoas muscle. B, obturator test. Flex the hip to 90º and gently rotate it internally and externally. If this causes pain, there is inflammation in relation to the obturator muscle. C, fist percussion test. Percuss gently with your fist over the chest wall. On the right, a sharp pain indicates an inflammatory lesion of the diaphragm or liver; on the left, one of the diaphragm, spleen, or stomach. *Kindly contributed by Jack Lange.*

CAUTION!
(i) An obstructed hernia is usually tense, tender, or painful but does not have to be so.
(ii) It may be small, especially if it is a femoral hernia, <2cm diameter.
(iii) The patient may be quite unaware of it.
(iv) *Femoral hernias are very easy to miss in fat patients*.
(v) *Don't overlook a small umbilical hernia lying deep in fat, or think a lump is not a hernia because the symptoms are not very acute*.
(vi) Ask if the hernia has not been pushed back in recently: there may be obstruction from 'reduction en masse', *i.e.* without relief of strangulation.
(vii) In a baby, it is not the bulging inguinal hernia which will strangulate, but the small slim one containing only a thin loop of the tiny bowel. It may only feel like slightly thickened cord and testicle, with reddening and oedema of the scrotal skin (18.5).

(10) **The pelvis** is just as important as the abdomen. You will find a vaginal examination often more useful than a rectal examination (except in a child). Do both. *Never forget to examine the rectum.* Lay the patient in the lateral position. Pass a well-lubricated finger as far up the anal canal as it will go. Feel for tenderness in all directions. Feel forwards, in a man for an enlarged prostate, a distended bladder, or enlarged seminal vesicles; and in a woman for swellings in the pouch of Douglas or displacements of the uterus. Feel upwards for a stricture, the apex of an intussusception, or the bulging of an abscess against the rectal wall. Feel laterally for the tenderness of an inflamed swollen appendix. Feel bimanually for a pelvic tumour or swelling, or for any fullness in the pouch of Douglas. Look if there is blood or mucus on your glove afterwards.

OTHER SYSTEMS
Don't forget to listen to the chest; there might be a basal pneumonia, pleural effusion or empyema. Examine the spine (spinal tuberculosis or a tumour can cause root pain felt in the abdomen). Feel for a stiff neck (meningitis can cause vomiting and abdominal pain). Look at the testes to exclude torsion.

SPECIAL TESTS
Get a full blood count: *N.B. in untreated HIV+ve patients, the white count may not be raised even in severe peritonitis*. Check the blood or urine for sugar, red cells and white cells. Check urea and electrolytes if there is dehydration or the urine output is poor, if you can, and the amylase if you suspect pancreatitis. If there is septicaemia, do blood cultures and check the clotting time. If the albumin is very low, this is a bad sign.

ULTRASOUND is possible at the bedside, and is the investigation of choice: it will show fluid accumulation and tissue necrosis, and usually allows immediate aspiration of pus, proving the diagnosis and providing a specimen for bacteriological examination.
Occasionally you can remove all the pus in an abscess by means of aspiration; this is more reliable, however, if you can position the tube for drainage and irrigation under ultrasound control.
You can also diagnose free fluid, recognize para-aortic lymphadenopathy, ectopic gestation, abdominal masses, and note any inflamed thickened structures, as well as an aneurysm. *However, beware of trying to diagnose appendicitis exclusively by ultrasound! Note, also, that an ultrasound will not easily show if an aneurysm has ruptured.*

RADIOGRAPHS must be good, because you are interested in the disposition and pattern of gas. Be selective, and look at the films yourself. *There is no point in ordering radiographs* (*except* of the chest) *if you have already made a diagnosis of peritonitis and have decided to operate*. An erect chest film is best to see gas under the left hemi-diaphragm (and also to look at the condition of the lungs). It may also show a pleural effusion or empyema.

If the patient is too sick to sit up, you may see free gas under the abdominal wall in a lateral decubitus film of the abdomen.

Signs on abdominal films (erect and supine) are subtle:
(1) Air in the small bowel: this is always abnormal except in a child <2yrs. You may see gas *both inside and outside* the bowel wall, though (Rigler's sign, which is a sign of bowel perforation), and air on the lateral border of the liver, outlining its edge clearly.
(2) Displacement of the colon: a ruptured spleen may displace the shadow of the splenic flexure downwards and medially.
(3) Obliteration of the psoas shadow: this can be caused by pyomyositis of the psoas, a psoas abscess from a tuberculous spine, a retroperitoneal abscess or by bleeding from an injured kidney.
(4) Multiple small bowel fluid levels, identical in radiographic appearance to small bowel obstruction: occurs in longstanding paralytic ileus.
(5) Urinary calculi: look for these along the lines joining the tips of the transverse processes of the vertebrae to the sacroiliac joints.
(6) A faecolith in the area of the appendix, when there are good symptoms and signs of appendicitis, confirms the diagnosis.
(7) Gas in the portal vein (you see the outline of the veins *peripherally* in the liver: from mesenteric ischaemia, portal pyaemia, or severe colitis *(this is a real danger sign)*).
(8) Gas within the wall of the bowel or the gallbladder (this implies necrosis or ischaemia).
CAUTION! There may still be a perforation without gas under the diaphragm (especially early on), and in closed bowel obstruction with strangulation, there may be no fluid levels.

LAPAROSCOPY. If you cannot make a diagnosis, think of using a laparoscope (19.5) to help.

Don't rely on the findings unless you are very experienced with this technique. You may easily miss some relevant pathology. On the other hand, you may, having initiated an operation under GA, be persuaded unnecessarily to remove a normal appendix.

A. GENERALIZED PERITONITIS

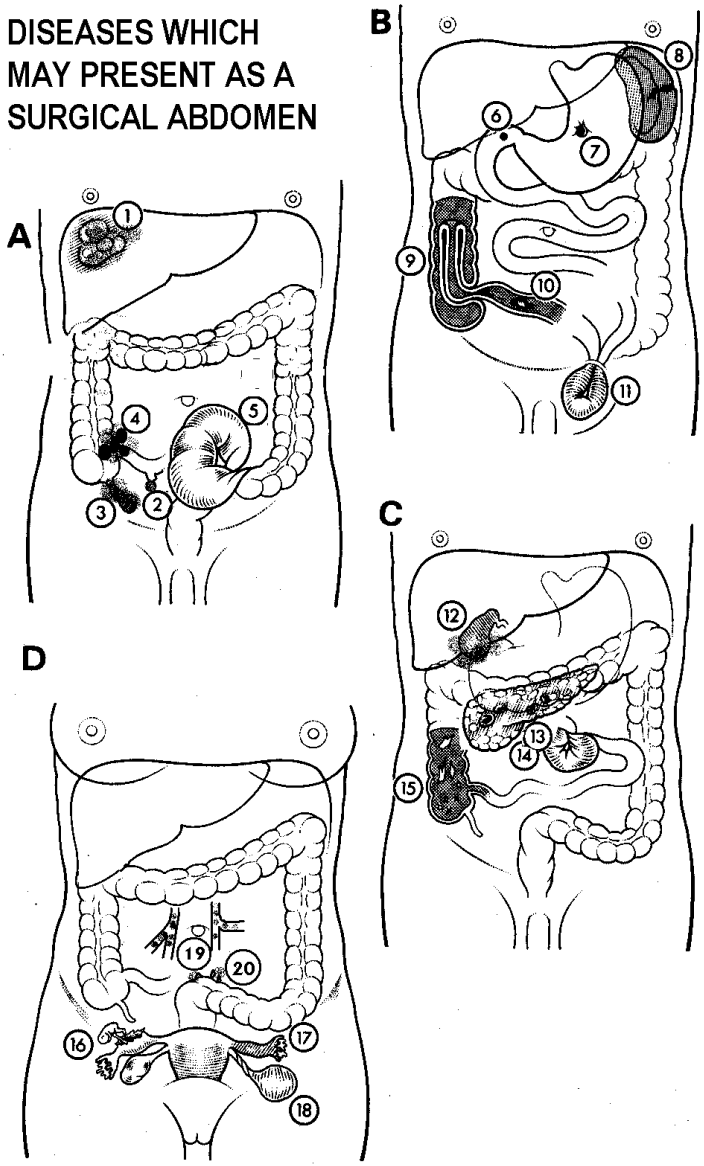


Fig. 10-3 DISEASES WHICH MAY CAUSE PERITONITIS.
A, (1) liver abscess. (2) perforation of Meckel's diverticulum. (3) appendicitis. (4) ileocaecal tuberculosis. (5) sigmoid volvulus. B, (6) perforated duodenal ulcer. (7) perforated gastric ulcer. (8) ruptured spleen. (9) intussusception. (10) perforation of terminal ileum (typhoid or HIV). (11) strangulated groin hernia. C, (12) empyema of gallbladder. (13) pancreatitis. (14) volvulus of the small bowel. (15) amoebic colitis. D, (16) rupture of an ectopic gestation. (17) PID. (18) torsion of an ovarian cyst. (19) mesenteric thrombosis. (20) diverticulitis.

DIAGNOSING PERITONITIS

How are you going to diagnose all the many causes of peritonitis, if the pattern of the symptoms they produce is so similar? Here is a checklist of the more important features of each to help you sort them out, in order of their frequency.

As is usual in medicine, a patient is more likely to have a rare presentation of a common disease, than a common presentation of a rare one. Be familiar with the pattern in your own area. "Common things are common, and most people get what most people get." If you think there is peritonitis, but are not sure, the list below may help you.

Don't be frightened by its length.
Concentrate on the *pattern* of symptoms and signs.

CLUES TO THE DIAGNOSIS OF PERITONITIS
(the diagnoses are listed in general order of frequency):

CENTRAL ABDOMINAL PAIN:
- Small bowel obstruction/strangulation (12.3)
- Appendicitis (14.1)
- Acute pancreatitis (15.13)
- Primary or tuberculous peritonitis (16.1)
- Colitis
- Necrotizing enterocolitis (14.4)
- Bleeding intestinal Kaposi sarcoma (5.6)

CENTRAL ABDOMINAL PAIN AND SHOCK:
- Ruptured ectopic gestation (20.6)
- Perforation of small bowel (14.3)
- Small bowel volvulus (12.8)
- Acute haemorrhagic pancreatitis (15.13)
- Mesenteric thrombosis (12.14)
- Dissecting/leaking aortic aneurysm (35.8)

CENTRAL ABDOMINAL PAIN AND SHOCK, AND RIGIDITY:
- Perforated peptic ulcer (13.3)
- Perforated small bowel (14.3)
- Perforated gall bladder (15.4)
- Ruptured oesophagus (30.7)

LOCALIZED PAIN AND TENDERNESS
(the causes depend on where these are found):

In the right hypochondrium:
- Leaking duodenal ulcer (13.3)
- Liver abscess (15.10)
- Acute cholecystitis (15.3).
- Empyema of gallbladder (15.4)
- Amoebiasis (14.5)

In the epigastrium:
- Leaking duodenal ulcer (13.3)
- Acute pancreatitis (15.13)

In the left hypochondrium:
- Splenic infarct of sickle cell disease (15.17)
- Ruptured spleen
- Leaking gastric ulcer (13.3)

In the right iliac fossa:
- Acute appendicitis (14.1)
- PID (23.1)
- *Ascaris* mass (12.5)
- Ileocaecal TB (16.6)
- Chronic ectopic gestation (20.7)
- Torsion ovarian cyst (23.9) or testis (27.25)
- *Amoebiasis* (14.5)
- Typhoid (14.3)
- Iliopsoas abscess (6.16)
- *Angiostrongyliasis* and *oesophagostomiasis*
- *Actinomycosis*

In the hypogastrium:
- Septic uterus after delivery (22.14)
- Pelvic abscess (10.3)
- Cystitis (27.36)

In the left iliac fossa:
PID (23.1)
Intussusception and strangulation (12.7)
Chronic ectopic gestation (20.7)
Torsion ovarian cyst (23.9) or testis (27.25)
Diverticulitis (14.2)

In both iliac fossae:
Septic miscarriage (23.2)
Puerperal sepsis (22.14)

In the abdominal wall:
Pyomyositis (7.1)
Necrotizing fasciitis (6.23)
Haematoma

RENAL CONDITIONS can sometimes present as an acute abdomen.
Renal colic produces a sharp severe colicky pain spreading from the loin down to the groin, vomiting, a vague diffuse tenderness in the flank. Reflex intestinal ileus is not uncommon (27.13).
Pyonephrosis or retroperitoneal abscess produces a high fever, pain in the costovertebral angle, a tender enlarged loin mass, often with toxaemia (6.15).

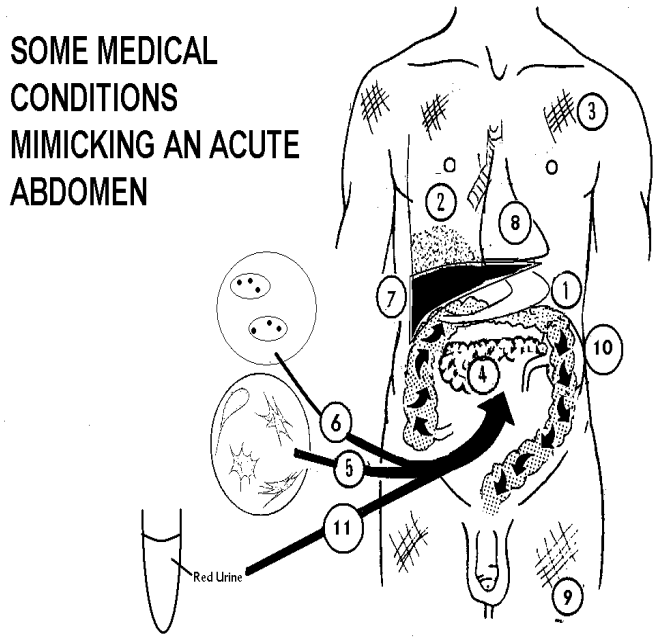


Fig. 10-4 SOME MEDICAL DISEASES MIMICKING AN ACUTE ABDOMEN.
(1) acute gastroenteritis and gastritis. (2) basal pneumonia and pleurisy. (3) viral myalgia. (4) diabetic precoma. (5) sickle cell crisis. (6) malaria. (7) chlamidial perihepatitis. (8) myocardial infarction. (9) brucellosis. (10) uraemia. (11) porphyria.

SOME MEDICAL DISEASES may mimic an acute abdomen. In most of them the fever is higher, the general symptoms worse, and the abdominal symptoms less evident than in the surgical acute abdomen. But *beware of peritonitis when the patient is so ill that the general signs predominate over local surgical signs*.

(1) **Acute gastroenteritis:** diarrhoea, vomiting and fever, colicky pains, minimal abdominal tenderness, hyperactive (but not obstructive) bowel sounds, fever early, perhaps with rigors.

(2) **Acute gastritis:** copious vomiting, severe epigastric pain doubling up the patient, usually associated with *helicobacter*, an alcoholic binge or dietary indiscretion. This is particularly common in the Caribbean after ingestion of *Ciguatela* fish.
(3) **Basal pneumonia and pleurisy:** early high fever, cough, rapid breathing, spasm of the upper abdominal muscles, and tenderness. Signs of consolidation on chest radiography, usually in the right lower lobe. Abdominal pain and rigidity may be very marked in a child, and involve the whole of the upper half of the abdomen, or the whole of one side.
(4) **Chlamydial perihepatitis** (Curtis-FitzHugh syndrome): pain in the right upper quadrant, and vaginal discharge.
(5) **HELLP syndrome** (haemolysis, leucocytosis and low platelets) in pre-term pregnancy, often associated with pre-eclampsia, may cause stretching of the liver capsule and so pain in the right hypochondrium.
(6) **Viral myalgia** (Bornholm disease): sudden onset with high fever, local or general abdominal and chest pain; marked superficial muscle tenderness and rigidity of variable intensity, quickly changing its position; tender intercostal muscles on one or both sides; lateral compression of the chest is painful; nausea but seldom vomiting, no chest signs. Often during an epidemic of 'flu'.
(7) **Diabetic precoma:** slow onset of abdominal pain and vomiting, dehydration, sugar and ketone bodies in the urine and breath.
(8) **Sickle cell crisis** caused by a hypoxic trigger factor (such as a chest infection): vomiting, central abdominal pain, guarding frequently, rigidity sometimes, sickle test +ve, with headache, a high fever, and pains in multiple sites, especially the limbs and back. Bowel sounds remain present.
(9) **Myocardial infarction:** sudden collapse with hypotension, sweating, chest and left arm pain especially in a smoker classically associated with ECG changes of ST depression.
(10) **Malaria** may cause diarrhoea, vomiting and abdominal pain; fever is usually present; look for splenomegaly.
(11) **Uraemia** may simulate ileus by causing abdominal distension and vomiting. The signs and the history are vague and variable.
(12) **Porphyria** is a hereditary condition found in specific areas (*e.g.* Ethiopia, Ghana, South Africa) often made worse by drinking alcohol, by anaesthetics (*e.g.* thiopentone) or sulphur drugs, where the urine goes red and sun-exposed skin blisters; abdominal pain is not associated with rigidity, but with emotional outbursts and polyneuropathy.
(13) **Brucellosis:** fever, headache, fatigue, bone and joint pains in herders of goats and sheep. Gastrointestinal symptoms are variable.
(14) **Polyserositis** is one of a number of rare familial conditions (typically found around the Mediterranean) which presents with fever and peritonitis. You may only diagnose this after you have done a laparotomy and found no obvious cause! The best treatment is with colchicines.
(15) **Angio-oedema** is an allergic reaction (usually with prodromal minor episodes of swelling of lips, ears or throat) which can cause intestinal oedema. If associated with

urticaria and itching, you can use adrenaline and salbutamol to reduce the oedema. *Beware! Surgery may precipitate an even worse reaction resulting in death by asphyxia.*
(16) **Snake bites**, particularly krait in India, may not cause local pain, but give rise to severe cramps before producing neurotoxic effects.

DIFFICULTIES DIAGNOSING PERITONITIS
If you are in any doubt about the diagnosis when you first see a patient, admit him. *Then come back repeatedly to re-examine him*, feel the abdomen and monitor him carefully, if necessary every hour. He will be easier to assess in the ward than in the outpatient or casualty department. You are also likely to get a more reliable reading of the pulse and temperature. This is especially important if you suspect strangulated bowel, appendicitis, or a peptic ulcer. The diagnostic use of a single opioid dose may be helpful: if he feels much better after one dose of opioid and no longer has any signs of peritoneal irritation, it is very unlikely that anything serious is going on. This is very useful if you think he might be hysterical. There is a small chance it is the Münchhausen syndrome (exhibited by a clever group of patients, *including medical personnel*, who persistently fake their symptoms).

Decide if he needs an operation, and if so, when? However, if he is no better, or in fact worse, peritonitis is likely. *If he deteriorates, operate*. *Don't leave a patient like this if you hand over to a colleague without giving a very clear picture of his condition.* This is where recording of your findings is most important. Often, when you have made the diagnosis, all you will know before you operate is that there is peritonitis, without knowing why. Try to establish how advanced it is from the history and the signs. A laparotomy is usually mandatory and even if you are in doubt, *most patients who are young will not suffer untowardly from a negative laparotomy, but they will die from neglected abdominal sepsis.*

BETTER A SCAR ON THE ABDOMEN THAN A SCAR ON YOUR CONSCIENCE

As you will see below, there are some special indications for non-operative treatment. However, if you are uncertain about these in a particular case, it is wiser to operate than not to do so. It is more important to decide when to operate and when not to operate, than the exact diagnosis.
"If in doubt, it is better to look and see than to wait and see."

But, *don't operate if the only symptom is pain and there are no abnormal signs, radiographs and lab results whatsoever*.

N.B. Don't delay operation on a pregnant woman with peritonitis because you fear for premature delivery. The need to act quickly is even more vital: if you delay, the toxaemia may well kill the baby, or even the mother!

INDICATIONS FOR OPERATION, *after adequate resuscitation*, are:
(1) Diagnosis of peritonitis.
(2) Diagnosis uncertain, and condition deteriorating after 4h of active management.
(3) Diagnosis uncertain after a routine laparotomy, but signs suggestive of peritonitis or condition deteriorating.

INDICATIONS FOR DRAINAGE:
(1) Localized collections of pus identified by ultrasound.
(2) Pelvic abscess (10.3).

INDICATIONS FOR NON-OPERATIVE TREATMENT:
(1) Acute pancreatitis (15.13).
(2) Acute cholecystitis (15.2).
(3) Abdominal mass with resolving peritoneal irritation.
(4) A typhoid perforation of slow onset showing no signs of deterioration (14.3).
(5) Necrotizing enterocolitis (14.4).
(6) Medical conditions giving rise to abdominal pain.

N.B. Note that in these medical causes of an acute abdomen, there is rarely abdominal guarding present.

RESUSCITATION. The need for this varies:
If the pulse is rapid, there is postural hypotension and especially if the blood pressure is low, delay operation for a few hours (preferably ≤4h) for proper resuscitation. Use Ringer's lactate or 0·9% saline IV (provide an adult 1L per day of illness). Be sure to correct potassium deficiency. If possible, measure the CVP and keep it at 6-8cm of water. Pass a nasogastric tube and *don't allow oral intake*. Monitor the urine output hourly and keep a fluid balance chart. Catheterize the bladder. Start gentamicin, chloramphenicol or a cefalosporin *and* metronidazole. (*Note gentamicin may prolong activity of relaxants, and is a hazard in renal impairment.*) Improve hypoxia by administering oxygen by a mask. Operate as soon as the pulse rate falls, the blood pressure rises, and the peripheral circulation improves. Resuscitation should not take >6h.

If the CVP remains low, continue rapid intravenous fluid infusion! *If signs of peripheral circulatory failure don't respond to generous resuscitation, death may occur despite all your efforts*. Dopamine and noradrenaline infusions need ICU care.

If there is confusion, severe hypotension, and hyperventilation, with a fast pulse, and warm pink extremities, or cold clammy ones, this is septic shock. The patient may be so sick that you should do the minimum just to save his life while you proceed with resuscitation and antibiotics. This is the so-called '**damage control laparotomy**'. If you can, drain the septic focus. Timing is important: the patient must be fit enough to withstand the operation, so overcome shock, and then do the simplest possible operation.

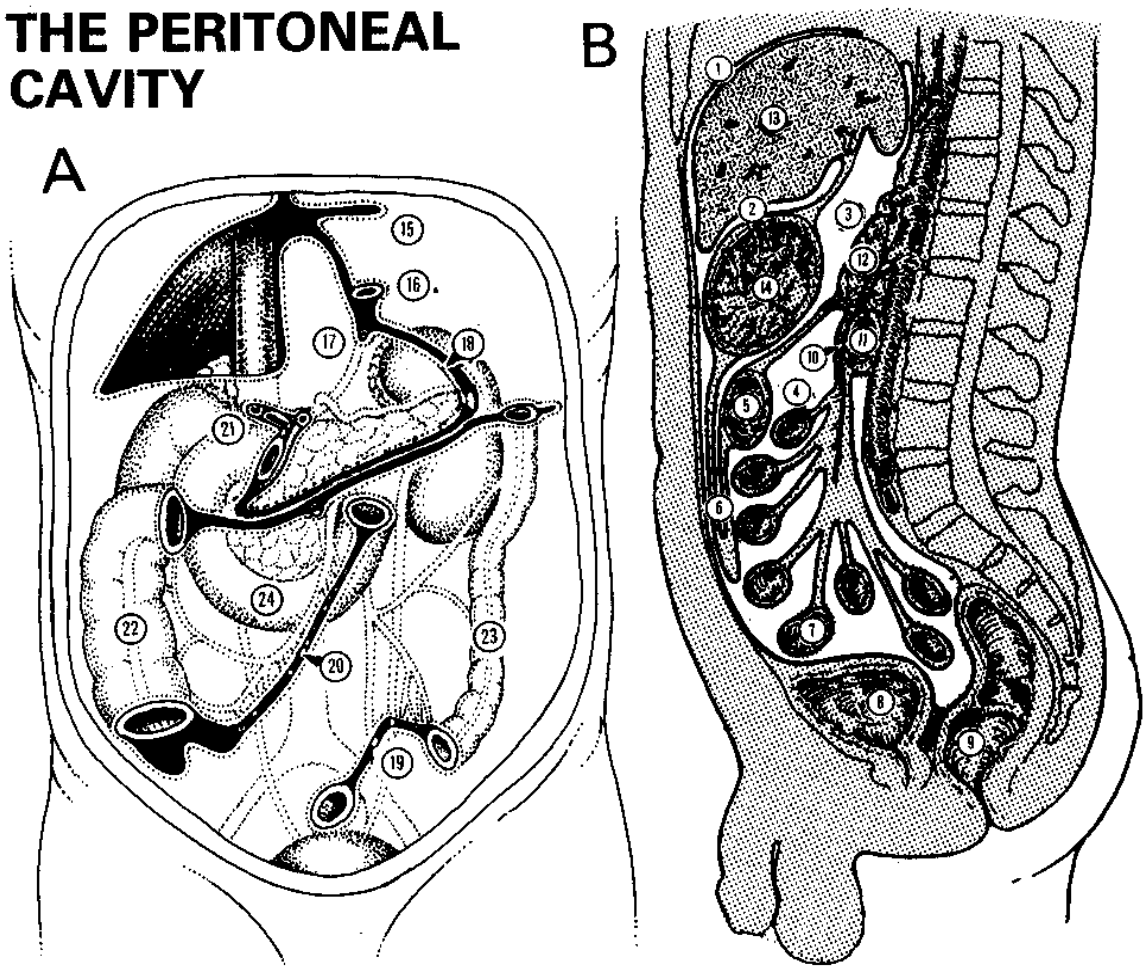


Fig. 10-5 THE ABDOMINAL CAVITY in generalized peritonitis fills with pus. A, posterior abdominal wall showing the lines of peritoneal reflection after removal of the liver, spleen, stomach, jejunum, ileum, and the transverse and sigmoid colon. Organs on the back of the abdominal wall are seen through the posterior parietal peritoneum. B, longitudinal section of the abdomen: (1) anterior superior subphrenic space. (2) subhepatic space. (3) lesser sac. (4) coils of jejunum. (5) transverse colon. (6) greater omentum. (7) coils of ileum. (8) bladder. (9) rectum. (10) mesenteric artery. (11) duodenum. (12) pancreas. (13) liver. (14) stomach. (15) left triangular ligament of the liver. (16) oesophagus. (17) upper recess of the omental bursa (lesser sac). (18) lienorenal ligament. (19) root of the sigmoid colon. (20) root of the mesentery. (21) cut edge of the lesser omentum. (22) ascending colon. (23) descending colon. (24) duodenum.

This may be just inserting drains into the flanks under LA, to allow pus to drain and give you the opportunity to wash out the abdomen. The condition may then improve sufficiently to perform a laparotomy much more safely later.

LAPAROTOMY FOR PERITONITIS (GRADE 3.2)

PREPARATION. This is very important: check everything is ready (11.1-2).

PHILOSOPHY. The primary objective of the operation is to remove all dead tissue (necrosis, pus, clots, fibrin, faeces etc), to clean the abdominal cavity and to prevent leakage and further accumulation of noxious materials. *Repair of intra-abdominal anatomy and of the abdominal wall are desirable but not the primary objectives of the emergency operation.*

INCISION

As soon as the patient is draped and anaesthetized, and the abdomen is relaxed, *palpate it*. A mass may appear under GA which was quite impalpable before! Make a midline incision or, particularly in children, a transverse incision. If there is already an incision, go through this if it gives you good enough access. It is usually best to cut out the previous scar.

ENTERING THE ABDOMEN

Aim quickly to discover what is wrong: *this may not be obvious*. Be careful and observant, learn to recognize what you see, and search thoroughly.

SMELL can tell you a lot. If a puff of gas greets you as you open the peritoneum, the bowel has probably perforated. In advanced sepsis, especially from the pelvis, a bad smell may be from gas-forming organisms.

If there is an abnormal smell, it may be:
(1) acrid (perforated peptic or typhoid ulcer),
(2) faeculent (ruptured large bowel),
(3) characteristic of anaerobes (suppurative appendicitis),
(4) putrid (gynaecological sepsis), or
(5) urine (from an intraperitoneal rupture of the bladder).

LOOK FOR FLUID in the abdominal cavity, which may be:
(1) **purulent fluid of various thickness**
(appendicitis, salpingitis, perforated peptic ulcer, primary peritonitis, mesenteric adenitis, including tuberculous, diverticulitis)
(2) **foul, turbid brown fluid**
(peritonitis from appendicitis or sigmoid volvulus, ruptured ovarian cyst, ruptured liver abscess)
(3) **blood**
(ectopic gestation, injured liver, spleen, or mesentery, ruptured ovarian lutein cyst)
(4) **pale straw-coloured fluid**
(intestinal obstruction without strangulation, mesenteric adenitis, tuberculous ascites, cirrhosis)
(5) **blood-stained**
(ischaemic bowel, torsion of ovarian cyst, *streptococcal* peritonitis, acute pancreatitis)
(6) **bile-tinged**
(perforated stomach, duodenum or small bowel)
(7) **frank bile**
(ruptured gallbladder, injured liver)
(8) **watery, reddish-brown, offensive fluid**
(strangulation with incipient gangrene)
(9) **clear colourless fluid**
(ruptured hydatid cyst)
(10) **porridge-like material**
(ruptured dermoid cyst)
(11) **sticky mucous fluid**
(ruptured ovarian cyst)
(12) **urine**
(ruptured bladder, divided ureter)

If there is any exudate, send it for culture, if you can.

If the peritoneum is fiery red with flakes of fibrinous exudate, this is severe peritonitis. If these flakes are clear or nodular, they are probably due to tuberculosis. Firmer nodules are from metastatic carcinoma. Fatty flakes, which may look very much like tuberculosis, are from severe pancreatitis.

BREAK DOWN ADHESIONS with the greatest possible care. Break down light ones with your fingers. If they are dense, define them carefully, and cut them with scissors. If you are rough, you may drastically worsen the peritonitis by adding more organisms to the bacterial soup already present *and* you increase the chances of a faecal fistula, which may be a disaster.

If you do make a hole in the bowel, isolate and cover it with a swab, clamp the bowel either side of the hole with non-crushing bowel clamps, and carry on. *Don't waste time at this stage by repairing the perforation:* do this after you have freed all the bowel.

You may need to sacrifice an impossibly matted segment of bowel (11.3): this is better than causing a lot of bleeding trying to free it and using up a great deal of time with a sick patient on the operating table. When faced with bowel that is very stuck, approach it from a normal segment on both sides, and try to massage bowel content out of the affected segment and hold it empty between non-crushing bowel clamps. If then you do perforate it, you won't spill its contents.
Pack away the rest of the abdomen with swabs, so if you do spill content, you contaminate the peritoneal cavity as little as possible. Always lift up bowel from behind with your fingers, *never pull it!* If a loop is hopelessly stuck in the pelvis where you can't see it properly, you may be able to pinch it off and, whilst still holding the bowel wall tight in your fingers, deliver it out of the abdomen. This way, although you will certainly have to repair the perforation (11-5) or resect that piece, you will have avoided spilling bowel contents in an uncontrolled manner. Occasionally, you will be able to lift a whole clump of bowel out of the abdomen, and be able to work on it outside, whilst packing away the rest of the incision.

If thickened oedematous omentum is adherent to something, it points to the site of acute inflammation, strangulation (12.2), or abscess formation (14.1,2). *Take care you don't damage a viscus by pulling off the omentum*: this may actually be sealing off a perforation. Think carefully if it is best left alone; if necessary, cut away the omentum with the damaged viscus.

MINIMIZE THE RISK OF SEPSIS
(1) If you have to open a hollow organ, or a septic collection, pack the abdominal cavity round it with moist swabs. If it is walled off from the rest of the abdominal cavity and this is unaffected by sepsis, just drain it and leave the remaining abdomen alone or you will spread infection into a clean peritoneal area.
(2) Handle an inflammatory mass carefully: *don't let it burst and discharge pus everywhere*.
(3) If an area does become contaminated, wash it out.
(4) *Insert drains only when indicated* (4.9).

DECOMPRESS THE BOWEL (12.4) because this will:
(1) give you a better view to see what you need to do next,
(2) reduce the risk of rupturing the bowel if its wall is thin,
(3) help you close the abdomen at the end of the operation, and reduce the risk of the *abdominal compartment syndrome* (11.10),
(4) greatly reduce the risk of post-operative vomiting and aspiration,
(5) reduce the risk of any bowel leaking by reducing the volume of fluid passing any anastomosis you make.

N.B. Generalized peritonitis, particularly of some days' duration, will always have distended loops of bowel present.

If the bowel is already open, suck its contents out through the perforation using either a Poole's or Savage sucker; or else, mobilize the bowel out of the abdominal cavity, and drain the contents into a bowl holding the open bowel end with Babcock's forceps.

This is messy, but as long as you take care to avoid spillage of contents into the abdominal cavity, it is extremely worthwhile.

If the bowel is not open, you can decompress its content either by (a) massaging contents proximally towards the stomach and suctioning via a wide-bore nasogastric tube, or (b) clamping an appropriate segment of bowel, making a small hole on the anti-mesenteric border of the bowel, and decompressing it through that, as above.
Do (a) if the bowel distension is relatively slight, you have good suction and an experienced anaesthetist who knows how to suction the mouth if there is spillage there.
Do (b) if there is serious bowel distension with thick bowel content, your suction machine is unreliable, the anaesthetist is inexperienced, or the patient is *a child with an uncuffed endotracheal tube.* You may hesitate to open bowel if it is not already opened. Do so, if otherwise closure of the abdomen would in any way be difficult: the advantages far outweigh the disadvantages!

EXAMINE THE ABDOMINAL CAVITY
If peritonitis is widespread, search systematically until you can find its cause. You should know where this might be from:
(1) The history.
(2) The nature of the exudate.
(3) The place where pus and exudate are most intense.
(4) The density of the adhesions; the densest ones may indicate the origin of the infection.

CAUTION!
(1) Suck out all free pus before you start.
(2) You must have good exposure.
(3) If you find localized pus, try to minimize its spread around the peritoneum!
(4) You face the dilemma described above: when to divide adhesions and when not to.

Inspect the abdominal cavity thoroughly, unless you are certain from the onset that sepsis is localized to one area. Inspection of the entire cavity may be difficult because of adhesions. All necrotic tissue must be removed; this may entail resection of bowel.
You need:
(1) adequate exposure: make the incision big enough,
(2) a good light focused in the right place: get assistance or use a head-light,
(3) good retraction: you can't operate on your own,
(4) a good view: position the patient in the best way, *e.g.* Trendelenburg for pelvic sepsis, and pack away the bowels *(but count the packs and don't forget them inside at the end of the operation!).*

MANAGING THE UNDERLYING CAUSE
First, you will have to find it, *and this may not be easy*. Let the smell and nature of the fluid guide you. Go first for your pre-operative diagnosis. Look for signs of inflammation (pus or adhesions), of perforation, or tumour.

If you are faced with something you are not sure how to handle, unscrub and refer to this text, or get an assistant to do so: *you don't need to pretend you know it all!* For this reason, keep this book *in theatre!*
Some places are difficult to see. If the source of sepsis is not obvious, look: in the lesser sac, under the liver, behind the duodenum (mobilize it using the Kocher manoeuvre, 13.5), and behind the colon (especially both flexures) and rectum.
Play safe: the patient is desperately sick, and *you must not risk complications*. Remember *'damage control laparotomy'*. The decision how to proceed depends on the condition.

If the patient is still in shock, is hypothermic, acidotic or has signs of coagulopathy, terminate the operation in the most expeditious way. This may entail the sealing of all holes or intestinal ends by using, for instance, umbilical tapes and leaving the abdomen open. *Physiology takes precedence over anatomy.* The best place to correct physiology is the ICU. Try to finish emergency surgery within 45mins.

N.B. Don't remove the appendix 'prophylactically'.

LAVAGE
If there is generalized peritonitis, lavage the abdominal cavity.
If the peritonitis is localized, pack off the affected area and then lavage or mop out the infected space. Sometimes, you can safely wash out only the pelvis.
Tip in several litres of *warm (c.30ºC)* fluid, slosh it around with your hand, and suck it out until the fluid which returns is clear. You may need 8-10L; usually 3-4L are enough. Wash out the upper abdomen, the sub-diaphragmatic and sub-hepatic spaces, the paracolic gutters, the infracolic area, between all the loops of bowel and the pelvis. *You don't need to use saline*: 10L of warm sterile water is preferable to 500ml of saline!
If there is much adherent septic fibrin on the bowel, liver, spleen and omentum, only peel away the parts that come away easily. Otherwise you may damage the viscera or bowel, simply in a vain attempt to make the abdomen look clean!

DRAINS. You cannot drain generalized peritonitis and multiple intra-abdominal collections, because the area to be drained is too large and the drains block anyway: so, wash out the pus vigorously. It may, however, be appropriate to drain a retroperitoneal collection, *e.g.* in the pelvis (vaginally in a female, or rectally in a male) using a Penrose drain (4.9).
Drains have limited use (11.8): if you do use them, insert a soft tube drain with several perforations cut in it on low suction, and irrigate it with liquid to keep it open.
Place the drain away from bowel and vessels, protecting these with omentum; bring it out away from a wound by the shortest route.

If the intra-abdominal tension remains high, *don't attempt to close the abdomen!*

Leave the abdomen open as a *laparostomy* (11.10) if:
(1) you expect to have to look inside again within 48h, *e.g.* if you are uncertain about an anastomosis, or the vascularity of bowel, or if you had to leave a pack inside, or if you have limited your intervention because of severe metabolic disturbance (damage control),
(2) there is gross faecal soiling or sepsis requiring repeated lavage,
(3) there are multiple bowel fistulae,
(4) there is severe haemorrhagic necrotizing pancreatitis,
(5) there is more tissue necrosis which you were unable to remove,
(6) you simply cannot close the abdomen without tension.

N.B. Your hands may smelly horribly after cleaning out a septic abdomen, even wearing 2 pairs of gloves: no amount of soap or perfume will remove the odour, but washing with coffee will!

CLOSURE. Confirm all the routine checks are done (1.8). *Then, not before*, close the abdomen with continuous through-and-through sutures of stout monofilament nylon, polydioxanone, poliglecaprone or steel deep to the skin (11.8). For skin, use subcuticular absorbable or nylon. *You should not have to pull the abdominal wall together with tight tension sutures!* Go back and decompress the bowel.

POST-OPERATIVE CARE AFTER LAPAROTOMY FOR PERITONITIS

N.B. The post-operative care is just as important as the operation itself!

Checklist:
(1) **Nasogastric suction.** If there is generalized peritonitis, ileus is sure to follow; suction will reduce the distension, although it will not reduce its duration. You may suck out 2-6L fluid daily. Replace the fluid loss with IV 0·9% saline or Ringer's lactate in addition to the standard requirements.
(2) **Intravenous fluids.** For maintenance an adult needs at least 3-4L in 24h: use 1-2L 0·9% saline, or Ringer's lactate, and 2L 5% dextrose in 24h.
(3) **Bladder drainage.** You need to monitor the urine output (if possible 2hrly for the first 48h). Use a Paul's tubing (condom catheter) in a young male; remember catheterization is invasive and potentially hazardous.
(4) **Fluid balance.** Keep an accurate fluid balance chart till the fluid balance is stable (at least for 48h, usually 3-6 days). The common error is to not infuse enough fluid and not chart the fluid input and output properly. If the initial fluid resuscitation was inadequate, there may still be a deficit to make up. Infuse sufficient IV fluids to keep the urine output >1ml/kg/hr.
(5) **Potassium supplements.** *Don't forget these*, especially if there is a large volume of gastric aspirate. Start them when the post-operative diuresis begins.
(6) **Blood.** If there was major blood loss during the operation (>2L), especially if previously anaemic and this loss was not replaced, check the Hb level; if it is <80g/L, transfuse 2 units of blood.
(7) **Acidosis.** There are several ways you can correct this:
(i) Treat with 200ml of 8·4% sodium bicarbonate (200mmol), or with 500ml of 4·2% sodium bicarbonate (250mmol).
(ii) Treat with 1l $^{1}/_{6}$molar lactate.
(iii) Infuse adequate IV fluids and let the kidneys correct the acidosis.
If the condition is poor, use (i) or (ii), and repeat daily.
(8) **Antibiotics.** If there was generalized peritonitis, continue the same antibiotics you used pre-operatively for 3-5 days. Look at the clinical response, rather than by the sensitivities reported by the laboratory. If there is no improvement after 2 days, consider re-laparotomy rather than changing the antibiotics.
(9) **Chest exercises.** Patients after laparotomy, especially for peritonitis, tend to have shallow respiration. To prevent pneumonia, provide adequate amounts of analgesics and encourage deep breathing exercises with physiotherapy. Serious cases need mechanical ventilation. As soon as you can, get the patient out of bed.
(10) **Mobilization.** Mobilize *early*, even from the 1st day post-op, to prevent thrombo-embolic and respiratory complications as well as bed sores (34.16).

OTHER MEASURES. Review the charts carefully each day for complications. Watch the temperature chart, the general state of alertness, the respiratory rate, the abdominal girth, the bowel sounds, the urine output and ask whether the patient passed flatus or stools. Start feeding early. Look if the wound is soiled or smelly.

POST-OPERATIVE COMPLICATIONS are many, and may occur after any laparotomy (11.9,15); so be especially aware of:
(1) Abdominal sepsis (may lead to septic shock).
(2) Wound infection.
(3) Urinary infection.
(4) Pneumonia.
(5) Malnourishment.

If there is NO IMPROVEMENT, there may be residual sepsis and you should arrange another **('second-look')** laparotomy. Intra-abdominal sepsis is an extremely difficult diagnosis to make, particularly post-operatively, and you will often wish you had made it earlier. *Be very gentle* because the bowel will be friable and oedematous: *don't use sharp dissection*. Direct your attention to the source of the problem, rather than randomly extracting loops of bowel and dividing adhesions unnecessarily. Re-open a patient with severe generalized peritonitis *routinely* after 48h to:
(1) lavage residual sepsis,
(2) detect any missed pathology,
(3) examine the viability of the bowel,
(4) check bowel anastomoses for patency,
(5) check for walled off abscesses in all far and deep corners of the abdominal cavity,
(6) prevent abdominal compartment syndrome (where the intra-abdominal pressure is >20mm Hg) (11.10).
In very severe cases, particularly where fistulae are present, and to decompress the abdominal compartment, leave the abdomen open as a *laparostomy* (11.10).

If sepsis persists, a 3rd or even 4th laparotomy is necessary, but with diminishing chance of success. Remember to change the antibiotics you are using after the 2nd re-laparotomy.

If there is NO IMPROVEMENT but no sign of fever, there may be HIV disease: test for this because you need to know how far you are likely to succeed in this case. Make sure you don't fail to treat tuberculosis.

If the ABDOMEN DISTENDS and the volume of the gastric aspirate remains high (maybe with vomiting), either the normal short period of ileus is continuing, or the bowel is obstructing (12.15).

If there is pain, a fever and a leucocytosis, suspect leakage of a bowel anastomosis, iatrogenic or spontaneous bowel perforation in HIV. This is much more likely than bleeding inside the abdomen. *Don't hesitate to re-open the abdomen for a second look.*

If serous fluid discharges from the wound, dehiscence is imminent. *Don't wait for a complete 'burst abdomen'*; return to theatre for closure of any residual defect with interrupted sutures.

If the abdominal wound becomes infected and breaks down (11.13), inspect it bd. If there is localized redness only, treat with antibiotics, but when a discharge or abscess develops, remove sutures, open the wound generously, wash it and *pack* it daily with diluted betadine.

If fever persists, there may be a post-operative wound, chest or urinary infection, deep vein thrombosis or there is further intra-abdominal sepsis.
If there is a mass which was not present previously, get an abdominal radiograph: it may be a retained swab! With HIV there may be a fever without any specific known cause.

If there is diarrhoea, especially with the passage of mucus, suspect a pelvic abscess (10.3). This may be due to an anastomotic leak.

If bowel content discharges from the wound or a drain, this is a fistula (11.15). If this is upper small bowel fluid (thin yellow), it may produce disastrous fluid and electrolyte losses and severe wasting.
Divert the effluent if possible, especially by suction. However, if the bowel is not obstructed distally to the fistula, and the output is <500ml/day, it should close spontaneously. Use low-pressure suction to keep the fistula wound dry, and make sure feeding continues and you correct potassium losses.
Unless you can drain a localized septic collection in the abdomen properly under ultrasound guidance, perform a laparotomy with all the same requirements as for generalized peritonitis.
If you find the collection walled off, or extra-peritoneal, drain it without contaminating the rest of the abdominal cavity. *Don't try to drain it blind from the outside or via the rectum or vagina.*

B. LOCALIZED PERITONITIS

Localized septic collections (these are rarely true abscesses) in the abdominal cavity can be the result of:
(1) Generalized peritonitis: they are one of its major complications.
(2) Some primary focus of infection, such as appendicitis or salpingitis.
(3) An abdominal injury in which the bowel was perforated or devitalized.
(4) Infection of a residual haematoma.
(5) Leaving a foreign body (*e.g.* a swab or faecolith) behind.
(6) Peritoneal dialysis.
(7) Any laparotomy.
Fever has a characteristic spiky pattern (10-8). Prostration with anorexia, weight loss, and a leucocytosis are the norm, but may not be if there is HIV. If loops of the bowel pass through the abscess, they may become obstructed, acutely or subacutely (12.3).

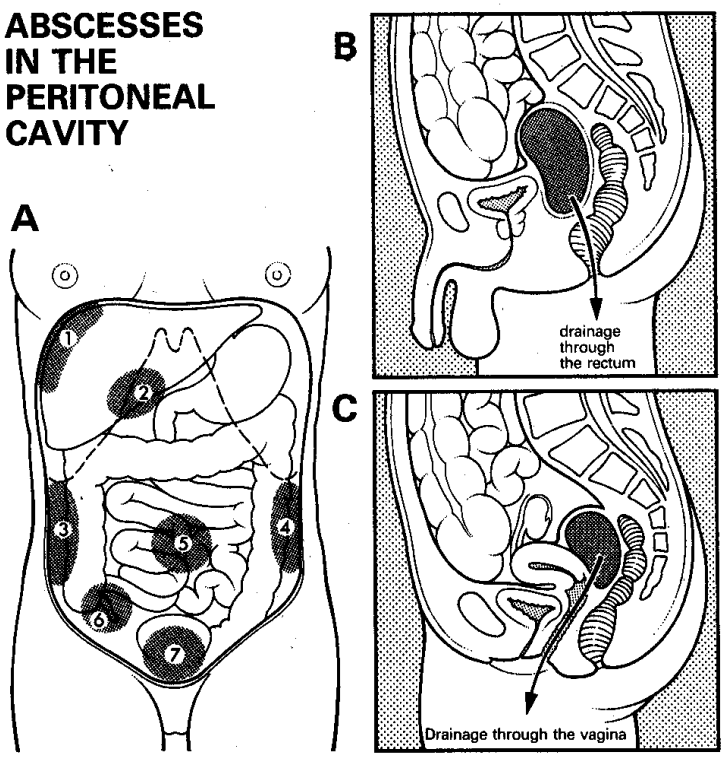


Fig. 10-6 SEPTIC COLLECTIONS IN THE ABDOMINAL CAVITY. A, common sites: (1) between the diaphragm and the liver. (2) under the liver. (3,4) in the right and left paracolic gutters. (5) among coils of the bowel. (6) around the appendix. (7) in the pelvis. B, pelvic abscess pointing into the rectum. C, pelvic abscess pointing into the vagina.

10.2 Subphrenic abscess

Pus under the diaphragm has usually spread there from somewhere else in the abdomen. A subphrenic abscess may be secondary to:
(1) Peritonitis, either local or general, particularly following a perforated peptic (13.3) or a typhoid ulcer (14.3), or appendicitis (14.1), or PID (23.1) or infection following Caesarean section (21.13).
(2) An injury which has ruptured a hollow viscus and contaminated the abdominal cavity.

(3) A laparotomy during which the abdominal cavity was contaminated (10.1).
(4) A ruptured liver abscess (15.10).

Suspect that there is a subphrenic abscess if there is a deterioration, or recovery followed by deterioration, 1-3wks after a laparotomy, with a low, slowly increasing, swinging fever, sweating, and tachycardia. This, and a leucocytosis (unless there is HIV disease), show that there is 'pus somewhere', which is causing anorexia, wasting, and ultimately cachexia. If there is no sign of a wound infection, a rectal examination is normal, and the abdomen is soft and relaxed, the pus is probably under the diaphragm.

The pus might be between the diaphragm and the liver, in the right or left subphrenic space, or under the liver in the right or left subhepatic space in the lesser sac. There may be pus in more than one of these spaces.

Ultrasound is easily the best tool to diagnose and drain such a collection of pus (38.2); if you can't do this, explore the abdomen on the suspicion that there might be a subphrenic abscess; the difficulty is knowing where to explore. If you fail to find pus, you will probably do no harm; missing a subphrenic abscess and doing nothing is far worse. If it is anterior, you can drain it by going under the costal margin anteriorly. If it is posterior, you can pass through the bed of the 12^{th} rib posteriorly.

SIGNS AND SYMPTOMS
Thoracic signs are more useful than abdominal signs.
Ask or look for:
(1) Cough.
(2) Shoulder-tip pain on the affected side.
(3) An increased respiratory rate, with shallow or grunting respiration.
(4) Diminished or absent breath sounds.
(5) Dullness to percussion.
(6) Dull pain.
(7) Hiccup (rare).
(8) Tenderness over the 8^{th}-11^{th} ribs.

A subhepatic abscess may cause tenderness under the costal margin *anteriorly*. A subphrenic abscess, pyelonephritis, pyonephros or perinephric abscess can all cause similar tenderness *posteriorly*. If the patient is thin and the pus is superficial, you may feel a tender indurated mass under the costal margin in front (right subphrenic space), in the right flank (right subhepatic space), or posteriorly.

ULTRASOUND. A fluid collection is easily seen around the liver, which remains homogeneous in appearance (38.2b). You can use the ultrasound to guide you to drain the abscess.

CHEST RADIOGRAPHY will only be helpful to make the diagnosis if you can screen the diaphragms; you may find a pleural effusion, or collapse or consolidation at one lung base.

CAUTION!
(1) The white count is usually raised but may be normal.
(2) 10% of patients have no fever.
(3) *Don't try to diagnose subphrenic abscesses blind by aspiration*. This is dangerous and misleading: do it with ultrasound guidance.

KIMANI (15yrs) was admitted with abdominal pain and vomiting of sudden onset, about 4h previously. He had shoulder-tip pain, but he also said he had pain when he put the tongue out, so it was first thought that he might be hysterical. He had no abdominal signs, so he was admitted for observation. The following day the abdomen started to distend, and aspiration of the abdominal cavity withdrew greenish fluid. A laparotomy was done, and an ulcer on the greater curve of the stomach was found and repaired. Initially he recovered well, but as he was about to go home 10 days later, he was not well, he ran a fever, he looked toxic, and there was tenderness and induration on the right side of the upper abdomen. He was suspected of having a subphrenic abscess, the abdomen was reopened through a midline incision, and a large quantity of foul-smelling pus was evacuated from under the right side of the liver, after which he eventually recovered.
LESSONS (1) If you are not certain that a patient is hysterical, it always pays to observe him. (2) Beware of the 'latent interval' 3-6h after a perforation, when there may be few abdominal signs. (3) Use the ultrasound if you have one to make the diagnosis. (4) You may be able to drain a subphrenic abscess through the original laparotomy incision, but the incisions described below may be better. (5) When a peptic ulcer causes general peritonitis, a thorough lavage of the abdominal cavity is as important as the repair.

DIFFERENTIAL DIAGNOSIS includes:
(1) Liver abscess (15.10),
(2) Empyema (9.1),
(3) Pulmonary collapse (11.11).

MANAGEMENT OF SUBPHRENIC ABSCESSES (GRADE 3.2)

WHICH APPROACH?
If you suspect a subphrenic abscess, and the general state does not improve, and the fever does not settle, prepare to drain the abscess. Try to do this with ultrasound guidance, if necessary on several occasions. Otherwise, especially if the pus is too thick, the collection does not resolve, or keeps recurring, explore the abscess! Avoid antibiotics which may mask the symptoms.

If there is a swelling, or oedema, or redness or tenderness just below the ribs or in the loin, make the incision there.

If the abscess follows appendicitis, a perforated duodenal ulcer, or cholecystitis, it will probably be on the right. If a high gastric ulcer has perforated, it is more likely to be on the left. If an ulcer in the posterior wall of the stomach has perforated, there will be pus in the lesser sac.

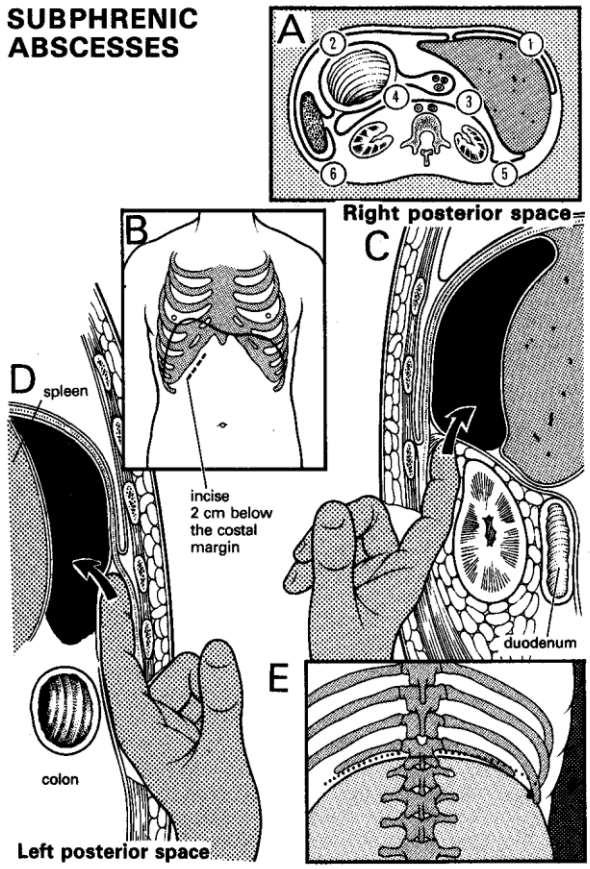


Fig 10-7 SUBPHRENIC ABSCESSES.
A, spaces where pus can collect under the diaphragm: (1) right anterior subphrenic. (2) left anterior subphrenic. (3) right subhepatic. (4) left subhepatic (lesser sac). (5) right posterior subphrenic. (6) left posterior subphrenic. B, anterior approach: make a subcostal incision. C, explore the right posterior subphrenic abscess. D, explore the left posterior space. E, explore the posterior spaces through the ribs.

If you don't know which side it is on, there is about a 75% chance that it will be on the right, probably anterior. Approach it anteriorly, if possible through the old laparotomy wound, unless there are very clear signs that it is posterior. If one route fails, try another. You cannot readily reach the posterior surface of the liver through an anterior incision, or the anterior liver surface through a posterior incision, so use the anterior approach by preference, and the posterior approach only if you are certain pus is there, or you have found none anteriorly.

ANAESTHESIA. If GA is risky, block the lower 6 intercostal nerves. *Don't hesitate to explore the abscess* because the patient seems too weak for surgery!

ANTERIOR APPROACH. Preferably re-open the previous laparotomy incision; otherwise make a subcostal incision which is large enough to take your hand, a finger's breadth below and parallel to the right (usually) or the left costal margin. Cut from the middle of the rectus muscle laterally (10-7B). Cut the muscle fibres in the line of the incision. This way you can often drain the septic collection without entering the general abdominal cavity.

If you have entered through the previous incision, beware of adhesions, go carefully, and pack off the rest of the abdomen before you come to the abscess which you will find by noting tissues adherent to each other. Sweep your finger gently above the liver from one side to the other to explore the subphrenic space. If you don't find pus, sweep your finger laterally and explore below the liver. Break down any loculi, and send pus for culture. Insert a drain. If the liver is not adherent to the diaphragm, there may still be pus posteriorly, pushing the liver forwards.

CAUTION!
(1) *Try not to enter above the diaphragm*. This is more likely to happen with an anterior approach. If you enter the pleura, lavage the thoracic cavity thoroughly, especially if you have spilled pus inside it, then close the diaphragm with a #1 suture and insert an underwater seal drain. If you have not yet found the pus, make sure the diaphragm is well closed before you approach the abscess.
(2) Be sure that there is only one abscess.

If you damage the pleura accidentally, close it and insert an underwater seal drain.

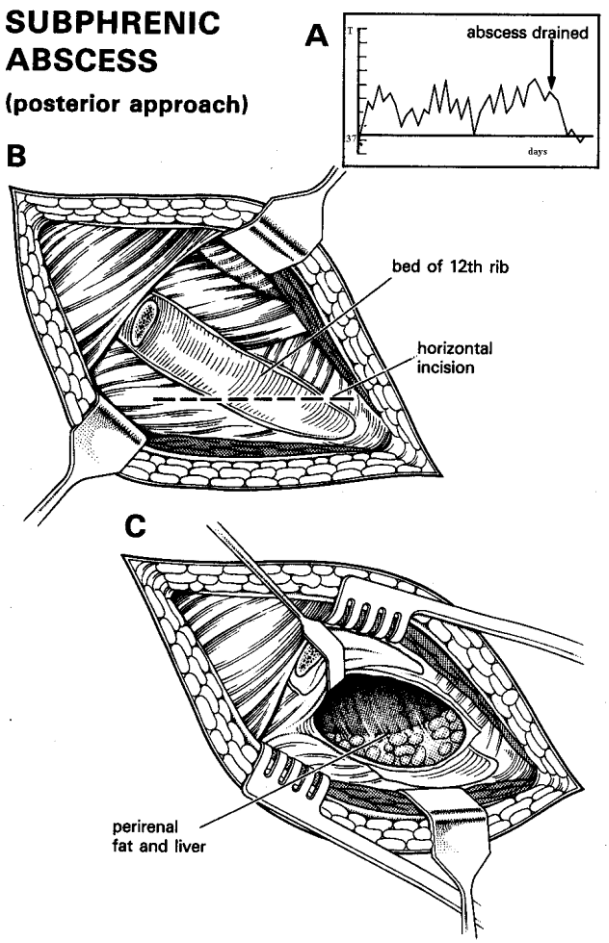


Fig. 10-8 POSTERIOR APPROACH TO A SUBPHRENIC ABSCESS. A, temperature chart: a hectic fever subsided after the abscess was drained. B, excise the 12th rib and make an incision in its bed. C, divide the bed of the 12th rib, showing the liver and the fat round the kidney. *After Ochsner A, Graves AM. Subphrenic Abscess: An Analysis of 3,372 Collected and Personal Cases. Ann Surg. 1933;98(6):961-90.*

POSTERIOR APPROACH. Lay the patient on his sound side with the lumbar region slightly elevated by breaking up the table or placing pillows under the opposite side. Make an incision which is big enough to take your hand over the 12th rib posteriorly (10-7E, 10-8). Remove the distal ⅔ of the 12th rib; divide it at its angle. Cut through the periosteum, reflect this from the whole circumference of the bone with Farabeuf's rougine, as for an empyema (9.1). Occasionally, you may need to tie the intercostal vessels.

CAUTION! Take great care not to damage the diaphragm. Incise the inner aspect of the periosteum horizontally. Push your finger upwards and forwards above the renal fascia to enter the abscess (10-7C,D).

If pus ruptures into a bronchus in a spasm of coughing, death from asphyxiation may result unless you institute urgent postural drainage (11.12). The pus is more likely to have spread from an amoebic liver abscess than from a subphrenic abscess.

If the subphrenic collection is recent after laparotomy, it may not have walled off, and be associated with other intra-abdominal collections. In this case, re-open the original laparotomy incision, aspirate all the abscess cavities, and lavage thoroughly with warm water. *Don't insert drains.* Plan a repeat laparotomy if the sepsis was severe.

10.3 Pelvic abscess

Pus in the pelvis is nearly as dangerous and difficult to manage as pus under the diaphragm. It is rarely completely walled off and so not strictly an 'abscess'. Infection in the pelvis usually arises following:

(a) Infection of the female genital tract (commonest) which can be any of the varieties of pelvic inflammatory disease (PID, 23.1). Those caused by anaerobes are particularly serious and likely to spread. The patient may be very ill; you may have difficulty finding pus, and knowing when and how to drain it. The danger is that pus may build up as a mass above the pelvis, and spread upwards into the abdominal cavity, perhaps fatally, instead of discharging spontaneously and harmlessly through the vagina or rectum. Drain this type of pelvic abscess early, and *don't 'wait and see*'.

(b) Appendicitis (14.1).

(c) Caesarean section (21.13) **or hysterectomy** (23.15).

(d) Vaginal delivery (22.14).

(e) Generalized peritonitis (10.1).

(f) Diverticulitis.

A septic pelvic collection can grow quite large without causing very obvious illness or signs. As the signs are subtle, it is worth becoming experienced in rectal or vaginal examination. One danger is that a pelvic abscess may obstruct the bowel (12.16). You can sometimes drain a man's pelvic collection rectally, and a woman's collection vaginally. This may be easier than performing a laparotomy. However, if coils of bowel lie between the pus and the posterior fornix, it will be more difficult to diagnose, and you will have to drain it abdominally. Sometimes, an abscess drains into the rectum spontaneously, but you will still need to drain it properly surgically.

SYMPTOMS AND SIGNS

Look for fever and the passage of frequent loose stools with mucus. There is often a history of tenesmus.

Feel for:

(1) A boggy, tender mass above a man's prostate filling the rectovesical pouch, or a soft bulging swelling in a woman's pouch of Douglas. Sometimes, the mass is almost visible at the vulva. You will not find fluctuation.

(2) Tenderness and occasionally an ill-defined mass suprapubically. If you suspect a pelvic abscess in a woman, put one finger into the rectum and another into the vagina. Normally, they should almost touch. If there is an abscess, you will feel it between your fingers.

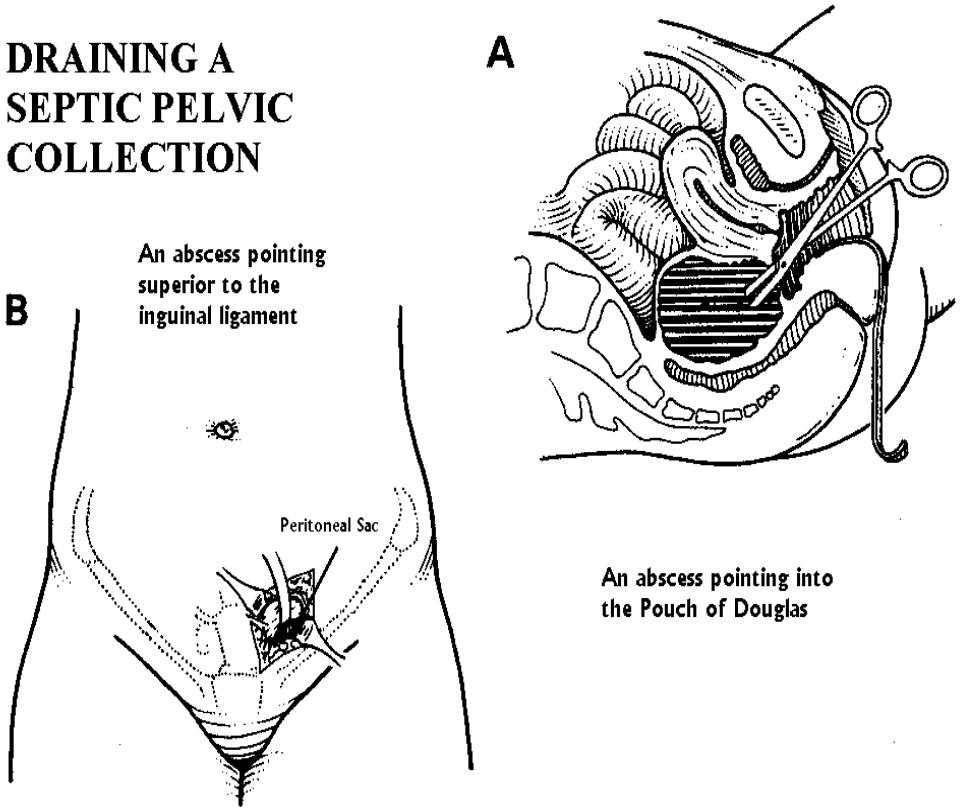

DRAINING A SEPTIC PELVIC COLLECTION
A
B
An abscess pointing superior to the inguinal ligament
Peritoneal Sac
An abscess pointing into the Pouch of Douglas

Fig. 10-9 DRAINING A SEPTIC PELVIC COLLECTION.
A, make sure the collection is pointing into the pouch of Douglas before you drain pus vaginally, or you may penetrate loops of bowel. B, drain a pelvic collection anteriorly by a Lanz or McBurney incision (11-1).

You can confuse a pelvic abscess bulging into the pouch of Douglas with:
(1) A 'chronic' ectopic gestation (20.7).
(2) An ovarian cyst (23.9). Some suspected cysts turn out to be post-inflammatory collections of fluid (post-inflammatory pelvic pseudocysts).

ULTRASOUND will most easily show a fluid collection in the pelvis (38-5), where it is, and how big it is: make sure the bladder is full before the examination! Always attempt drainage under ultrasound control, if possible.

VAGINAL DRAINAGE OF A PELVIC ABSCESS (POSTERIOR COLPOTOMY; CULDOCENTESIS) (GRADE 1.3)

INDICATIONS
(1) A pelvic collection which extends into the pouch of Douglas or upwards suprapubically into the abdomen.
(2) A pelvic collection which has ruptured in a patient too sick to undergo formal laparotomy.
(3) A pelvic haematocoele ('chronic' ectopic gestation) extending into the pouch of Douglas (20-7).

PREPARATION
Use the lithotomy position and catheterize the bladder. Perform a vaginal examination to confirm the diagnosis. Clean the vagina with 1% chlorhexidine.

INCISION
Expose the vaginal wall of the posterior fornix (*which must be bulging*) with a short broad speculum. Ask an assistant to depress the vaginal wall with a Sims speculum, while you raise the posterior lip of the cervix with a vulsellum forceps.
Use ultrasound guidance if you can: push a long large needle on a 20ml syringe into the swelling in the midline and aspirate:

If you aspirate pus, this confirms a pelvic abscess, or **if you aspirate a pale yellow fluid**, you are probably draining a post-inflammatory pseudocyst. Make a 2-3cm transverse incision in the vaginal wall in the place where you found pus. Push in gently a blunt instrument; pus or fluid should flow. Enlarge the opening. Explore the abscess with your finger; feel for loculations in the abscess cavity and gently open them. Insert a large drain and suture it to the perineum or labia. Leave it in and continue antibiotics. Pus may discharge for up to 2wks.

If you aspirate blood which clots easily, you have punctured a blood vessel (which should not happen if the needle is in the midline.)

If you aspirate >2ml dark, free-flowing blood which does not clot even after 10mins, this is a haematocoele due to a chronic ectopic gestation (20.7): perform a laparotomy.

If you aspirate clear fluid, this may well be liquor (or a cyst in the pouch of Douglas) suggesting an acute ectopic gestation. Check whether the abdomen is full of ascites, suggesting TB or liver disease.

If you aspirate faeces, you have probably entered the colon, which may be stuck to the vagina. Withdraw the needle and repeat the aspiration in the midline. If a faecal fistula results, manage this conservatively (11.15).

CAUTION!
(1) If pus is pointing laterally, drain it as close to the midline as you can, to avoid injuring the ureters (23.15).
(2) *Don't push too deeply into the abscess with a haemostat*, or its roof may give way and spread the pus into the abdominal cavity; or you may damage a loop of bowel. Be safe, and gently insert your finger through an adequate incision. The effect should be spectacular, and marked improvement should result within 1-2 days.
(3) *If you don't find pus, perform a laparotomy*.

If there is no improvement, there must be more pus somewhere. Repeat the ultrasound or perform a laparotomy.

RECTAL DRAINAGE OF A PELVIC ABSCESS (GRADE 1.5)

PREPARATION
Use the lithotomy position. While the abdomen is relaxed, palpate it gently. Then make a bimanual examination with one finger in the rectum, and your other hand on the abdomen. If you can ballot a fluid collection, using ultrasound guidance, needle it, taking a 1mm blunt needle attached to a syringe. Place the tip of your gloved right index finger over the place in the anterior wall of the rectum where you feel pus. Slide the point of the needle up alongside your finger, then push it through the wall of the rectum for about 2cm and aspirate.

If no pus comes out, inject a few ml of saline, and aspirate again. The needle may be blocked.

If you aspirate pus, drain it. Either push the tip of your index finger into it: pus will burst out. Or take a long curved haemostat, and with your index finger again acting as a guide, push its tip through the anterior wall of the rectum into the abscess. Enlarge the hole by opening and closing the jaws a few times.

CAUTION!
(1) Make sure you find pus before trying to drain it.
(2) The rectal wall may bleed seriously; if so, pack it with gauze.

SUPRAPUBIC DRAINAGE OF A PELVIC ABSCESS (GRADE 2.2)

This is sometimes needed in women (it is almost never necessary in men), particularly after an induced termination of pregnancy, or a Caesarean section when you can feel a mass suprapubically but not vaginally. Fortunately, you can usually drain an abscess from below, which is easier and safer. Rarely, if more pus collects after vaginal drainage, especially if there is distension, tenderness and induration behind and above the pubis, and if there is also severe toxaemia, drain the pus suprapubically.

PREPARATION

Catheterize the bladder to make sure it is empty. Make a 10cm Pfannenstiel incision immediately above the pubis. Incise the *linea alba* and the peritoneum. Inspect the general abdominal cavity and pack off the upper abdomen with some large moist abdominal packs. Gently feel for the collection. Retract the bladder and bowel out of the way. Look for pus, for loops of bowel stuck down in the pelvis, and for oedematous or congested tissues. Insert a self-retaining retractor.

Use a 'swab on a stick' and gently mobilize adherent loops of bowel, until you have found the pus.

CAUTION!

(1) *Don't use the Trendelenburg position to improve exposure*; this may spread the infection.
(2) Keep manipulation to a minimum.
(3) When you have found pus, do nothing more than is necessary to ensure adequate drainage. *Don't break down the outer walls of the abscess cavity*, but do break down any loculi inside. Distinguishing between them may be difficult.
(4) *Don't remove a normal appendix*.

Culture the pus and insert a drain. Remove all the packs; suture the abdominal muscles securely, but *don't close the skin immediately* (11.8).

DIFFICULTIES WITH PELVIC ABSCESSES

If there is colicky pain, vomiting, and abdominal distension, the small bowel is obstructed. Try to treat this non-operatively, with nasogastric suction and intravenous fluids (12.16). Draining the abscess will usually cure the obstruction. If it does not, you may have to relieve the obstruction operatively (12.4,6).

If you rupture the bowel whilst exposing the pelvic collection, control the spillage with soft bowel clamps and gently extract the remaining adherent bowel from the pelvis. Wash out the cavity with copious warm fluid. If the bowel perforation is small and the patient's condition is good, close it (11.3); otherwise bring out the proximal portion as an enterostomy.

11 Methods for abdominal surgery

11.1 Before a major operation

A patient is much more likely to withstand major surgery successfully if he is fit. So, do all you can to get him into the best possible condition first. For example, if you find that he is anaemic, malnourished, or suffering with tuberculosis, and the operation is not urgent, correct these things before surgery. Severe malnutrition will greatly reduce his ability to withstand the operation. Above all, *don't operate on a patient who is dehydrated;* you must correct this.

Assess the need for surgery, the best time for it, and the risks it will involve. If a particular procedure risks being too much to cope with, ask yourself if there is a lesser alternative, or whether you could do something under LA, or what will happen if you do nothing. If there is a choice of procedures, do the simplest and safest thing, for example, colpotomy for a pelvic abscess rather than a laparotomy (10.3).

Consider whether you need to perform the operation straight away, or after fluid resuscitation, urgently but at a more convenient time, or electively. Consider the help you need: are the people you have available actually present? Are there other demands on your time: are these really important?

(a) Follow these rules:
(1) *Don't start an operation without thinking it through step by step first*. If you are not familiar with the patient's case, make sure you go through the notes and review the indications for surgery carefully *yourself*.
(2) Monitor a patient closely for 48h after any emergency or major operation.
(3) Prevent aspiration of stomach contents, and treat respiratory depression immediately.
(4) The most common post-operative complications are respiratory, and the answer to most of them is vigorous chest exercises (11.12).
(5) Correct any dehydration and make sure enough IV fluids are infused perioperatively to obtain a urine output of 1ml/kg/hr.
(6) Discuss the case beforehand with your anaesthetist, mentioning possible hazards and blood loss.
(7) The operation may be routine to you, but it is sure to be a major event in the patient's life, so try to establish a good relationship with him and his family. Tell him why you are operating, and explain what to expect afterwards, particularly how much pain he will have, and when he will recover. For example, if you might have to remove a testis or make a colostomy, discuss this before you operate. Further, if you promise to close a colostomy eventually, be sure to do so.

THE ANTERIOR ABDOMINAL WALL

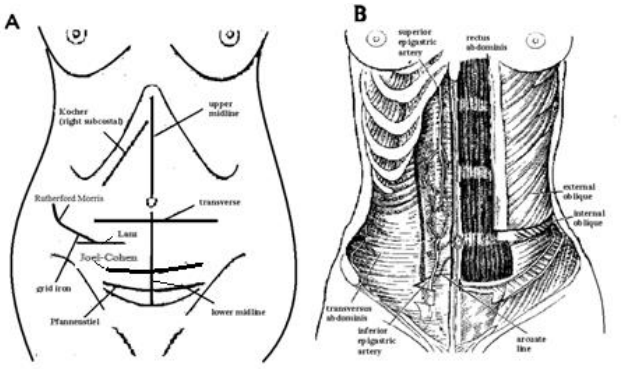


Fig. 11-1 INCISIONS AND ANATOMY OF THE ANTERIOR ABDOMINAL WALL.
A, abdominal incisions. *After Kirk RM, Williamson RCN, General Surgical Operations, Churchill Livingstone 1987 p.42, Fig.4.1,2.* **B, anatomy of the anterior abdominal wall.** *N.B.* Below the arcuate line, the posterior rectus sheath is absent. *After Maingot R, Abdominal Operations, HK Lewis 4th ed 1961 Fig. 1, with kind permission.*

(b) Pre-operative preparation
(1) **History and examination.** What previous illnesses has the patient had? Is he taking any medicines? Is he allergic to anything, particularly to streptomycin, sulphonamides, penicillin, or chloroquine?
Assess the degree of wasting. Ask about a cough, fever, chest pain, dyspnoea, and smoking. How fit is he? Can he climb hills, or do a day's work in the fields? Can he step up and down off a chair for half a minute without becoming short of breath? Or, can he hold his breath for 20sec? Can he get out of bed independently on one side, walk round the bed, and get in again on the other side? (This tests coordination, mental faculties, as well as cardiac output.) Look for signs of anaemia. Feel the strength of his grip (this is a good predictor of surgical risk in men, less so in women).

(2) **Ask about tubal ligation.** *Always* remember to ask any pre-menopausal lady over 30yrs who has had ≥2 children if she is interested in having a simultaneous tubal ligation. Most women either forget or are too afraid to ask but always regret not having done so. If the lady is agreeable, remember to add the procedure to the consent form *and* the operation list!

(3) **Special tests.** Measure the Hb or haematocrit. If sickle cell disease is common in your area, test for this routinely. Test the urine for albumin and sugar, and examine its deposit. This will exclude any serious disease of the urinary tract, and help you to diagnose renal colic, which may present as an acute abdomen. Test the blood group, and if necessary cross-match blood. Remember to test for HIV. If you suspect heart or lung disease, obtain a chest radiograph and/or ultrasound scan.

(4) **Associated disease.** If necessary, and if time allows, try to improve the patient's general health, especially his nutrition and hydration. Look for tuberculosis, chronic renal disease, and signs of HIV disease.

If the patient is dehydrated, make sure you provide enough fluid (usually IV) so that he has a good urine output.

If the patient is malnourished, especially if he is a child, and the disease permits, feed him by mouth or by nasogastric tube, even for as short a period as 2wks before you operate. If he is anorexic, feeding him will be difficult, so try nasogastric feeding (11.10).

If there is pyrexia, consider the possibility of malaria, HIV or typhoid fever, in addition to the possible surgical causes of fever.

If there is anaemia, consider the urgency of the operation in relation to the severity of the anaemia. Most 'routine' operations can be done with an Hb count as low as 80g/L. If it is 6-7g/dl, only do urgent procedures. For example, you can transfuse a woman with an Hb count of 30g/L who is bleeding slowly from an ectopic pregnancy overnight with 20ml/kg of blood from her abdominal cavity (5.3), if necessary adding 1mg/kg of furosemide. If an operation is less urgent, for example a hysterectomy for chronic anaemia due to fibroids, treat with iron sulphate for 4-6wks. For non-urgent surgery where you expect bleeding, you can take a unit of blood 4wks and 2wks before a planned major operation, and store the blood.

If there is jaundice, it will greatly increase the risks of surgery, but not operating may worsen liver failure. Treat with vitamin K 10mg IM od and repeat this if the jaundice is severe. Exclude hepatitis and malaria first, especially in the acute stage, when anaesthesia can be dangerous.

If there is much purulent sputum brought up, arrange chest physiotherapy and a course of antibiotics prior to surgery, if possible. Use appropriate anaesthesia, using LA or regional blocks where possible. If there is a common cold, cancel anything but an emergency operation.

If the patient is diabetic, check the glucose level. If the diabetes is out of control, start a 'sliding scale' regime of soluble insulin IV qds, with the total adjusted according to the total dose, if any, of insulin being taken. If he takes oral hypoglycaemics, omit these on the day of surgery. If he uses insulin bd, reduce the pre-operative evening dose by 20%; omit the insulin on the morning of operation and start a 5% dextrose IV infusion. Check the glucose again just before operation and try to operate on him first on the list.

CHEST PHYSIOTHERAPY before and after the operation will reduce the risk of lung complications (11.11,12), especially for the bronchitic, the smoker and the elderly.

SKIN PREPARATION. If the skin is very dirty, wash the operation site several times. If there are pustules, boils, or eczematous patches near the site of your proposed incision, treat them before you operate. Bacteria from them may infect the wound, so if you must operate urgently, use delayed primary closure (11.8).

(5) **Nasogastric suction.** Insert a nasogastric tube (4.9) before all stomach or bowel operations, and in emergencies if you don't know when the patient last ate or drank. The danger of aspiration pneumonitis is even greater if there is intestinal obstruction or ileus. It is especially present when the patient is relaxed before intubation. Aspirate the stomach contents before you go to theatre. The risks of aspiration are greater if the patient is lying down or sedated. Get an assist to apply cricoid pressure before and during intubation; aspirate the stomach contents from time to time during the operation.

(6) **Urinary catheterization.** Insert a catheter into the bladder for pelvic and long operations, where monitoring of the urine output during the operation is helpful, or in an elderly man with a large prostate where you fear urinary retention post-operatively. *Don't insert a catheter routinely!*

(7) **Perioperative antibiotics** may be life-saving (2.9); *distinguish between prophylactic and therapeutic use!*

DON'T OPERATE ON A SEVERELY DEHYDRATED PATIENT:
RESUSCITATE TILL YOU OBTAIN YELLOW-COLOURED URINE

11.2 Laparotomy

A laparotomy for an acute abdomen will be a major test of your surgical skill. It will be important to know what to do when you open the abdomen; if you are not sure, be humble enough to have this book in theatre to help you. Try to make a correct diagnosis before you start, *but don't delay just because you are not certain what you will find*. Before you start, get a proper consent and discuss the operation with the patient and his family, and if he is to have drainage tubes or a colostomy for example, make sure they understand what these entail.

N.B. In some societies the presence of a drain or colostomy may appear to indicate imminent death, so *take time to explain details such as these carefully*.

Try to avoid complications by making an appropriate incision, handling tissues carefully, and closing accurately. Make an incision which is big enough; a common mistake is to make it too short. Incisions heal from side-to-side, not from end-to-end, so *don't be afraid of making a long incision*.

If an incision is too small:
(1) *you will not be able to dissect safely*, and
(2) *your assistant will have to exert excessive traction on its edges*, which will kill tissue, and increase the risk of wound sepsis and breakdown.

Which incision?
The ideal incision is one which gives adequate exposure, is quick to make, easy to close, and provides a secure, cosmetically acceptable abdominal wall afterwards. So, *avoid an incision in the wrong place!*

(a) A transverse incision in children <12yrs is ideal because it heals better, and gives less respiratory problems. The transverse incision also heals better in adults, and is less likely to develop hernias than the midline, but you need to be careful with good haemostasis.

(b) The Joel-Cohen incision (21.10, 23.15) allows for easier access to the pelvis, especially in pregnancy, through a transverse incision 3cm below the anterior superior iliac spines; it bleeds far less, is easier to learn and use, is quicker, heals stronger, less painful, less at risk of keloid formation, and is more versatile than other incisions particularly for Caesarean section.

(c) A midline incision in an adult will enable you to get access to everything in the abdomen. You can start with the middle part to begin with, and extend it from the xiphisternum to the pubis, if necessary. If exposure is particularly difficult, you can extend the incision laterally to make a 'T' incision. Many patients prefer an incision going straight through the umbilicus rather than going round it: ask them.

A midline incision above the umbilicus is quick, simple, and bloodless. But access to the organs at the sides of the abdomen is not easy. Even so, midline incisions are usually best for trauma, for Caesarean sections where unusual pathology is expected, and almost everything else. If you are planning a purely exploratory laparotomy, and don't know what you are going to find, make a midline incision in the correct half of the abdomen, upper or lower.

(d) A previous incision needs cutting out if you are re-opening the abdomen. Extend it so that you can enter the abdominal cavity above or below where any adhesions to the undersurface of the abdominal wall are less likely. Work your way up or down carefully, dividing any adhesions you find, so as not to injure any adherent bowel. *Don't make a 2nd incision parallel to an earlier one or crossing an earlier one at an acute angle*, because the skin in between will have no sensation and may become ischaemic.

(e) A McBurney (grid-iron) or Lanz incision gives good but limited access to the appendix: use it only if you are sure of the diagnosis of appendicitis with local inflammation alone.

(f) A Kocher (right subcostal) incision gives good access to the gallbladder, but not much else. A left subcostal incision gives good access to the spleen, and may be extended as a chevron (gable) on the opposite side.

(g) A lower oblique (Rutherford-Morrison) incision is good for the ureters and ascending (right) and descending (left) colon.

(h) A Pfannenstiel incision is ideal for gynaecological and prostate operations and **a left thoraco-abdominal incision** ideal for operations on the gastro-oesophageal junction.

N.B. ***A paramedian incision*** (parallel but 2-3cm lateral to the midline) *does not have any benefit over a midline incision*, and is more bloody and difficult to do properly.

CAUTION! If you are in doubt, make the 'incision of indecision' in the midline 5cm above and 5cm below the umbilicus. Enter the abdomen and then extend it in the most useful direction.

When you get inside, you will have to decide what to do. Here, only experience can tell you what is normal and what is not. For example, some *ascaris* worms inside a child's bowel may feel so abnormal as to convince you that they must be the cause of the symptoms, when in fact they are normal for the community.

Be gentle, bowel is highly sensitive. If you handle it roughly, especially if it is obstructed, ileus will follow as sure as night follows day. Bowel does not like being frequently drawn out of a wound. So, if you need to draw it out, do so only once, and hold it with a moist swab. While it rests on the abdominal wall, keep it covered with warm moist packs or towels, or place it in a large sterile plastic bag. If it is grossly distended, even the most gentle handling may burst it, so decompress it (12-6). Break down adhesions gently (12-8).

N.B. Remember, in an emergency, do what has to be done as efficiently as you can: don't be tempted to do unnecessary things! It is often best to wait 48h to stabilize a patient and return when the condition has improved.

ENTERING THE PERITONEAL CAVITY

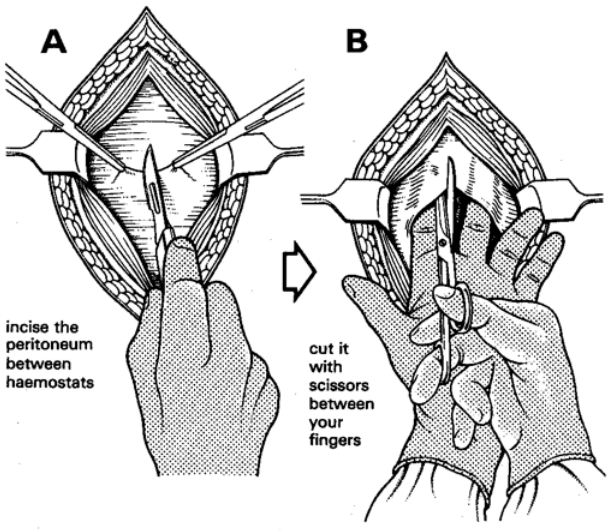


Fig. 11-2 ENTERING THE PERITONEAL CAVITY.
A, incise the skin and divide the anterior rectus sheath. The posterior rectus sheath and peritoneum form a single layer. Pick this up with a haemostat, and then apply another one 5mm from it. With the peritoneum still tented up, make a small incision between the 2 haemostats. Air will enter the peritoneal cavity, and the viscera will fall away. B, put your fingers into the incision to make sure that there are no adhesions to the undersurface of the abdominal wall, and then extend the incision with scissors.

LAPAROTOMY (GRADE 3.1) **technique**

(a) Resuscitation. Make sure that there is a functioning infusion with a large cannula. You can infuse approximately 85ml/min through an 18G cannula, but >200ml/min through a 14G. Make sure the IV line is secure and not kinked: you may have to splint the arm on an armboard. If significant bleeding is possible, have blood cross-matched and a 2nd IV cannula in place.

(b) Radiographs. A chest radiograph is most useful; have the films available in theatre. Make this hospital routine.

(c) Anaesthesia.
(1) GA, preferably with relaxants, is the norm. Ketamine 1mg/kg IV is very useful if the blood pressure is low. *Don't induce anaesthesia with thiopentone in a shocked patient:* the blood pressure may crash!
(2) Subarachnoid (spinal) or epidural anaesthesia is useful for the lower abdomen, but requires good hydration and a fluid pre-load IV.
(3) LA may be safer in a gravely ill patient: mix 10ml of 2% lidocaine with 40ml of saline to give 50ml of 0·4% solution. To this add 0·5ml of adrenaline 1:1000. Inject 1ml of this solution into each of five sites in the rectus muscle on either side of the midline to block the segmental nerves. Use another 20ml to infiltrate the midline. Use the remaining 20ml to infiltrate the root of the mesentery if you need to resect the bowel and top up infiltrations as required.

If you have to perform a more extensive procedure, use ketamine rather than struggling to work with LA alone, and if the patient is critical, do the minimum necessary (10.1). *N.B. Unconscious patients often need no GA at all!*

(d) Position. For most abdominal operations, lie the patient supine with the bare buttock or thigh in contact with the diathermy pad (if you have one).

If your table does not tilt from side to side, and you want to turn the patient to one side, place pillows under his back on each side, or use a wedge block under the mattress.
If you are operating on the pelvic organs, you will find the Trendelenburg (head-down) position helpful. It will allow the bowel to fall towards the diaphragm, so that you get a better view into the pelvis. You will need well-padded shoulder rests to prevent the patient sliding downwards. *Don't tip the patient too steeply*, or the pressure on the diaphragm will impair his breathing. If he is in >10° of Trendelenburg, you must intubate him, keep him on relaxants, and control his ventilation.
If you need access to the rectum or pelvic organs, put the legs up on lithotomy poles or on Lloyd-Davies stirrups.

Use one arm for a blood pressure cuff and the other for an IV line, unless you need 2 IV lines. Keep the hands by the side, or out on arm boards, or folded on the chest with suitable ties; *don't place them under the buttocks or under the head*.

(e) Examination.
Once anaesthetized and relaxed, feel the abdomen carefully. You may feel a mass which you could not feel before because of guarding.

(f) Preparation.
Shave the abdomen (2.3). Make sure the operation light is correctly positioned and directed. Drape the abdomen and fix the drapes with towel clips. Cover these with a large windowed sheet, and add additional sterile towels as necessary. Make sure you have suction and diathermy connected, clipped to the drapes, switched on, and tested, with a foot pedal within reach.

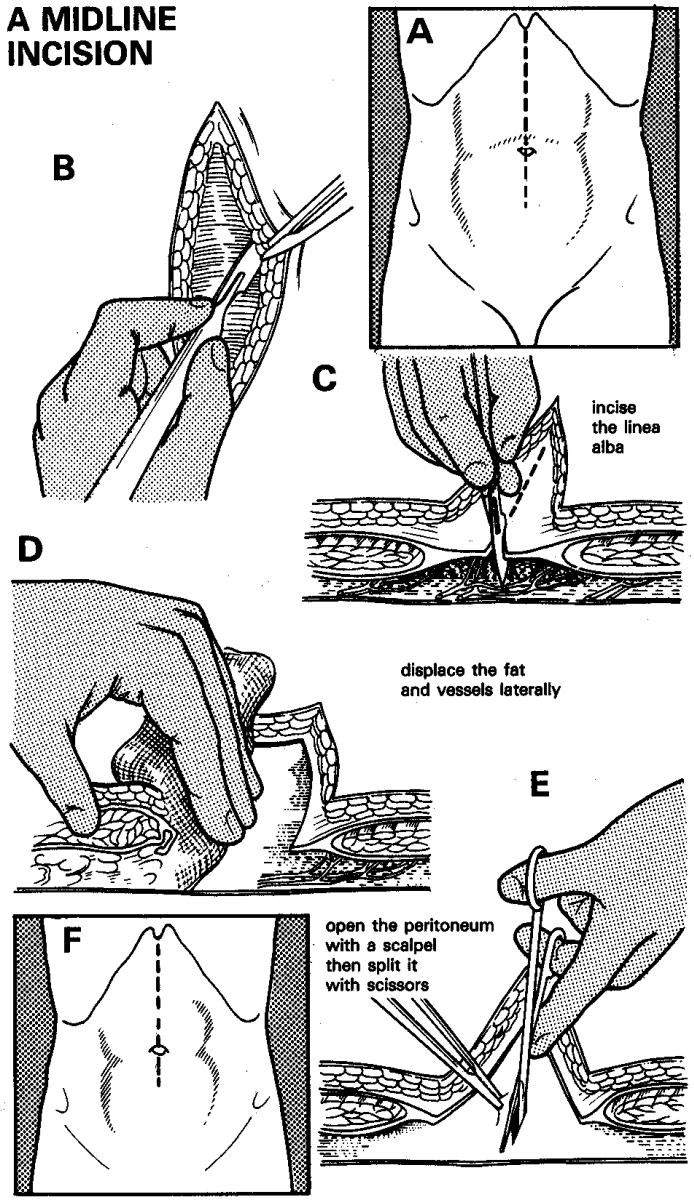


Fig. 11-3 MIDLINE INCISION.
A, the site. B, cut down to the *linea alba*, and then carefully dissect the fat for 1cm on either side, using the flat of the knife. C, incise the *linea alba* to expose the underlying fat and peritoneum. D, displace the fat and vessels laterally by blunt gauze dissection. E, pick up the peritoneum, incise it with a knife and divide it with blunt-ended scissors. F, if you want to continue the incision downwards, there is no need to go round the umbilicus. *After Rob C. Operative Surgery: the Abdomen. Butterworth, 1977 Permission requested.*

TRANSVERSE INCISION. Make a cut 3cm above, or below the umbilicus (Joel-Cohen, 23.15) and cut into the central part anterior rectus sheath, and open this and the peritoneum centrally under the muscle; stretch open the muscle longitudinally with fingers (Misgav Ladach method). This way you retract the vessels in the muscle layer laterally. You can extend the incision in a curve upwards laterally, if you need access to the spleen or liver.

UPPER MIDLINE INCISION. Use the xiphoid and umbilicus as landmarks, keep strictly to the midline, and *don't cut into the rectus muscle on either side*. Cut down to the *linea alba* and cut through it gently to expose the extraperitoneal fat. Pick up the *linea alba* with straight forceps, and cut gently with the knife in the midline till you see the posterior rectus sheath and peritoneum. Lift this up with 2 straight forceps, feel it with your fingers, making sure you have not also picked up underlying bowel. It is a good idea to release one of the forceps, and re-apply them to let anything caught the first time slip away. Cut into the peritoneum with the knife (11-2A) and so allow air to enter the peritoneal cavity; the viscera will then fall away, allowing you to put in 2 fingers to check if there are any adherent underlying structures, tent up the peritoneum and divide it with blunt-ended scissors (11-2B).

N.B. You can easily open bowel by mistake if:
(1) it is obstructed,
(2) it has stuck to the scar from a previous operation,
(3) you are hasty or rough,
(4) you use diathermy to open the abdomen.

N.B. Don't use the paramedian incision: it has no real advantages.

LONG MIDLINE INCISION. Don't hesitate to open the abdomen from top to bottom *e.g.* for advanced generalized peritonitis (10.1). You can go straight through the umbilicus: it leaves a neater scar than going round it, but remember to clean it thoroughly beforehand. You can also get a little more length by incising between the xiphisternum and the costal cartilage.

LOWER MIDLINE INCISION. Make this in a similar way to the upper midline incision. If you cannot see exactly where the midline is between the rectus muscles, split them apart gently. Make sure you use haemostats or diathermy on bleeding vessels. You will see the *pyramidalis* muscle at the lower end of the wound. Note that below the umbilicus, in the lower ⅔ of the midline, below the arcuate line, the peritoneum is attached only to *transversalis fascia*.

PFANNENSTIEL INCISION. Make a slightly curved transverse incision 2cm above the *symphysis pubis.* Cut through the anterior rectus sheath and hold upper and lower flaps with straight forceps. Gently lift this sheath off the underlying rectus muscle, and split this longitudinally to expose the peritoneum in the midline.

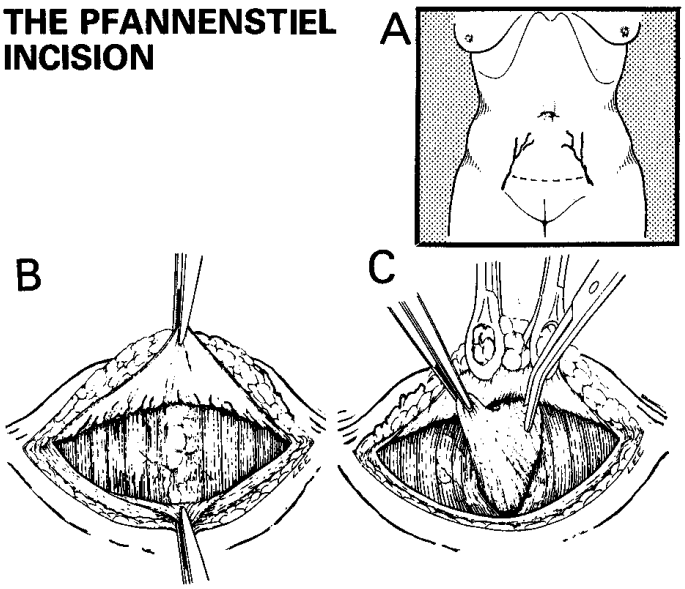


Fig. 11-4 THE PFANNENSTIEL INCISION.
A, incision: the inferior epigastric arteries lie at the ends of the incision on the deep surface of the rectus muscles. B, reflect the anterior layer of the rectus sheath. C, part the rectus muscles; prepare to enter the abdomen. *After Dudley HAF (ed), Hamilton Bailey's Emergency Surgery, Wright, 11th ed 1986 p.446 Fig 42.1 with kind permission.*

(g) Get adequate exposure and a good light. *You cannot do good work if loops of bowel are always getting in the way, or if the light is bad*, so adjust it as best you can. Sterilizable light handles are very useful; otherwise make sure someone else in the theatre knows how to move and direct the light. Alternatively use a head-light. Get one or two assistants to help you.

Make an adequate incision. If necessary, extend it or close it (*e.g.* if you find a Lanz incision for appendicectomy inadequate) and make a good midline incision. If you are working on a lateral organ through the midline, make a long incision. Or, make a lateral T-shaped extension.

Get good retraction. A self-retaining retractor may not be enough by itself. Use Deaver's retractor, or any large right-angled retractor, and make sure your assistant knows what you want him to do with it. *Don't become cross with him when the position of the retractor slips if he cannot see properly!*

Get the patient into the best position. You will never get adequate exposure in the pelvis unless the body is tilted in the Trendelenburg position. Similarly, if you are working on the upper abdomen (as when exposing the oesophagus), tilt the head up a little. Extending the back by breaking the table or by putting a pillow under the back will also help. If you want to draw the splenic flexure and small bowel towards you, consider rolling the patient to the right, either by tilting the table or by using sandbags, or a wooden wedge under the mattress. If you are operating on the kidney, a kidney bridge or folded plastic-covered pillows will bring it forwards.

DON'T MAKE THE INCISION TOO SHORT:
ALWAYS BE READY TO EXTEND IT!

If loops of small bowel (or anything else) get in your way, pack them away. This may save you much time, but *don't forget to remove the packs afterwards!* Anchor each pack by its tape or corner to a large haemostat hanging outside the abdomen.
Make sure the scrub nurse checks and counts the packs and instruments *with you* at the end of the operation. *Avoid leaving loops of bowel hanging outside the abdomen exposed, especially under hot theatre lights:* they easily get desiccated and the weight of bowel contents may impair its mesenteric blood supply. Keep the bowel covered with warm wet packs.

(h) Follow a routine. Take note of the smell, look at the fluid, gently divide adhesions, minimize the risk of sepsis, decompress the bowel, and make a thorough examination of the abdomen (10.1). How extensively you do this will vary. Limit your exploration to what is easily practicable if:
(1) there is localized sepsis and you aim to limit its spread to the rest of the abdomen,
(2) there is an inoperable carcinoma, or
(3) you are operating on a known problem.
However, even then it is good practice to take the opportunity to make a full examination. *Decide carefully though, if the bowel is very stuck together, whether you will do more harm than good by separating all the adhesions* (10.1).
Explore the abdomen in an orderly way: look at the diaphragm, liver, intra-abdominal oesophagus, spleen, stomach, duodenum, gallbladder, and then the whole small bowel. Draw each loop out of the wound, looking at both sides, and at the mesentery. If the bowel is distended, trace it distally to find the cause. Look at the large bowel from caecum to rectum. Feel the major vessels and kidneys, look at the appendix, ureters, bladder, uterus, Fallopian tubes, and ovaries. Finally look at the hernial orifices from inside. If necessary, look into the lesser sac through the greater omentum; or behind the duodenum by 'Kocherizing' it (13.3); or at the oesophageal hiatus by dividing the attachments of the left lobe of the liver to the diaphragm.

Don't forget to record your findings: even negative ones, which can be most helpful later.

(i) The specimen. If you have removed tissues from the patient and want to examine them, hand them to someone else. Ask him to open them away from the patient, who will then not be contaminated by infection or malignancy.

DIFFICULTIES WITH A LAPAROTOMY

If you cannot do an operation through one incision, make another. Keep your original one open until you have finished: it may be useful!

Bleeding can be difficult. You must know how to:
(1) tie vessels in the depth of a wound (3.2),
(2) place ties, without letting go of the thread (3.2),
(3) use curved and angled forceps,
(4) secure temporary tape control over major vessels.

If a surface is merely oozing, consider applying haemostatic gauze (3.1).

If the bleeding is annoying, rather than brisk, you may be able to suck it away while you go on working.

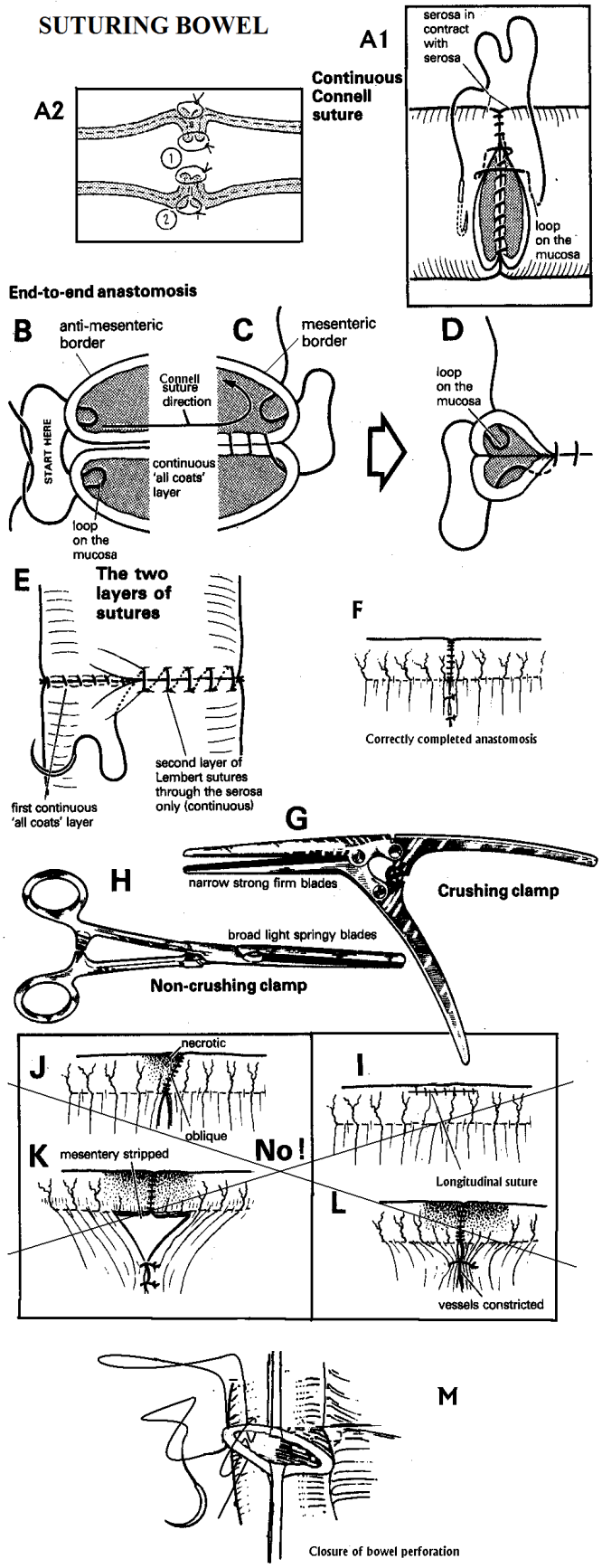
If you have diathermy, consider applying it to the bleeding point with a fine-tipped dissecting forceps. You should do this with pinpoint accuracy.

If there is a constant ooze during the operation:
(1) There may be an excess of citrate after transfusion of many units of blood. This will not happen if you add 10ml of 10% calcium gluconate after every 4th unit (500ml) of blood.
(2) There may be disseminated intravascular coagulation (DIC), or some other clotting defect. If possible, transfuse at least 2 units of fresh blood to replace clotting factors. Check the blood clotting time.

If bleeding becomes unmanageable, *don't panic:* apply pressure, then packing. Either ligate or repair a major vessel (3.1,2).
CAUTION!
(1) *Don't stab blindly with a haemostat in a pool of blood!*
(2) Similarly, *don't apply diathermy through a pool of blood:* it won't work!

If you accidentally perforate a loop of bowel, *don't try to stop the leak of bowel contents with gauze.* Pinch it closed between your fingers while someone gently places non-crushing clamps proximally and distally. Suction any spillage. Surround the injured loop with packs to prevent the contents of the bowel flooding into the peritoneal cavity. Repair the perforation (14.3); if there are ≥2 perforations near each other, it is best to sacrifice a segment of bowel and make one anastomosis (11.3) rather than attempt to repair several holes.

If you open the pleura by mistake, there is a danger that the lung may collapse and cause marked hypoxia, because only one lung is being ventilated, and also because blood is passing through the collapsed lung unaltered. If the patient is not intubated, stop operating to make it easier for the anaesthetist to pass a cuffed tracheal tube using suxamethonium. Intubation is not essential, but chest drainage usually is. To do this, you may have to move the patient. As soon as the tube has been inserted, close the hole in the pleura with a continuous multifilament suture. As you insert the last suture, ask the anaesthetist to blow up the lung so that it almost touches the pleura. At the end of the operation insert an intercostal water seal drain (36.1) and leave it in place for at least 48h. Obtain a chest radiograph, and once the lung is fully expanded, remove the drain, usually at 2-5 days.



11.3 Resecting and anastomosing bowel: end-to-end anastomosis

A laparotomy will often mean resecting bowel. This is one of the most critical procedures you will have to undertake and, if you are inexperienced, one which will give you much anxiety. It is one of the few surgical methods, however, which you can usefully practise before you operate on a living human patient. Go to the butcher's, get some animal bowel, and practise anastomosing that. The penalty for failure in the patient will be peritonitis or a fistula.

You will usually anastomose bowel end-to-end, but there will be occasions when you will have to do it end-to-side, (as in a Roux loop) or side-to-side (as in a gastro-enterostomy, or cholecysto-jejunostomy).
Don't be worried by the complexity of the methods which follow. The really important points are to:
(1) Make sure that you start with 2 nice viable pink bleeding ends.
(2) Empty the bowel as best you can.
(3) Get their serosal surfaces together. If you do this, they will soon unite. If you bring only the mucosal surfaces together, there will be no strength in the join and a leak is likely.
(4) Close the bowel in 2 layers using round-bodied needles. If you rely only on one layer, you need to be extremely neat and accurate. Beware of mucosa pouting out after the first layer; it can easily do this at the mesenteric border. *Everted mucosa leaks*. So if it does evert as a 'dog ear', push the 'ear' back and close it over with the serosal layer.
(5) Do the suturing outside the abdominal cavity on a towel, or pack away the rest of the abdominal contents. Contamination will then be less likely and clamps less important.
(6) Wash the bowel with warm fluid after you have done the anastomosis.
(7) Gently squeeze the bowel on either side to test the anastomosis for leaks.

Fig. 11-5 SUTURING BOWEL.
A1, suture bowel with continuous Connell sutures, showing the principle of 'the loop on the mucosa' inverting the bowel. A2, anastomose bowel end-to-end with 2 layers of sutures: an 'all-coats' layer and a layer of Lembert sutures through the serosa only. B, method of anastomosing bowel end-to-end (11-7) using Connell sutures. This starts on the antimesenteric border to the mesenteric border C, where it turns round to close the anterior layer of the bowel and D meets the beginning of the suture again back at the antimesenteric border. E, 2 layers of sutures: the 1st continuous absorbable 'all-coats' layer and the 2nd continuous serosal or Lembert layer. F, anastomosis correctly done. G, Payr's crushing clamp, with firm, narrow blades. H, Lane's non-crushing clamp with springy, broad blades.
***N.B. Pitfalls*: I, bowel closed longitudinally (which will result in stricture formation). J, bowel cut obliquely in a way which reduces the blood supply to an area on the antimesenteric border of one loop. K, bowel partly deprived of its mesentery, and thus of its blood supply. L, mesentery bunched together with a suture which occludes the vessels supplying the bowel.**
M, closing a bowel perforation, starting at one corner with a Connell suture.

If you follow the 7 points above, you won't go far wrong. Note that any sutures which go right through the wall of the bowel (and so might leak) are usually infolded by a 2nd layer of sutures which go through serosa and muscle only; these are called Lembert sutures. Put the first layer through all its coats: this is the 'all-coats' layer. Make the Lembert sutures of the 2nd layer bring the serosa of one loop into contact with the serosa of the other loop. Only put them through the outside peritoneal layer, the muscle, and the submucosa (the strongest layer of the bowel), and don't go through the mucosa into the lumen of either loop.

Use a continuous suture: it is easier, cheaper, and probably more reliable than using interrupted sutures, even with large bowel. *Avoid catgut*: it dissolves just when the bowel is healing, and so needs a 2nd layer of sutures for protection. If your stocks are limited, preserve some longer-lasting absorbable sutures (*e.g.* polyglycolic acid) specially for bowel (4.6). *Avoid cutting V-shaped needles on bowel* as these can produce a leak.

You will need to hold the bowel with stay sutures, Babcock forceps (4.4) or clamps while you work on it. It is also desirable to hold it shut so that its contents don't leak out. Clamps do this best but you can use a cloth tape. There are 2 kinds of clamp: non-crushing ones and crushing ones.
Non-crushing clamps, such as Lane's or Kocher's, have thin, wide, flexible blades, and a ratchet with several teeth, so that you can adjust the way you close them to the thickness of the bowel. Use non-crushing clamps to hold bowel without injuring it; hold them between your fingers and 'milk' the bowel contents away from the area you are working on. Apply only as many 'clicks of the ratchet' as you need to stop the contents of the bowel from escaping, and blood from flowing from the cut ends. You can use bulldog vascular clamps for baby bowel.
Crushing clamps have narrower, stiffer blades, a ratchet with fewer teeth, and sometimes interlocking ridges on the blades to grip the bowel more firmly. Crushing clamps prevent leaking completely. 'Milk' the contents of the bowel away from the area to be crushed, and then apply a crushing clamp with its jaws protruding well beyond the edge of the bowel, because bowel widens as you crush it. Close the jaws tightly. Crushed bowel dies, so *cut the crushed bowel away with the clamp before making an anastomosis*. Cut the bowel strictly transversely, *not obliquely* (11-5J). As you do this, be sure there is a non-crushing clamp applied to stop the contents of the bowel spilling out. Crushing clamps are thus always used in conjunction with non-crushing ones.

You will often have to operate on bowel when it is distended and full of intestinal content: this fluid has millions of bacteria, particularly anaerobes. Spillage into the peritoneal cavity will cause septicaemia very quickly because the peritoneal layer is such a good absorptive surface. So, in this situation, you will have to use clamps; however, if you then join bowel which is full of intestinal fluid, this will all have to pass the anastomosis! The chance of leakage is then high.

Avoid this disaster by emptying the bowel every time you make an anastomosis. You will need to make sure that the bowel reaches outside the abdomen, and emptying it does not contaminate the peritoneal cavity, the very thing you want to avoid! Therefore before you empty the bowel, pack away the abdomen as a protective measure. If you can't make the bowel reach outside the abdomen, use a strong sucker to decompress it. This will not work in the distal small bowel or colon because the content is usually too thick, but that is where it is more important to empty it!
You will then have to allow the bowel content to pour out into a bowl, getting an assistant to hold both bowel and bowl carefully so as not to spill the fluid, whilst you milk the content out. Once you have done this, clamp the ends of the bowel and clean them with swabs held in sponge forceps ('swab-on-a-stick'). Finally, dab the bowel ends with betadine.
You can decompress bowel *via* a nasogastric tube if the content is very fluid, and your anaesthetist is ready to suck out the contents. The danger is spillage into the mouth, and from there into the lungs, especially if the endotracheal tube is uncuffed: *don't use this method therefore with children!*

FORCEPS, intestinal, non-crushing, flexible blades, Kocher's, Doyen's or Lane's, 75mm. Use these to hold the bowel while you anastomose it. Non-crushing clamps have been designed to exert the right pressure without being covered with rubber tubes. If you fit them with rubber, they may crush too tightly.
CLAMP, Payr, intestinal crushing, lever action, medium size, 110mm. These are the standard crushing clamps.
CLAMP, Payr, intestinal crushing, lever action, small size for pylorus, 60mm.

VIABLE BOWEL ENDS
MUST BE NICE AND PINK

MINIMIZE *CONTAMINATION*

THE CHOICE OF THE METHOD depends on the nature of the operation, your skill, and the equipment you have.

IS THE BOWEL VIABLE? TO RESECT OR NOT?
CAUTION! For any method of anastomosis, the bowel must be viable, which also means that its blood supply must be good enough.

Wait to decide if the bowel is viable or not until you have removed the cause (divided an obstructing band, or untwisted bowel which has twisted on its mesentery). You can usually tell if bowel is going to survive or not. Base your decision on several of these signs, *not on one only*.

Bowel is viable if:
(1) its surface is glistening,
(2) its colour is pinkish, or only slightly blue,
(3) it feels resilient like normal bowel,
(4) it contracts sluggishly (like a worm) when you pinch it,
(5) you can see pulsations in the vessels which run over the junction between it and its mesentery.

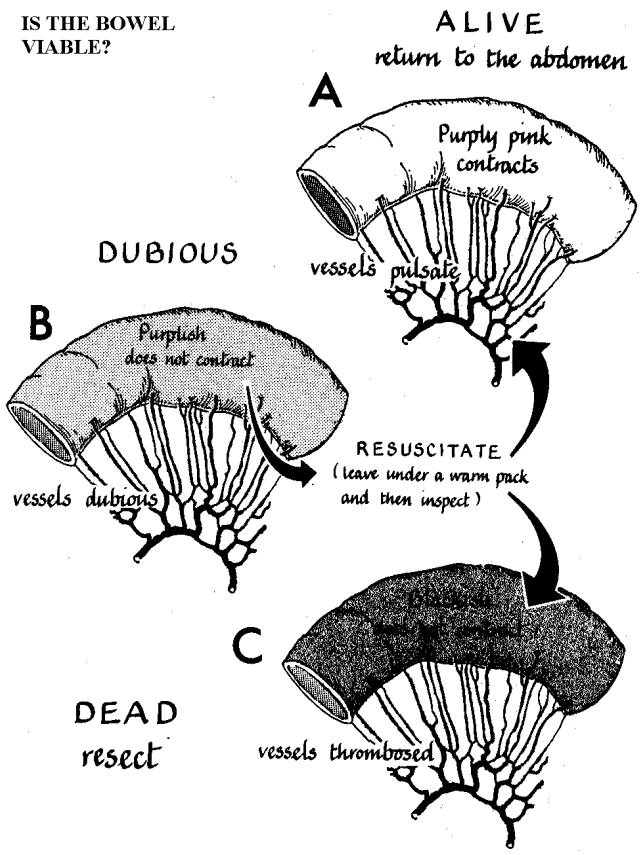


Fig. 11-6 IS THE BOWEL VIABLE?
A, it is viable if: (1) its surface is glistening, (2) its colour is pinkish, or only slightly blue, (3) it feels resilient like normal bowel, (4) it contracts sluggishly (like a worm) when you pinch it, and (5) you can see pulsations in the vessels which run over the junction between it and its mesentery.
B, if in doubt, remove the cause of the strangulation, apply a warm, moist pack to it, and wait 10mins. If it is viable, its colour will change from dusky to its normal pink.
C, it is dead and not viable if: (1) it tends to dry out and its surface is no longer glistening, (2) it is greyish purple, or a dark purplish red (or even black), (3) it feels like blotting paper, (4) it does not contract when you pinch it, (5) the blood vessels over it are not pulsating or are filled with black clot.

Bowel is not viable if:
(1) it tends to dry out and its surface is no longer glistening,
(2) it is greyish purple, dark purplish red or even black,
(3) it feels like blotting paper,
(4) it does not contract when you pinch it,
(5) its blood vessels are not pulsating or are clotted.

If you are in doubt, remove the cause of the strangulation, apply a warm, moist pack to it, and wait 10mins. If it is viable, its colour will change from dusky to its normal pink. If this happens, it is alive even if you cannot feel the pulsations of the mesenteric vessels. It may be alive if some areas remain purplish because of bruising. But if these areas are large, or don't improve in colour, consider all the discoloured bowel to be non-viable.

If a section of bowel is obviously non-viable, resect it and make an end-to-end anastomosis, making sure the ends you wish to join together are viable.

If only part of the wall of the bowel is non-viable, as with a Richter's hernia (18-2), you may be able to invaginate it. If you are going to do this, the non-viable bowel must:
(1) not be perforated,
(2) not extend over more than 30% of the circumference of the bowel,
(3) not extend to the mesenteric border, because suturing here may interfere with its blood supply,
(4) be surrounded by a border of healthy bowel.
Use 2 layers of absorbable suture to bring the serosal surfaces of the healthy margins together in the transverse axis, so as to invaginate the non-viable segment into the lumen of the bowel where it can safely necrose. It may actually be easier to cut out the non-viable portion, and close the V-shaped defect with invaginating Connell sutures.
If it does not satisfy these criteria, resect the non-viable portion formally.

If there is a completely encircling narrow band of greyish white necrosis, resect it and make an end-to-end anastomosis, otherwise it will turn into a Garré stricture of the bowel later.

If you release a loop of bowel from a constriction ring, *be especially careful.* The loop of bowel itself may be viable, but *there may be a narrow band of necrosis at both the afferent and the efferent ends*. It may slough at these narrow areas. Experts would resect the bowel. But, if you are not expert at bowel resection, oversewing the necrotic areas with Lembert sutures may be safer. If so, make a note of what you have found and done. A Garré stricture may form anyway, and the obstruction may recur.

IS THE BLOOD SUPPLY GOOD ENOUGH?
If the mesenteric vessels of the bowel you are going to anastomose are not pulsating, trim it back boldly until its edge bleeds with healthy red blood. If this does not happen immediately, try waiting a few minutes. If the flow is not pulsatile, it may become so if you wait a few minutes. Pick up the bleeding vessels with 4/0 absorbable suture, and *don't rely on your anastomotic sutures to control bleeding*.

You can use what we describe here as 'the 2-layer method' or you can use 'the one-layer method'. Both methods are usually done with clamps, but can if necessary be done without them. Both the descriptions here assume you are doing an end-to-end anastomosis.

END-TO-END ANASTOMOSIS WITH 2 LAYERS USING CLAMPS (GRADE 3.3)

Using bowel clamps (11-7) is the standard method, because it causes the least contamination of the peritoneal cavity. You have first to join the back of the bowel (as it lies in front of you) and then the front. The important places for leaks are the 'corners', where the back and the front parts of the anastomosis join one another, at the mesenteric and the antimesenteric borders of the bowel.

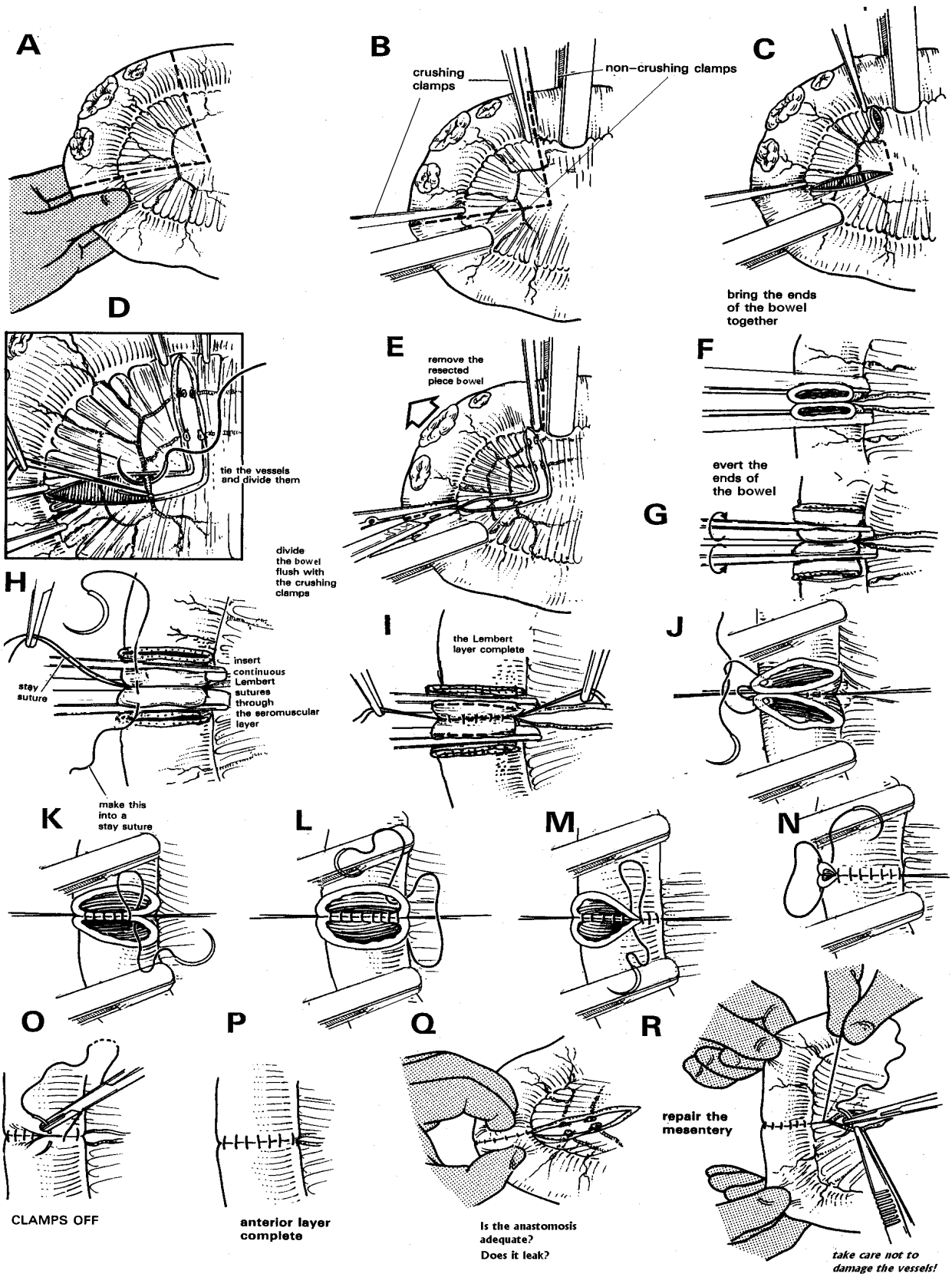


Fig. 11-7 RESECTION AND END-TO-END ANASTOMOSIS IN 2 LAYERS WITH CLAMPS.
This method uses 2 crushing clamps; it can be done without any clamps using stay sutures or tapes instead. Apply non-crushing clamps in Step B and keep them on until Step N. Remove the crushing clamps with the loop of bowel in Step E. The critical parts of this anastomosis are the inverting Connell sutures in steps J-N.

If serosa of one loop is to be in contact with serosa of the other loop at these critical points, invert the bowel here. The suture which does this best is the Connell (11-5B,C). The principle of this is that the suture starts outside the serosa and comes out into the mucosa; it then goes back into it again, and it comes out of the serosa of the one end of bowel. It then goes back into the serosa again on the other end of bowel to be anastomosed. It makes a 'loop on the mucosa' (11-5D). It is this loop which makes the mucosa invert. The bigger the bite on the outside (serosa) and the smaller on the inside (mucosa), the better the bowel ends will invert.

Decide the length of bowel you want to resect (11-7A). Apply 2 crushing clamps at each end of the non-viable bowel to be resected, including a small portion of viable bowel, and non-crushing clamps 2cm away on the viable parts of bowel to be joined together (11-7B). *Don't place clamps over the mesenteric vessels.*

If the mesentery is too thick for you to see the vessels clearly through it, even when the mesentery is held up against the light (as in the sigmoid colon, and the small bowel mesentery in moderately fat patients, especially distally), divide the peritoneal layer covering the mesentery carefully with fine scissors nearest to you to outline the vessels (11-7C). Dissect the vessels, place a small artery forceps on each and ligate them one by one, using a 2/0 or 3/0 suture (or smaller for children and babies). To save suture material, you can leave the haemostats untied on the part of bowel to be resected.

Pack away the other abdominal contents, and place one of 2 large abdominal swabs under the bowel to be resected. Make sure you protect the abdominal wound edges from contamination.

Divide the bowel on the outside of the crushing clamps (11-7E), using a sharp knife to give a clean cut. Empty the bowel of its content into a bowl, or using a guarded sucker. Bring the non-crushing clamps together (11-7F) and evert them (11-7G).

Insert a continuous Lembert suture through the seromuscular coat of the posterior layer of the bowel (the one which is furthest from you) starting at the anti-mesenteric border (11-7H), leaving one end long to act as a stay suture (11-7I), and continue till the mesenteric border, leaving the suture to hang. *Don't cut it off, or its needle*: you will use it again! Start the all-coats continuous inner layer at the antimesenteric border with a loop on the seromuscular layers of both ends of bowel, leaving one end long as a 2nd stay suture (11-7J): you can differentiate this from the first stay suture if you are using the same thread by putting a loop on it, and continue by passing inside the lumen as a single all-coats suture (11-5B-C). Continue as a simple over-and-over suture until you reach the mesenteric end (11-7K). Make sure you get your assistant to keep the right tension on the suture and let it snug down nicely on the bowel wall, so that you cannot insert a small artery forceps between the suture points.

If the sizes of the bowel ends differ, calculate taking more widely spaced bites on the bigger bowel end than on the smaller end, so that you reach the mesenteric ends of both bowel ends simultaneously. This takes some practice.

(Alternatively, use interrupted sutures: place stay sutures at the antimesenteric border of both ends of bowel, and tie these together; likewise do the same at the mesenteric border of both ends of bowel. Then place a suture midway between the 2 sutures on one end, and again midway between the 2 sutures at the other end, and tie them together. Continue with another suture midway between the first antimesenteric stay suture and this last one placed in the middle of the bowel, and so divide the bowel wall distance each time in half. That way, you will not end up with excess bowel on the distended side.)

Then, *using the same suture*, pass through from inside to outside the serosa (11-7L) and continue as an all-coats inverting Connell suture on the anterior layer (11-5A, 11-7M), till you join the beginning of the same suture at the antimesenteric end again (11-7N). Tie the 2 ends of the inner continuous suture together and cut them, leaving 5mm ends. Now remove the non-crushing clamps. You may immediately see a defect; the tension may be released by removing the clamps, making the final outside suture easier.

Continue with the first continuous Lembert suture which you left hanging long on the mesenteric border, and go round till you end at the antimesenteric border (11-7O). Tie the 2 ends of the outer continuous suture together and so complete the circle (11-7P). Test the patency of the lumen with your fingers (11-7Q). Massage some of the bowel contents past the anastomosis to test for leaks. If you are worried, place the anastomosis under water and squeeze: look for gas bubbles; if there are none, your anastomosis is sound.

Close the defect in the mesentery with continuous 2/0 or 3/0 suture, taking great care not to occlude the vessels.

END-TO-END ANASTOMOSIS BY A SINGLE-LAYER SUTURE (GRADE 3.3)

The important feature about this method is that it uses a single-layer suture.

Essentially this method is like the '2-layer' but uses a single all-coats layer, dispensing with the outer seromuscular layer; you need to be very careful to place the loops of the suture accurately and close enough together. These sutures cannot be made too neatly.

If the suture line is snug and inverted, stop at this stage. If not, complete the anastomosis with a final layer of Lembert 4/0 monofilament seromuscular sutures. This converts the one-layer method into a partial 2-layer method. You should be able to get most of the way round the bowel, but you may not be able to suture its mesenteric border.

BOWEL SUTURING METHODS

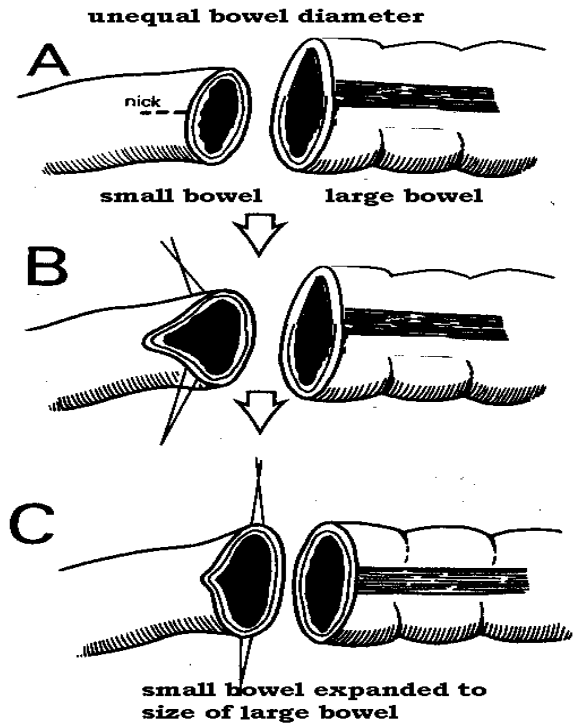


Fig. 11-8 SOME BOWEL SUTURING METHODS.
A, if the bowel that you want to anastomose end-to-end is unequal in size, B, make a cut in the antimesenteric border of the smaller piece, so C, it enlarges to the same size. *After Turnbull RB Jr. Ethicon, permission requested.*

END-TO-END ANASTOMOSIS BY THE OPEN METHOD (GRADE 3.3)

This method is indicated:
(1) When you don't have any clamps.
(2) When you cannot get clamps on to the bowel, as when joining large bowel to rectum after a Hartmann's procedure (12.10).
(3) In infants and small children, because all clamps crush their delicate bowel to some extent, although bulldog vascular clamps are less traumatic.

Hold the 'corners' of the bowel by stay sutures, and suture the posterior walls together by an all-coats suture. Instead of starting at the antimesenteric border, you may find it easier to start posteriorly opposite where it is easiest to finish. Place 2 separate Connell sutures at this point, and with each, work round anteriorly, finally tying both sutures together. The open method does however increase the risk of contaminating the peritoneal cavity and it is therefore essential to empty the bowel completely before starting.

Which parts of the bowel can you safely anastomose to which and when? You can anastomose large bowel safely if it is empty: give bowel preparation and enemas beforehand for the elective cases, or empty the bowel as described for emergency cases. If the colon is perforated or gangrenous, and particularly when there is heavy faecal soiling of the peritoneal cavity, *this is not safe especially if you only have catgut*. You should then make a diversionary colostomy (11.6).

If you are not happy that you have made a satisfactory anastomosis (no anastomosis is ever quite 'watertight'), you can bring up a loop of omentum and suture this loosely over the place which you think will leak. This is optional; there are certain occasions when it is very useful, notably the repair of a perforated peptic ulcer (13-11).

You should use preferably long-lasting absorbable sutures for the inner layer or in the one-layer method; the outer layer can use any type of suture, but long-lasting absorbable (especially in children) is best.

Remember to close the defect in the mesentery after you have completed the anastomosis, in order to prevent an internal hernia. Do this carefully so as not to pick up any blood vessels in the mesentery and damage the blood supply to the anastomosis (11-5L).

If, when you have completed the anastomosis, the bowel is not viable ('purplish'), *resect its ends and start again!*

If the loops are very unequal in size (as when anastomosing small to large bowel), you can make a small cut in the antimesenteric border of the smaller loop (11-8). The end-to-side or side-to-side anastomosis is a poor alternative, and probably more likely to leak.

If you don't have time (because the patient is too sick: damage control) **or cannot make an anastomosis** (because you have not practised), you can temporize by closing the bowel ends with tape and returning for a 2nd look laparotomy (10.1) in 48h. Alternatively, secure both ends of bowel over a piece of tubing connecting the two, especially if the proximal bowel is very distended. Plan a 2nd look laparotomy as before.

PURSE-STRING SUTURES
A purse-string suture is an invaginating suture around a circular opening, and can be used to fix a caecostomy tube (11.6) or prior to draining the gallbladder (15-1B). Use it also to bury the necrotic base of an inflamed appendix (14.1).

Place a continuous Lembert suture through the serosa and muscle only, all round the appendix. Tie the first hitch of a reef knot, pull the ends of the suture upwards, and push the stump of the appendix downwards. If necessary, ask your assistant to pull up the opposite side of the purse string as you do so. If you happen to penetrate all layers of the bowel, reinforce the purse string with some more inverting sutures.

ENTEROTOMY AND CLOSING A BOWEL PERFORATION (11-5M) (GRADE 3.2)

An enterotomy is an opening in the bowel. You may have to make one to decompress the bowel (12.4), to make a stoma (11.5), to inspect the bowel to see where bleeding is coming from (13.4), or to remove *ascaris* worms (12.5) or a foreign body (12.15). Make a longitudinal opening in the antimesenteric border of the bowel.

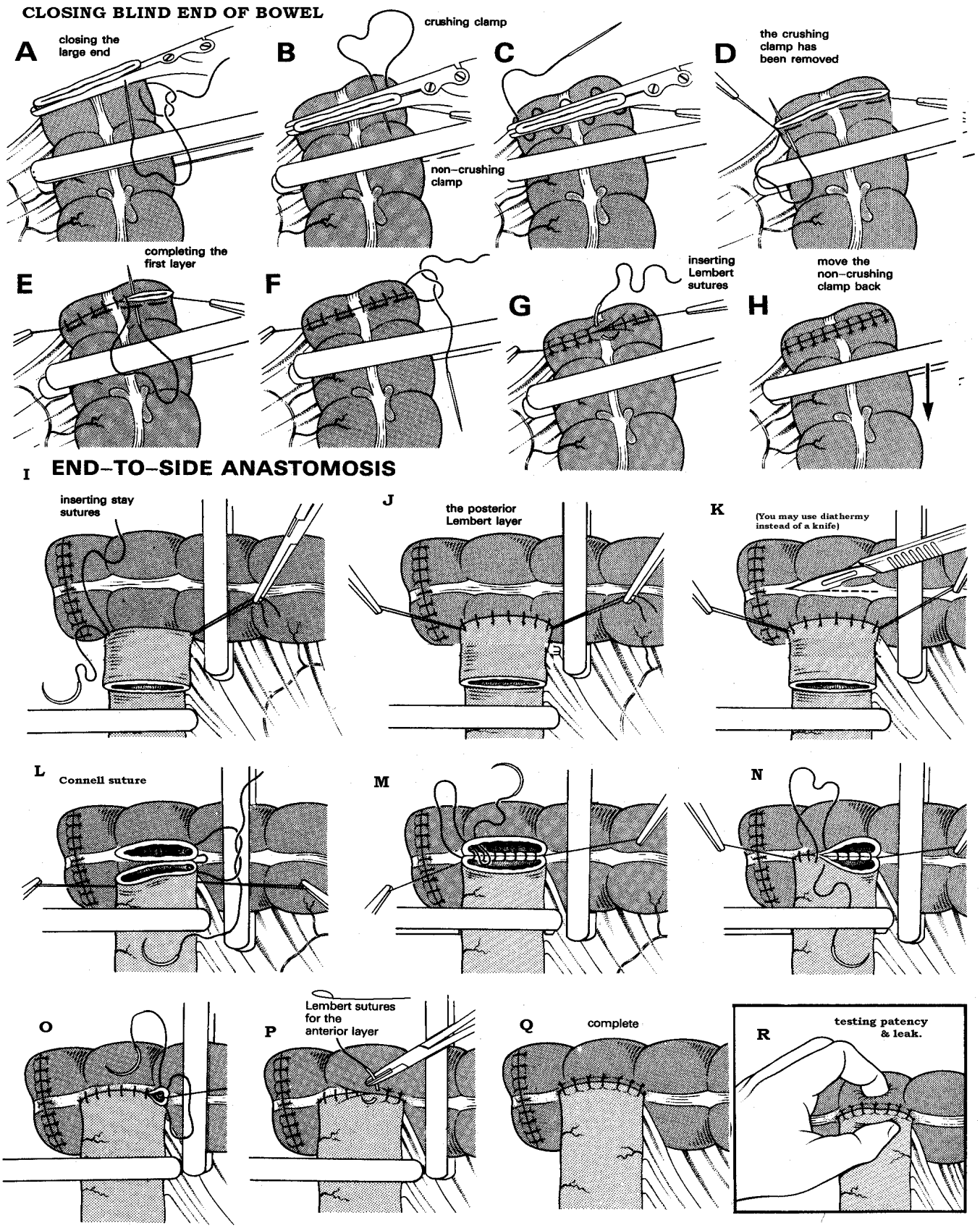


Fig. 11-9 CLOSING A BLIND BOWEL END AND END-TO-SIDE ANASTOMOSIS.
A-D, close the blind end of the bowel. E-H, invert the previous layer of sutures. I, insert stay sutures. J-K, insert posterior Lembert layer. L-M, insert the posterior all-coats layer. N-O, insert the anterior all-coats layer. P-Q, then the anterior Lembert layer. R, test its patency and check if there is a leak.

Don't close an inflamed small bowel perforation: a leak will be certain. Resect the involved segment or drain it!

Close the hole transversely in 2 layers as if you were anastomosing bowel. In this way you will not narrow its lumen. Start with a seromuscular suture just beyond the hole, leaving one end long as a stay. Continue this as a Connell all-coats suture till you have closed the hole, and tie a knot again leaving the end long as a stay. Cover this suture with a continuous seromuscular Lembert suture from just beyond the 1st knot to just beyond the 2nd, thus inverting the first layer completely. Test the closure with your fingers by milking intestinal content past it.

CLOSING A BLIND END OF BOWEL (11-9A-H, GRADE 3.2)

Using a straight or curved needle, close the end of the bowel with continuous atraumatic sutures working from side-to-side from one end to the other (11-9B,C). When you have got to the other end, pull the suture tight and remove the clamp. Work back to the end where you started, this time making over-and-over sutures (11-9D,E). Tie the ends of the suture and cut them off 5mm from the knot (11-9F). Cover the closed end of the bowel with a layer of inverting Lembert 2/0 sutures through the seromuscular coat (11-9G).

11.4 End-to-side and side-to-side anastomosis

END-TO-SIDE ANASTOMOSIS (GRADE 3.3)

INDICATIONS
(1) Constructing a Roux loop.
(2) Bypassing an inoperable colon tumour, septic or tuberculous adhesions, or as an alternative to ileocaecal resection.
(3) When there is a big difference in the size of 2 loops of bowel to be anastomosed (though the method of 11-8B is better).

METHOD. Bring the clamped loop of bowel close to the other viscus, and insert stay sutures through the seromuscular layers only (11-9I). Complete the layer of interrupted seromuscular sutures (11-9J).

Clamp the other viscus and open it preferably with diathermy so as to make a stoma equal in size to the small bowel (11-9K). Trim the loop of bowel if necessary. Start the inner all-coats layer with a Connell inverting suture (11-9L). Continue this as an over-and-over suture to the other end, and return using a continuous Connell suture for the anterior layer (11-9N).

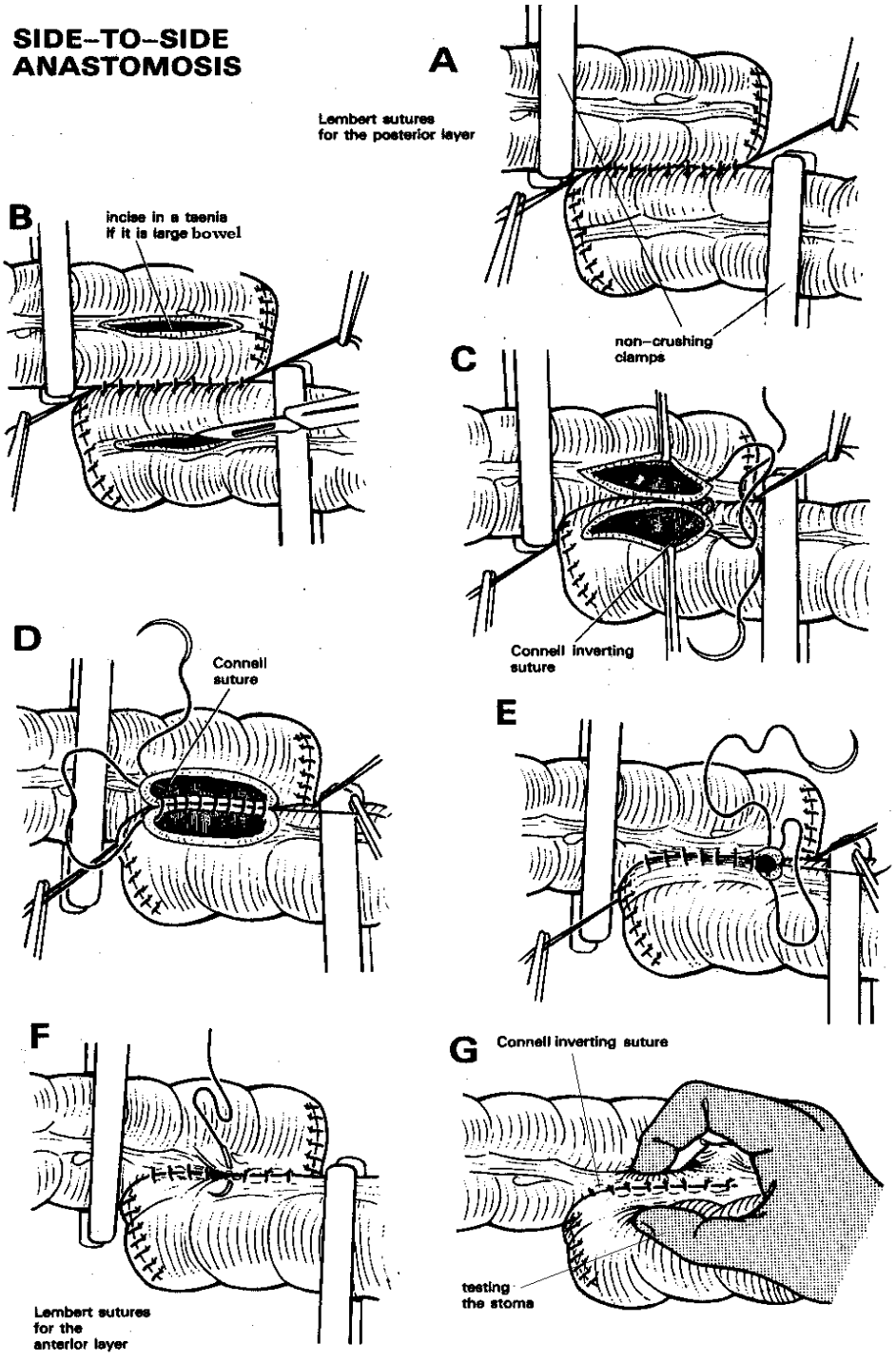


Fig. 11-10 SIDE-TO-SIDE ANASTOMOSIS is useful for creating a bypass without resecting bowel. A, hold the bowel loops with stay sutures and join them with the Lembert sutures that will form the posterior layer of the anastomosis. B, open both pieces of bowel. C, start the posterior all-coats layer with a Connell suture. D, the posterior all-coats layer has reached the other end, so now continue anteriorly as a Connell suture. E, complete the Connell suture. F, insert the anterior Lembert layer. G, test the anastomosis for patency and for a leak.

When you reach the end, tie the 2 ends of the continuous all-coats suture together and leave the ends 5mm long (11-9O).

Insert a layer of interrupted inverting seromuscular Lembert sutures (11-9P-Q). Test the lumen for its patency and any leakage: it should admit 2 fingers (11-9R). Repair the defect in the mesentery with 2/0 or 3/0 suture.

SIDE-TO-SIDE ANASTOMOSIS (GRADE 3.3)

INDICATIONS
(1) Gastrojejunostomy (13-16)
(2) Cholecystojejunostomy (15-5)
(3) Bypass without resecting bowel.

METHOD. Bring the clamped bowel close to the other viscus as before and insert a layer of continuous Lembert sutures through the seromuscular coats of both of them, starting with stay sutures at each end about 1cm from the line of your proposed incision (11-10A).

Clamp the other viscus and incise both bowel and viscus for about 3cm (11-10B) with diathermy if possible. Starting with a Connell inverting suture (11-10C), use absorbable to join the posterior cut edges of the bowel with an all-coats continuous over-and-over suture (11-10D). When you reach the other end, continue as a Connell inverting suture along the anterior layer of the anastomosis (11-10E). Finally, complete it and tie the ends of the suture together, leaving 5mm cut ends. Insert an anterior layer of Lembert seromuscular sutures (11-10F). Test the lumen of the stoma with your fingers (11-10G) and move the bowel contents over the anastomosis to check for leaks.

11.5 Stomas

In the upper part of the bowel, the purpose of a stoma (gastrostomy or jejunostomy) is usually to allow input of food and fluid for nutrition; in the lower part (ileostomy, caecostomy or colostomy), it is to let the bowel contents drain outside the abdominal wall. The bowel can become obstructed at any point. It is possible to bypass such an obstruction by making such a stoma (opening) proximally. If making an anastomosis carries a high risk of leaking, it is best to fashion such a protective stoma proximally, and close it later when you restore bowel continuity.

GHOST STOMA
An alternative method is to secure a loop of bowel, as if to make a stoma, to the parietal peritoneum with a looped feeding tube, acting as a sling, through a small hole in its mesentery, but *not open it, unless there are signs of an anastomotic leak.* If you have to, pull the bowel out and fashion a loop ileostomy. Otherwise, if all is well with the anastomosis, you can just remove the sling, and close the abdomen.

There are 2 main ways of making a stoma:
(1) Bring a loop of bowel to the surface and make a stoma at its apex, without resecting any bowel.
(2) Bring the affected loop of bowel out of the abdomen through a special opening and then resect the loop.
This leaves a proximal and distal end-stoma. If you are not skilled, it is a useful way of resecting gangrenous or injured bowel, and making a stoma without soiling the abdominal cavity.

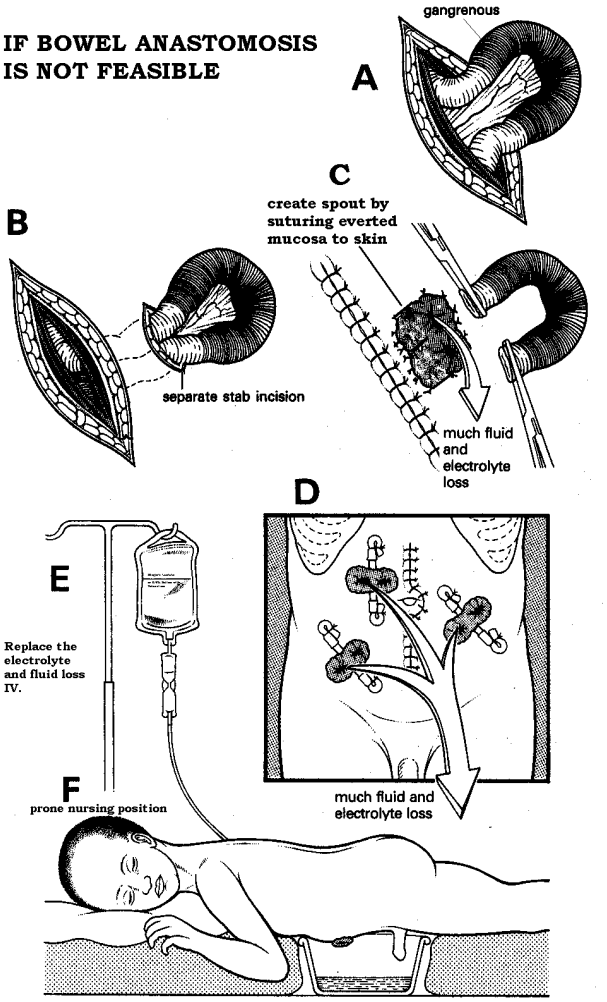


Fig. 11-11 IF BOWEL ANASTOMOSIS IS NOT FEASIBLE, *e.g.* in a typhoid perforation, bring the gangrenous segment (A) out through a separate incision (B), cut it off so as to make an ileostomy with a spout (C), and suture all coats of the bowel to the skin of the abdominal wall with interrupted sutures. D, there may be several stomas. E, there will be much fluid and electrolyte loss, which you must replace IV. F, nurse the patient prone. *After Ian Hulme-Moir.*

In many cultures having a stoma is abhorrent. So you may have to use your best persuasive skills to encourage your patient to tolerate one. Show the patient what the stoma bags look like, and *mark the site for the stoma pre-operatively.* This should be away from natural skin creases (*which may only appear on lying down, sitting or standing up*), scars, the umbilicus, and away from bony points. It should preferably be within the rectus muscle below the belt line, but be readily accessible *and visible* to the patient, so should then be above the level of the umbilicus and *not on the underside of a fat pendulous abdomen*! There should be about 6-7cm around the stoma of smooth abdominal skin to stick the stoma bag. Mark the correct site pre-operatively with indelible ink or henna paste.
Never fashion a stoma inside a laparotomy wound! (This would be like siting a toilet in the kitchen!)

Commercial bags come as one-piece or two-piece systems, with a flange to attach the bag. The advantage of the two-piece is that the stoma can be examined easily and the skin adhesion is not disturbed: they are more expensive and need a flat surface to stick nicely. They are difficult to manipulate by the elderly and by those with arthritis.
Different manufacturers' flange sizes are not usually interchangeable: pouches of the wrong size which don't attach to the flange are then useless!
The biggest problems are leaks: it helps to have sealant pastes and skin barriers, adhesive tape and supporting belts. Make sure the opening of the bag fits exactly on the stoma: measure it with a paper template.
Fortunately, most stomas are usually only needed temporarily.

TYPES OF STOMA
(a) A gastrostomy (13.9) is an opening made in the stomach for feeding.

(b) A jejunostomy (11.7) is usually used also for feeding, but by inserting a tunnelled fine tube through the wall of the jejunum.

The common sites for distal bowel stomas are (11-12): (1) the terminal ileum, (2) the caecum, (3) the right side of the transverse colon, and (4) the sigmoid colon.

(c) An ileostomy is usually made by bringing an end loop of terminal ileum through the abdominal wall. Because of the liquid bowel content full of enzymes at this point, spillage onto the skin causes rapid excoriation. To avoid this, fashion the ileostomy carefully by everting it as a spout. You still need to evert the ileostomy stoma if you are making a loop ileostomy, unless you have very good stoma appliances.

A spout ileostomy is normally sited in the distal ileum. It can be used as a conduit for urinary diversion.

A loop ileostomy can be used for bowel diversion, but is more difficult to manage than a colostomy.

(d) A caecostomy can be made by placing a tube in the caecum and letting the liquid faeces drain. This is easier than doing a transverse colostomy, but:
(1) The risks of soiling the peritoneum are greater.
(2) A caecostomy often does not work well, and needs much washing out, so it is difficult to manage post-operatively.
(3) It diverts little of the faecal stream. But, provided the tube is not too small, it may do this adequately.
(4) It can only be temporary. A caecostomy is useful if a patient is desperately ill, and you can, if necessary, fashion one under LA without exploring the whole abdomen.

(e) A transverse colostomy can be made as a loop, or double-barrelled, or as a spectacles colostomy. Always make it in the right side of the transverse colon. This should not be difficult unless the colon is very distended, or the mesocolon is short.

(f) A sigmoid colostomy is an alternative to a transverse colostomy. Here again you can make a loop, or adjacent or end colostomy (as in the Hartmann operation, 12.9)

STOMAS

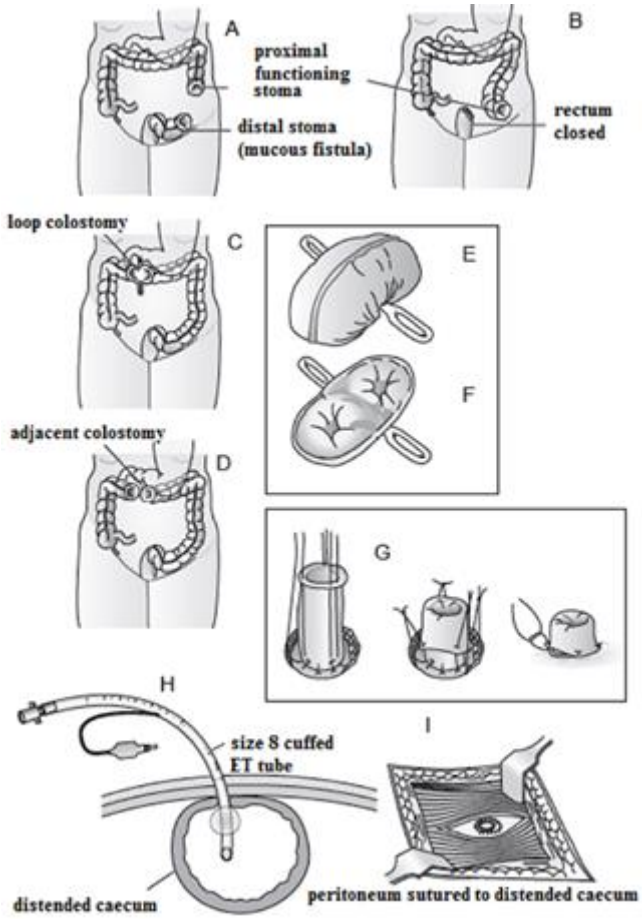


Fig. 11-12 STOMAS.
A, proximal descending colostomy and distal mucous fistula. B, Hartmann's operation: rectum closed. ***(In an abdomino-perineal resection, the rectum is removed.)*** **C, loop transverse colostomy. D, adjacent (double-barrelled) colostomy. E, loop colostomy prepared over a rod. F, loop colostomy opened with mucocutaneous sutures. G, Brooke ileostomy: (1) advance the bowel through the abdominal wall and secure it to the anterior rectus sheath; (2) place sutures through the cut edge of bowel, with a seromuscular layer at skin level and subcutaneous tissue to evert the spout; (3) completed ileostomy with mucocutaneous sutures. H, caecostomy using a size 8mm endotracheal tube. I, purse string and suture fixing the caecum to the peritoneum.** *After Hampton BG, Bryant RA. Ostomies and Continent Diversions, Mosby 1992 p.359-67.*

There are various types of loop or end colostomy:
(1) **A loop colostomy** brings a loop of bowel out of the abdomen over a short length of rubber tube, or a glass rod. This is the easiest stoma to make and close, and is suitable for most purposes. However, it does not completely defunction the distal colon.

(2) **A double-barrelled colostomy** is a loop colostomy modified by suturing the last few centimetres of its limbs together inside the abdomen, so that they resemble a double-barrelled shotgun. You can then later crush the spur (wall) between the 2 loops to make the colostomy easier to close, *but this method has dangerous complications.*

(3) **A 'spectacles colostomy'** has limbs that are separated by a small bridge of skin (11-14E). It is useful: (a) if a patient needs a colostomy for a long time, and (b) during the repair of a recto-vaginal or vesico-vaginal fistula, when work on the rectum and bladder has to be completed before the fistula can be closed.

(4) **An end (terminal) colostomy** forms the 'end' of the bowel after excision of the sigmoid in Hartmann's operation (12.9) or excision of the rectum.

(5) **A mucous fistula (colostomy)** is a stoma of the distal defunctioned large bowel, discharging only mucus.

N.B. Closing stomas can be more difficult than making them. A tube caecostomy will usually close by itself once the tube is removed, but a surgical procedure is needed to close a loop transverse or sigmoid colostomy, or ileostomy. Reversing Hartmann's operation is much more difficult (12.10). Leaving a stoma permanently may be safer for the patient, although much less popular.

11.6 Fashioning and closing stomas

There are some important general principles: always try to bring a stoma out through a separate smaller incision, and not through a laparotomy incision, because the wound is much more likely to become infected, and perhaps burst. Remember that psychological adjustment will be necessary, centred around the problems of *sight*, *smell*, *sound*, *sex*, *stigmata* and *secretiveness.*

ILEOSTOMY (GRADE 3.3)
INDICATIONS
(1) A gangrenous caecum.
(2) As diversion in very severe inflammatory colitis or megacolon.
(3) In exteriorization for ileocolic intussusception (12.7).

METHOD. Mark the site for the ileostomy beforehand. Cut out a 3cm circular disc of skin and subcutaneous fat with a knife, picking up the centre with a tissue forceps. Make an incision in the rectus sheath, split the rectus muscle 4-5cm wide, and open the posterior sheath and peritoneum.

For an end ileostomy, exteriorize 6-8cm of clamped ileum with its mesentery intact through the hole in the abdominal wall. Make sure the clamp does not come off! (If you don't have small clamps that will pass through the hole, tie the bowel with a tape.) Evert the bowel as a spout projecting 3cm from the abdominal wall (11-12G), and join the bowel and skin edges with interrupted absorbable sutures.

For a loop ileostomy, bring out a loop of ileum, without disturbing its mesentery. To do this, it is easiest to pass a thin lubricated rubber sling through a small hole in the mesentery adjacent to the bowel, and pull gently on this through the hole in the abdominal wall while simultaneously manoeuvring the bowel from inside. Check that the mesentery is not twisted or strangulated by the rectus muscle. Fix the ileum to the rectus sheath with some seromuscular absorbable sutures. After closing the abdomen, remove the clamp or tape off the ileum and trim it clean.

Apply a bag, making sure the opening is *exactly* the size of the stoma, *otherwise it will inevitably leak.* You can protect the skin by applying linseed oil.

CAECOSTOMY (GRADE 3.2)
INDICATIONS
(1) Penetrating injuries of the caecum.
(2) A grossly distended caecum about to burst.
(3) An obstruction proximal to the mid transverse colon, if you feel unable to perform a right hemicolectomy.
(4) Large bowel obstruction, if the patient is *in extremis* and too ill for a colostomy, or if you are inexperienced.

RADIOGRAPHS. Before you start, make sure exactly where the caecum is. Look for its gas shadow on the radiograph. It can be surprisingly high. Percuss the abdomen to make sure.

EQUIPMENT. An 8 or 9mm cuffed endotracheal tube. *(This is wider and less floppy than a catheter, so blocks less and is easier to insert, and causes fewer leaks.)*

METHOD
In extremis, this operation can be done under LA. If the abdomen is not already open, with the greatest possible care, make a gridiron incision well laterally over the dilated caecum: *you can easily burst it*. Put packs round the wound inside the abdomen to minimize the consequences of spillage. Have suction instantly available.

Partly deflate the caecum by wide bore needle aspiration: this releases air under tension (after placing a purse-string suture). As soon as you have done this, its walls will become thicker and more vascular.

N.B. Don't expect at this stage to evacuate faecal matter.

Try to mobilize the caecum to deliver part of it out of the abdomen, assisted by Dennis Browne forceps if necessary. *Beware, though, if it is very thin and likely to burst! If you cannot do this easily, don't attempt to do so.* Suture the cut edges of the peritoneum around the caecum, leaving enough space to insert a 2/0 atraumatic purse string 5cm diameter circle on the caecum itself (11-12H,I). Pick up its seromuscular layer only. *Don't penetrate its mucosa*. Leave the sutures long, hold them in haemostats and leave them untied.

With suction immediately ready and attached to the tube and the surrounding area carefully packed off, make a small cut in the centre of the purse string taking care not to cut the suture itself. Grasp the 8mm size endotracheal tube and, using a screwing movement, quickly push it through the cut in the centre of the purse string. Immediately tighten the purse string to secure the tube in place.

Inflate the balloon and gently pull on the tube to bring the caecum to the abdominal wall (11-12H).

CAUTION! Make sure the tube can drain *via* the suction off to the side, so that it does not flood the abdomen.

Close the muscle layers of the abdominal wall, but leave the skin open as the wound is likely to become infected. Suture the tube to the skin to prevent it being pulled out, cut it to a convenient length and connect it to a drainage bag or bottle. After 36h, flush it out with 1-2L saline, which need not be sterile, at least bd. After 10-14 days, the tissues will become adherent enough for you to remove the tube. The caecostomy will close on its own provided there is no longer any distal obstruction.

DIFFICULTIES WITH A CAECOSTOMY

If the caecum bursts with a puff of gas as you open, suck vigorously. This will not be a major disaster if you have previously sutured the cut edges of the peritoneum to the caecum, and so isolated the peritoneal cavity; pass the caecostomy tube into the tear in the burst caecum, provided it is not necrotic or gangrenous, and secure it as before.

If the caecum bursts as you are opening the abdomen, suck vigorously and quickly grab the caecal wall in order to introduce the tube, or exteriorize it. If you fail, perform a formal laparotomy.

If the caecum is gangrenous but has not yet perforated, perform a formal laparotomy and resect it. You can either make an end-to-end ileocolic anastomosis or you can close the distal colon, perform an ileostomy and then close this 3wks later. You will have to correct the subsequent ileostomy fluid and electrolyte loss.

TRANSVERSE LOOP COLOSTOMY (GRADE 3.3)

INDICATIONS

(1) Obstruction of the distal ⅔ of the transverse colon.
(2) A penetrating injury of the transverse colon.
(3) Gangrene of part of the transverse colon owing to strangulation or interference with its blood supply.
(4) To divert faeces prior to repair of a recto-vesical or recto-vaginal fistula.
(5) Protecting an anastomosis for sigmoid volvulus after resection.
(6) To defunction the bowel for Hirschsprung's disease (33.7).

A transverse colostomy is not difficult to fashion, and is better than a caecostomy. There are 3 types:
(1) A plain loop.
(2) An adjacent colostomy.
(3) A 'spectacles colostomy' (11-14).

CAUTION!

(1) Make the incision well to the right.
(2) It must be high enough to avoid the umbilicus, but not so high that the transverse colon cannot reach it.
(3) Make it just large enough to take the loop comfortably.
(4) Make sure you have picked up the transverse colon and not the stomach or the sigmoid colon! The transverse colon has *taeniae* (unlike the stomach), and is attached by a short omentum to the greater curvature of the stomach.

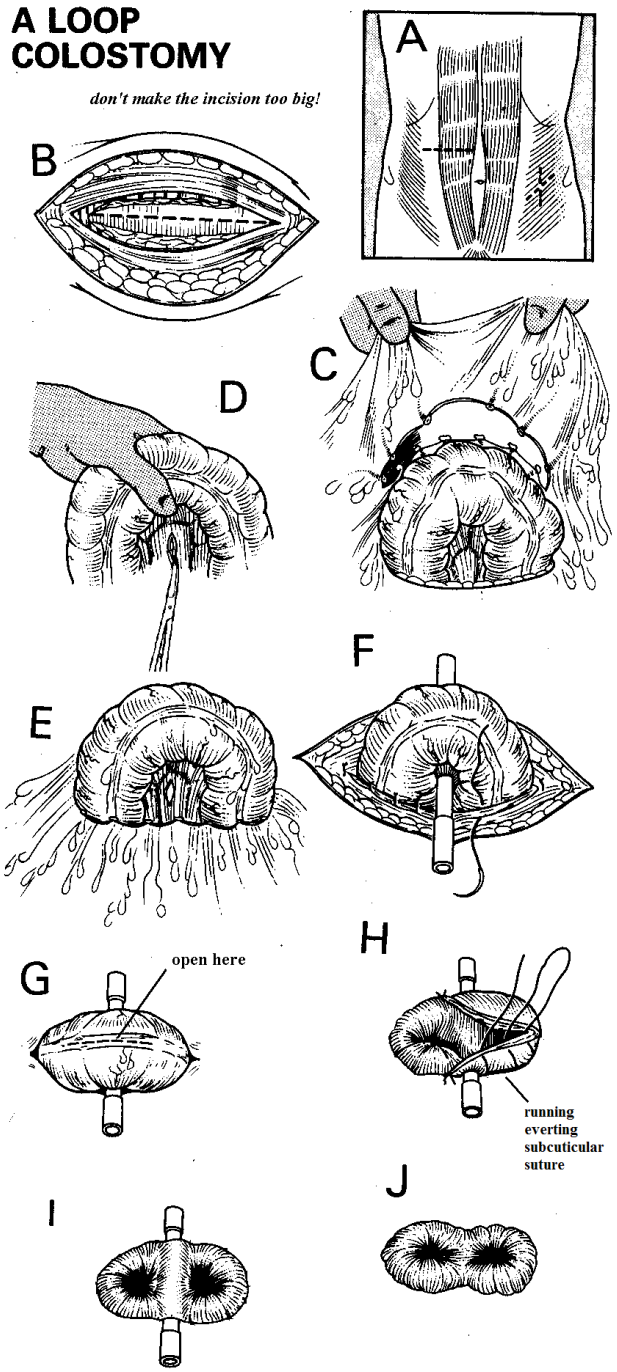


Fig. 11-13 LOOP COLOSTOMY.
A, make these incisions for a transverse or sigmoid colostomy. For a transverse colostomy: B, incise the rectus muscle. C, incise the greater omentum. D, incise the mesentery. E, bring the colon through the greater omentum. F, push a piece of tube or a glass rod through the hole, and fix the colon to stop it falling back. G, close the wound. H, open the colon and suture it to the edges of the skin wound. I, complete the colostomy. J, after healing of the wound. *After Goligher JC. The Surgery of the Anus, Rectum and Colon. Baillière Tindall 4th ed 1974 Figs 347-361 with kind permission.*

Choose an area of the transverse colon to the right of the midline. Trim off the omentum attached to 7-10cm of its anterior surface so as to make a gap in it (11-13C). Try to avoid tying any small vessels that may be present. Deliver a loop of the transverse colon through this gap. Make a small window in the transverse mesocolon next to the mesenteric border of the colon (11-13D).

Do this by pushing a large blunt haemostat through the mesentery close to the wall of the bowel, while you open and close its jaws. *Avoid injuring the branches of the middle colic artery as you do so*. Pass a rubber sling through the window you have made, and grasp both its ends with a haemostat. Test the colon for mobility again. If it is very tense and distended, decompress it.

CAUTION! You must be able to deliver the loop of colon you have isolated through the transverse incision comfortably. If you cannot do this, mobilize the colon more; a poor alternative is to open the colon, and suture the bowel edges directly to the abdominal wall skin.

This is a **'blow-hole colostomy'**. To prevent the bowel from detaching, fix it to the inner abdominal wall with 4 sutures. If you are still uncertain about its fixation, insert a Foley catheter into the stoma, blow up the balloon sufficiently to hold the bowel against the abdominal wall and apply a little traction on the catheter.

METHOD

Mark the site for the colostomy beforehand. Make a 6-7cm separate transverse skin incision above and to the right of the laparotomy incision (11-13). Divide the anterior rectus sheath in the same line as the skin. Stretch the rectus muscle transversely to admit 2 fingers only. Open the posterior rectus sheath and peritoneum.

Push a 2nd haemostat through the transverse abdominal incision, and grasp the sling you have placed round the colon. Release the first haemostat, and by pulling with one hand and pushing with the other, withdraw the loop of colon, so that it comes out through the incision and rests on the abdominal wall.

If the wound is loose enough to let you insert a finger alongside the loop of colon, there will be no risk of the lumen occluding, and the colostomy should function satisfactorily. If the colon is not loose enough, extend the incision.

Once you have closed the abdomen, pass a short piece of thick rubber tube, or a short glass rod attached to a piece of rubber tube, through the window occupied by the catheter (11-13F), and keep it there with 2 sutures anchored to the skin. Fix the bowel to the fascia of the abdominal wall with 2-4 seromuscular sutures. Leave the rod *in situ* 5-7 days.

CAUTION! Before you place these sutures, make sure the colon is not twisted, and that it runs transversely, as the transverse colon should.

Open the colostomy immediately, by making an incision longitudinally along a *taenia* (11-13G,H). It will open to form 2 stomas (11-13I). Evert the bowel wall and join the open edges of bowel to the skin with continuous absorbable sutures (11-13H,I), putting them through the full thickness of the bowel wall, but subcuticularly in the skin. (This ensures the bowel edge overlaps the skin and bowel content cannot flow under it.) Push a finger down the afferent loop to make sure that it is patent; a gush of gas and faeces is an encouraging sign. Apply a colostomy bag (11-15).

SIGMOID COLOSTOMY (GRADE 3.2)

INDICATIONS

(1) Wounds of the rectum.

(2) Chronic inoperable obstructive rectal lesions including carcinoma.

(3) To divert faeces prior to repair of a recto-vesical or recto-vaginal fistula repair.

(4) To defunction the bowel for imperforate anus (33.6).

(5) To divert faeces in uncontrollable faecal incontinence, especially because of HIV disease.

METHOD. Mark the site for the colostomy beforehand. Proceed as for a transverse colostomy (though there is no greater omentum to draw the sigmoid through) pulling out a loop of sigmoid colon: this is usually readily mobile.

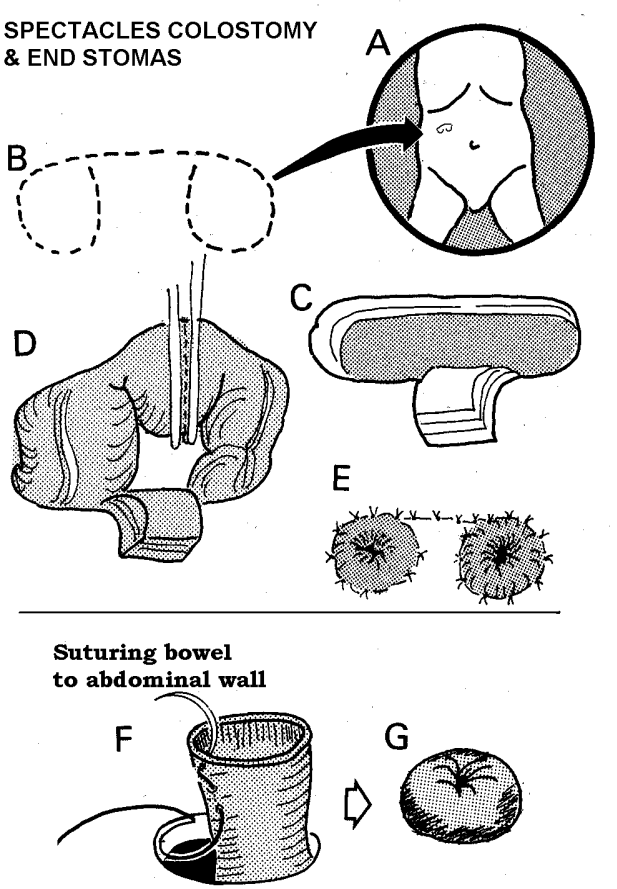


Fig. 11-14 SPECTACLES COLOSTOMY AND END STOMAS.
A, site of incision for a spectacles colostomy. B, spectacles incision. Remove the skin inside each loop. C, turn back the flap. D, exteriorize the transverse colon and clamp it with 2 non-crushing clamps. E, colostomy completed. F-G, secure method of everting colon onto the abdominal wall.

To make a spectacles colostomy, for complete faecal diversion, make the incision (11-14B) and remove the skin inside each loop. Turn back the flap (11-14C), and divide the clamped colon (11-14D) to make the colostomy with separated loops (11-14E).

END (TERMINAL) COLOSTOMY (GRADE 3.3)

INDICATIONS

(1) As part of Hartmann's operation (12.9).
(2) A permanent colostomy, as in an abdomino-perineal resection of the rectum.
(3) In exteriorization of an ileocolic intussusception (12.7).

METHOD

Mark the site for the colostomy beforehand. Draw out the end of the bowel with a clamp or tape attached to avoid spillage. Before you close the abdomen, put in a few absorbable sutures between the seromuscular coat of the bowel and the anterior rectus sheath of the abdominal wall to reduce the risk of prolapse and to stop the bowel falling back into the abdomen after you have closed it! Make sure that there will be 1.5cm of healthy bowel protruding beyond the skin. Try to close the lateral space between the colostomy and the abdominal side wall; then close the abdomen.

To open the colostomy, cut off the crushing clamp with a sharp scalpel. Control bleeding. Suture mucosa to skin all round with continuous absorbable sutures which take a bite of the full thickness of the colon, and run subcuticularly in the skin. You will find the colostomy will evert itself beautifully (11-14G). *Don't attempt to dilate the stoma with 2 fingers!*

If a baby with imperforate anus has a grossly distended colon, make the incision as before and put gauze swabs around the incision edges. Place a purse string, preferably over the *tinea*, and decompress the bowel with a stab incision at the centre of the purse string. The bowel will then collapse and become easy to manipulate.

DIFFICULTIES WITH COLOSTOMIES

If the colostomy 'flows like a river', this is likely to be a good sign in the early stages, because it means that the obstructed bowel is emptying itself. If it happens later, treat with kaolin mixture with 30-60mg codeine phosphate tds, and advise against drinking orange juice.

If the colostomy does not work, put a finger into the afferent loop to make sure that it has not become occluded. Twist your finger round gently inside the bowel lumen beyond the level of the rectus muscle to irritate the bowel. If this fails to start it 30-60mins later, get the patient to drink orange juice and mobilize. If this also fails, put a glycerine suppository into the afferent loop, or instil enema solution in using a Foley catheter with the balloon gently inflated to prevent the irrigation spilling out. If it is still not working after 3 days, check abdominal radiographs to see if there is proximal obstruction.

If an abscess forms next to the colostomy, drain it (preferably under LA) and allow it to drain into the stoma bag.

If the bowel forming the colostomy necroses and becomes dark purple, you probably damaged its mesentery by stretching or compressing it into too small a hole. Put a glass tube into the stoma and shine a light onto it to see how deep the necrosis extends. (You can get a better view if you pass an endoscope down the stoma.)

If it is more than superficial, return to theatre, enlarge the opening in the abdominal wall, and make a fresh colostomy by bringing out more bowel, and resecting the necrotic black or purple part. *Don't delay doing this, hoping the bowel will improve:* the risk is of further bowel necrosing and peritonitis resulting.

N.B. Take care you distinguish necrosis from *melanosis coli*, the blackish appearance of bowel from anthracene laxative abuse (*e.g.* senna). This bowel bleeds if you pinch it with forceps; *necrotic bowel does not.*

If the colostomy retracts, it will contaminate the peritoneum and cause faecal peritonitis if the bowel separates completely. You then need to re-open the abdomen to correct this. Retraction occurs because of:
(1) the mucocutaneous sutures giving way.
(2) chronic peristomal skin infections.
(3) inadequate mobilization of the bowel mesentery.
(4) too early removal of the rod supporting a loop colostomy.
(5) excessive weight gain.
Retraction makes it difficult to get a bag to stick.

If the colostomy stenoses, dilate it gently with sounds, using a lubricant, *not with 2 fingers*. It may be that the fascia or skin is too tight; if so, release it under local anaesthesia. If it is the result of a severely retracted stoma, refashion it. Check for recurrent inflammatory bowel or malignant disease at the stoma site.

If the skin around the colostomy becomes oedematous and inflamed, necrotizing fasciitis of the abdominal wall has developed because of leakage of faeces into the subcutaneous tissues. Return immediately to theatre to debride the affected skin and fascia widely, and refashion a colostomy in a different site.

If a hernia forms around the colostomy, it will probably only be a little bulge, and is unlikely to grow big. It has occurred because the opening for the colostomy is too big or the stoma has been placed lateral to the rectus muscle. You may be able to close the colostomy opening better by inserting sutures from fascia to the seromuscular layer of the bowel, but this is rarely necessary. Occasionally a hernia comes through at the side of a colostomy: this needs revision (18.13).

If signs of intestinal obstruction develop (12.4), adhesions may be forming inside the abdomen at the site where the colostomy emerges, or from the original disease process. They are no different from the adhesions developing after any other abdominal operation. Explore the abdomen if there is no improvement.

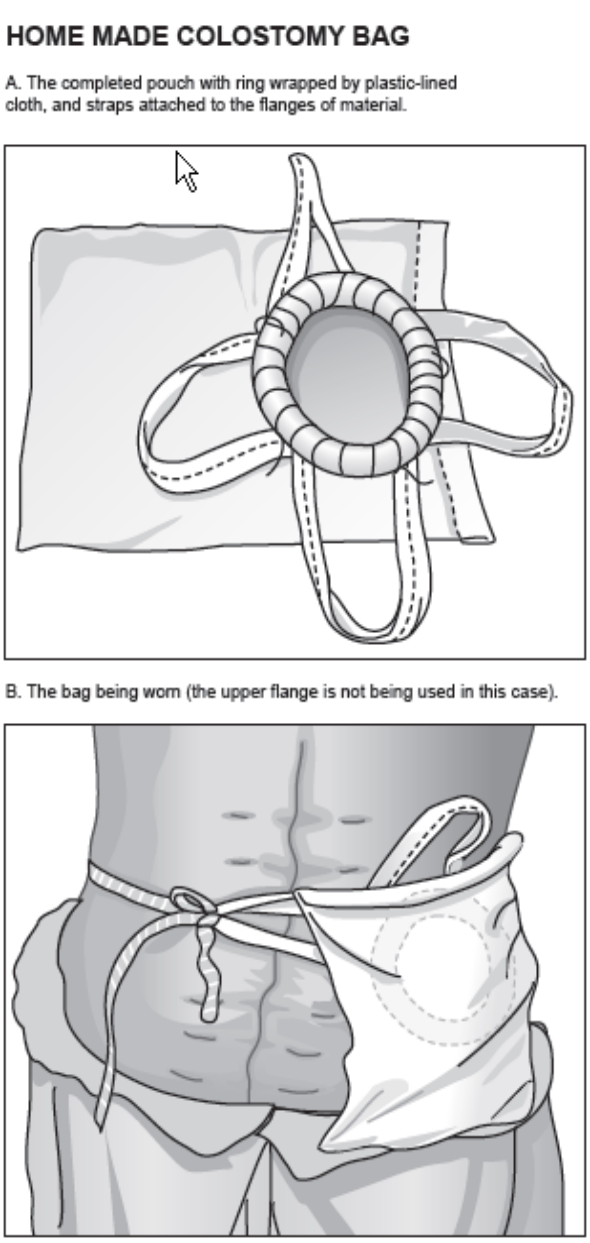

HOME MADE COLOSTOMY BAG
A. The completed pouch with ring wrapped by plastic-lined cloth, and straps attached to the flanges of material.
B. The bag being worn (the upper flange is not being used in this case).

Fig. 11-15 A HOME-MADE COLOSTOMY BAG.
You can make a functioning colostomy bag from an ordinary plastic shopping bag, a rubber ring and cloth. A, make a pouch 10x12cm and line its inside with a plastic bag: this can be open at one end or, better, closed around its 4 sides. Outline on the pouch a circle the size of the stoma, which should be protruding from the abdominal wall, and cut this out making a hole. Attach 4 flanges of cloth to a rubber ring 1cm larger than the patient's stoma. Place the ring over the hole, and roll the cloth/plastic surface over the ring and secure this with elastic bands, making sure the plastic is on the outside. It is best that the rubber ring is malleable so that it can be moulded onto the abdominal wall around the stoma. Karya gum will help seal the skin of leaks. B, attach straps to the flanges to hold the pouch in place around the abdomen, and if necessary over the shoulder. *After Zafar A, Malik E. Hand-made colostomy bag. Tropical Doctor 2002: 32(4):241, with permission.*

N.B. Neonates don't need a colostomy bag: the ordinary nappy will suffice, but it still needs changing every time the baby passes a stool!

N.B. Once the stools have become firmer (if necessary with the use of loperamide), patients may be able to function perfectly well by tying layers of cloth round their belly over the stoma, and removing these on a daily basis to empty and clean the stoma. The colon functions as its own reservoir.

If the colostomy prolapses, it will look just like a prolapse of the rectum. Bowel protrudes as an intussusception; you can usually push it back. This is quite common, especially in infants, and embarrassing. It is caused by too large an opening or inadequate fascial support for the colostomy.

If you cannot reduce it, put fine-ground or icing sugar on the prolapsed bowel for 2-3 days: it will then reduce in size and allow you to reduce it. Hyaluronidase (1,500U in 10ml water) injected into the stoma will also reduce the oedema and allow you to reduce the stoma. Persistent prolapse or necrosis will require a revision of the colostomy.

If the skin excoriates around the colostomy, try to reduce the fluidity of the output with kaolin and codeine phosphate. Make sure the bags fit nicely and don't leak: use sealant pastes.

If some varices develop around the colostomy, this is a sign of portal hypertension (usually due to cirrhosis or *schistosomiasis*).

CLOSING A COLOSTOMY (GRADE 3.3)
Do this *4-6wks later*, when the wound is healthy and there is good recovery from the original operation. Check that any distal anastomosis or repair is sound by introducing dilute Barium contrast through the distal stoma loop, or *via* the rectum, and taking radiographs.

CAUTION!
(1) The patient will be hoping for this as soon as possible. *Don't let him persuade you to do it too early*.
(2) It is not an easy operation.
(3) Make sure there is a loop colostomy and *not an end colostomy* by examining it carefully with your fingers, feeling for the afferent and efferent loops.

Wash out the bowel proximally, and distally through the rectum. Repeat this daily for 2-3 days before the operation. Treat with magnesium sulphate 10g to help empty the proximal bowel and to make sure that the next faeces passed will be soft. Use gentamicin, or chloramphenicol and metronidazole, as perioperative prophylaxis (2.9).

To minimize bleeding, infiltrate the skin and subcutaneous tissues around the colostomy with LA solution containing adrenaline 1:200,000. This infiltration is also valuable in demonstrating tissue planes.

Insert traction sutures round the colostomy (11-16A). Make an elliptical incision round it (11-16B). Use sharp scissors to dissect it free from the surrounding skin and fascia, and from the muscle of the abdominal wall (11-16C). (You can keep a finger in the lumen to tell you when you are getting dangerously close to it.) Raise the ellipse of skin from the abdominal wall (11-16D). Using sharp dissection, clean the sheath of the rectus muscle until you reach the edge of the opening through which the bowel is passing. Change any soiled gloves.

Free the parietal peritoneum round the circumference of the opening. Divide any adhesions that may be present. Draw the colon gently out of the incision, and place packs over the wound. Trim away the everted edges of the bowel (11-16E). Close it transversely with Connell sutures (11-16G,H).

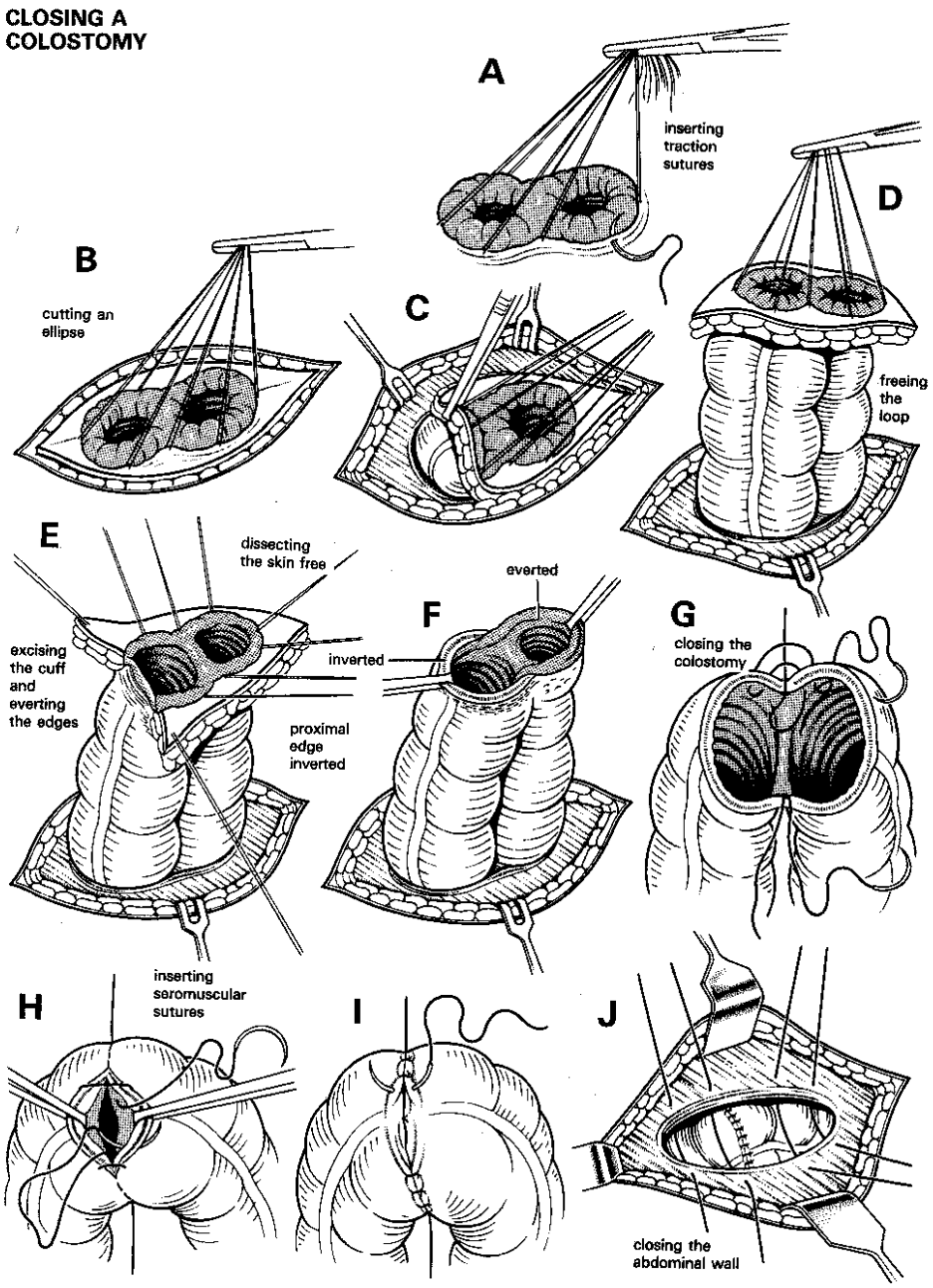


Fig. 11-16 CLOSING A LOOP COLOSTOMY.
A, insert traction sutures. B, raise an ellipse of skin round the colostomy. C, dissect an ellipse of skin free from rectus sheath. D, free the colostomy loop. E, excise a cuff of skin and evert the bowel edges. F, evert the proximal bowel edge; the distal bowel edge is still inverted. G-H, close the colostomy with Connell loop-on-mucosa sutures. I, insert a 2nd layer of seromuscular Lembert sutures. J, close the muscles of the abdominal wall in one layer. *After Maingot R. The Management of Abdominal Operations, HK Lewis, 4th ed 1961 Figs 396-404 with kind permission.*

Start by placing 2 atraumatic sutures through all the coats of the bowel where the proximal and distal colon meet. Tie the knot in the lumen, and work from each side. Finish with a seromuscular continuous Lembert suture (11-16I). Test the patency of the lumen with your fingers and return the colon into the abdomen. Repair the defect of the rectus sheath (11-16J), and close the stoma opening with a subcuticular purse-string suture: this allows a small hole to drain fluid and prevents infection and gives a neat scar avoiding 'dog ears'.

DIFFICULTIES WITH COLOSTOMY CLOSURE

If you are not sure what sort of colostomy there is, put a gloved finger (or two) into the stoma. *Make sure you don't close an end colostomy!* This needs anastomosing to the distal colon or rectum (12.10).

If there is a 'blow-hole colostomy', insert a Foley catheter into the bowel and inflate the balloon to hold onto the colon and prevent it falling into the abdomen and leaking content during closure.

If you perforate the colon whilst mobilizing it, mark the defect with a tissue forceps or suture and continue full mobilization of the loops of colon. Resect down to intact well-vascularized bowel, and make a formal end-to-end anastomosis (11.3).

If you cannot mobilize the colon because of adhesions, make a formal laparotomy to divide the adhesions from inside. You will need a good light and an assistant providing good retraction.

If the wound leaks faecal matter, there is a fistula (11.15). Open the wound, clean it thoroughly and apply a bag. Restrict the oral intake initially to fluids, and later to a low-residue high-calorie diet. The fistula should close in 3-4wks.

11.7 Feeding jejunostomy

Occasionally, you may need to make a feeding stoma to allow enteral nutrition if there is proximal obstruction. This is an alternative to a gastrostomy (13.9). Feeding jejunostomies are seldom needed, but they can be life-saving: for example, when a suture line in an injured duodenum needs protecting. To reduce the danger of a leak, introduce the tube into the bowel through a long oblique track.

You may occasionally use a feeding jejunostomy in a high-output fistula, using Foley catheters (11.15).

FEEDING JEJUNOSTOMY (GRADE 3.2)

INDICATIONS
(1) An oesophageal obstruction which is correctable.
(2) To protect a suture line in the duodenum following an injury or operation for duodenal atresia.
(3) To protect a suture line in the stomach which has leaked.
(4) A pancreatic abscess.

METHOD
Make a small laparotomy in the upper abdomen. Find the upper jejunum by following it downwards from the ligament of Treitz. Confirm you have found the duodeno-jejunal junction by identifying the inferior mesenteric vein along its left border and feeling it emerge from its fixed position behind the peritoneum. Take a loop about 25cm from the duodeno-jejunal junction, and make an incision on its antimesenteric border through the longitudinal muscle layer for about 8cm. At the distal end of this, make a hole through into the lumen. Insert a feeding catheter (Ch18 for an adult), or a long Ryle's tube, through this hole for about 10cm. Close the bowel around it with continuous absorbable suture (11-17C).

If the bowel wall is thin and you are afraid of tearing it, make a hole in the jejunum for the feeding tube, and then create a tunnel to bury it by suturing together the bowel wall longitudinally over it.

Make a 2nd incision in the abdominal wall at least 8cm lateral to the midline (to avoid the epigastric vessels) above where this loop of jejunum will comfortably lie. Draw the end of the tube back through the abdominal wall. Fix the jejunum longitudinally to the inside of the abdominal wall together with an absorbable suture, so that the jejunal suture line is now extra-peritoneal, *taking care not to create a space for an internal hernia*. Close the abdomen and anchor the tube to the abdominal wall with a 'Saxon stocking' type of anchoring suture (11-17E), attached to a purse string.

To remove the tube, snip the ligature anchoring it to the skin, and pull. The long oblique tunnel through the mucosa and submucosa will seal rapidly. The purse string anchoring it to the peritoneal wall will prevent the jejunal contents soiling the peritoneal cavity.

DIFFICULTIES WITH JEJUNOSTOMY
The tube readily blocks, especially if it is of narrow bore. Always flush it with water after use. *Don't introduce crushed tablets down the tube:* they will block it. If this has happened, try passing a long guide-wire gently down the tube, if flushing it does not clear the blockage. Make sure the tube is not kinked or twisted. Carbonated drinks or hydrogen peroxide may succeed in flushing the tube, if water does not. Otherwise, introduce some gastrografin down the tube and take a radiograph to see where it is blocked. In the last instance, you may have to replace it.

If the patient has persistent diarrhoea after feeds, you may have placed the feeding tube in the ileum. Insert some contrast material down the tube and take some abdominal radiographs quickly afterwards to see if its passage in the small bowel is very quick and short.

Rarely, a type of necrotizing enterocolitis occurs after feeding is started via a jejunostomy (14.4).

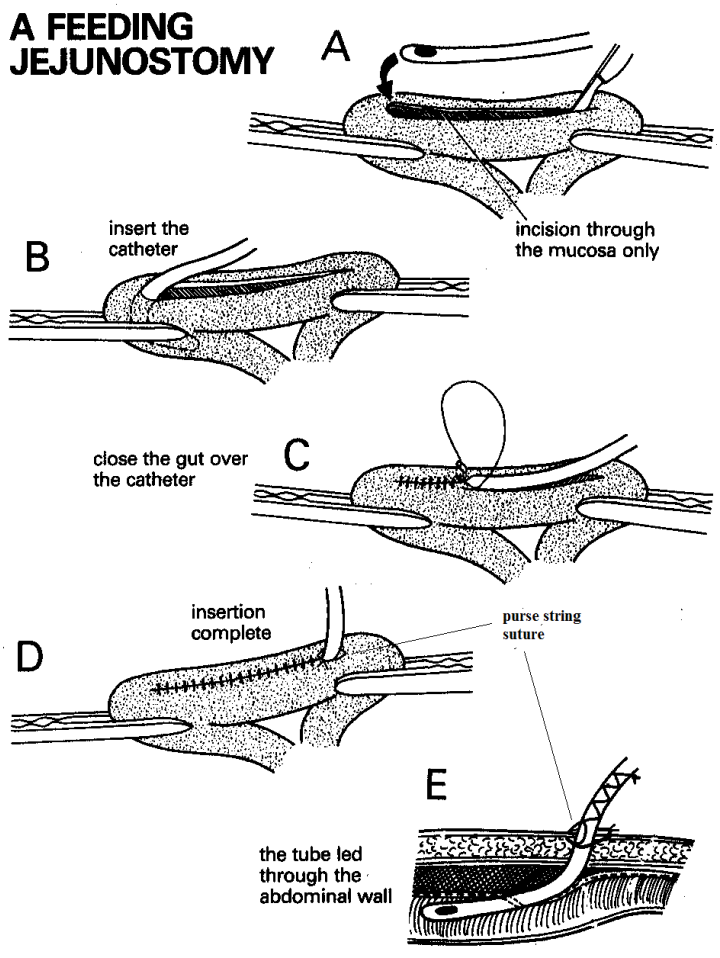


Fig. 11-17 MAKING A FEEDING JEJUNOSTOMY.
A, the incision. B, insert the tube. C, close the jejunum over the tube with a continuous absorbable suture. D, fix the tube in the bowel. E, lead the tube out through the abdominal wall, and fix the jejunum longitudinally to it, with a purse string.

11.8 Draining and closing the abdomen

After a laparotomy, consider if you wish to close the abdomen primarily. Leave it open (11.10) if closure would be too tight because of bowel distension or packs left in the abdomen, or if you plan a 2nd look laparotomy (10.1). Occasionally, even if you cannot safely close the fascia, you can close the skin: in this case, do so with interrupted mattress sutures. This is easier to handle post-operatively.

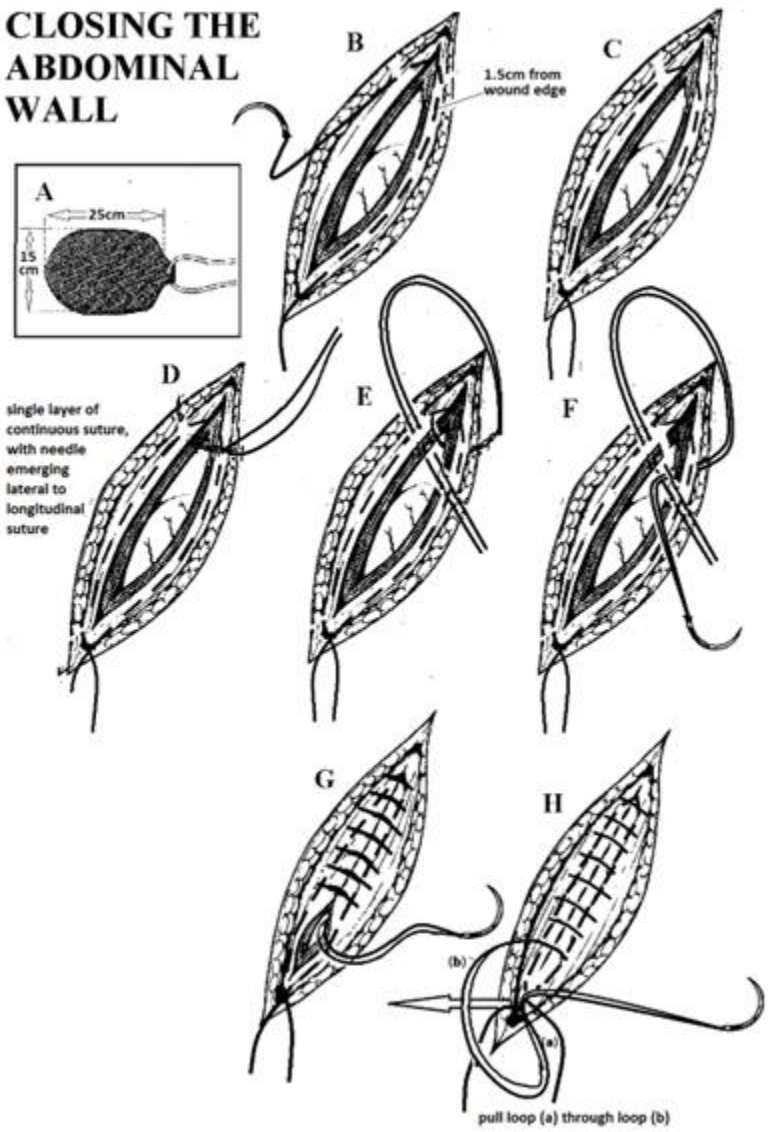


Fig. 11-18 CLOSING THE ABDOMEN. EVERETT'S METHOD: A, a rubber 'fish' to press down on bowel to prevent it getting in the way. B,C, inserting the longitudinal subcuticular suture 1·5cm from the wound edge *(you may prefer to put this suture in last)*. D, inserting the deep continuous suture lateral to the longitudinal one. E,F, making a buried knot, or alternatively pulling the thread through a loop. H, catch the loop in subsequent bites as a locking suture, taking sufficient width of sheath. Prepare for the Aberdeen knot closure (4.13) **and tie this. With the anterior and posterior rectus sheaths closed, you may leave the skin open for secondary closure if there is much sepsis.** *Partly after Agharwal A. et al. Reinforced Tension Line Suture Closure After Midline Laparotomy in Emergency Surgery. Tropical Doctor 2011;41(4):193-6.*

Normally though you have 2 layers to be closed: the peritoneum, which is fused to the posterior rectus sheath and the anterior rectus sheath. Close these layers together in a mass closure by the modified Everett method, which leaves knots within the muscle layer where they cause no discomfort. An ideal suture is a long-lasting absorbable monofilament such as PDS (4.6).

Consider leaving the skin open if there is severe sepsis. This will reduce the risk of wound infection in high-risk cases in the same way as in wounds of other kinds. Antibiotics are less effective than leaving the skin wound open for a few days (see below). Otherwise, close it as a separate layer.

A subcuticular skin suture leaves a neater scar, and probably is less prone to infection because the needle does not go through the skin surface; however, it is liable to dehiscence if there is anything to cause abdominal distension or bleeding post-operatively. So *don't use it if there is a lot of ascites, thrombocytopenia, or severe infection*. *Don't insert subcutaneous fat sutures:* they serve only as foreign bodies and are unnecessary.

(a) Lavage
Before you close the abdomen, make quite sure that, if it is contaminated, you wash it out *completely* with *warm* water (10.1).

(b) Drains are not useful, except for localized abscesses, or leaks of bile, pancreatic juice or urine (4.9), or draining extraperitoneal spaces.

(c) Final checks (1.8)
Check the operation site thoroughly before you close the abdomen to make sure that you have restored the anatomy as you wish, that there is no bleeding, and no leakage from hollow viscera. Close any mesenteric defects. *Make sure that you have left no instruments, swabs, or packs behind*. It is reckless to rely only on a swab count! *Never use small gauze pieces or sponges inside the abdomen:* they too easily get lost!

(d) Preventing adhesions
Bring the greater omentum down so that it underlies the incision. This will help to prevent adhesions forming between the viscera and the abdominal wall.

(e) Closure by the MODIFIED EVERETT MASS CLOSURE METHOD should be your standard way of closing the abdomen. It is best to use a looped suture; if you don't have a ready-made one, take a piece of #1 monofilament 6 times the length of the incision, thread onto it a 65mm ½ needle, fold it in half, and tie a 'figure-of-8' knot at its end, thus creating the looped suture. Alternatively, use a single strand, *but make sure the knot is secure when you tie it*.

Make sure the abdominal wall is relaxed: if the patient is pushing, ask the anaesthetist to increase his relaxation, or if you are operating under ketamine, add diazepam IV. Press down on the bowel with a 'fish' (11-18A) to make sure you don't catch a loop of bowel in your suture. This is a piece of stiff rubber sheet (such as that from a car tyre inner tube) with a tail on it. Alternatively use a specially constructed curved bowel depressor or the handle end of a Morris retractor. *Don't use your assistant's hands as retractors while you are putting in deep sutures!* Put your hand inside after every suture to check nothing is caught. *Don't put sutures in 'blind'.*

Insert a longitudinal suture using a 65mm ½ circle needle parallel to the *linea alba* (11-18B), starting at the lower end of the incision, as a continuous suture on both sides (11-18C), after first clearing the fat c.2cm from it. Hold the ends of this suture by haemostats inferiorly.

Then place the continuous suture using again a 65mm ½ needle, taking care the points are introduced lateral to the longitudinal suture (11-18D) by passing the needle from between the anterior and posterior layers of the rectus sheath out anteriorly, then going from the outer surface of the anterior rectus abdominal muscle inwards (11-18E) on the opposite side of the wound. Thread the needle through the loop if there is one (11-18F).

Otherwise, tie the knot carefully and securely with multiple throws so as to bury the knot between the layers. Proceed all the way along the wound like this taking deep bites and not pulling too tightly (11-18G). Place the sutures about 1cm apart. At the end of the wound come out anteriorly, pull one loop through another (11-18H) and tie an Aberdeen knot (4-13), burying the end of the suture between the layers.

Then pull the longitudinal suture taut and tie it, taking care not to pull it excessively as the function of this suture is to distribute the pressure on the tissue. Too much tightening will lead to bow stringing. *Take care that no bowel loops are caught within either suture*.

The longitudinal suture is an added safeguard in emergency surgery: however, for a clean elective procedure, you may omit it.

If you have made a transverse incision, close the peritoneum with the posterior rectus sheath, and then the anterior rectus sheath in 2 separate layers.

If you have made a Pfannenstiel incision (11-4), close the sheaths transversely.

(f) Skin closure. Now close the skin either with subcuticular absorbable or with continuous or interrupted monofilament; subcuticular closure leaves a neater scar and probably is less prone to infection because the needle does not pass through the outer skin surface. Use skin clips if you have them. Alternatively, if the wound is very septic, leave it open for a few days for delayed primary skin suture later.

CAUTION! If you use interrupted sutures:
(1) *don't take the bites too close to the wound edges,*
(2) *don't make the sutures too far apart,* and
(3) *don't make them too tight*.

DELAYED PRIMARY SUTURE FOR POTENTIALLY INFECTED ABDOMINAL WOUNDS

INDICATIONS. Severe sepsis which contaminates the abdominal wound puts the patient at risk, especially from:
(1) Caesarean section in the presence of infected amniotic liquor.
(2) Appendicitis.
(3) Perforation of the ileum.
(4) Perforations of the colon.
(5) Excision of gangrenous bowel.
(6) Generalized peritonitis.

METHOD. Use this where there really is a lot of sepsis with litres of purulent fluid in the belly, *not just for the localized case*. Close the muscles of the abdomen as above. Make the sutures just tight enough to bring the muscles of the abdominal wall together and prevent the bowel escaping. Test this as you go along by feeling the inside of the wound with your finger, as if it were a loop of bowel trying to escape. Then put a betadine dressing on the wound. Use antibiotics only if the condition you are operating for demands their use.

At 3-5 days, examine the wound. If it is clean, close it by delayed primary closure. If it is infected, apply hypochlorite, saline or betadine dressings regularly until it is fit for secondary suture, or secondary skin grafting. Occasionally, you will find the wound already healing so well that it will close spontaneously. If so, let it do so.

CAUTION!
(1) *NEVER close the fascia or muscle of the abdominal wall with catgut or short-acting absorbable*. It will be absorbed too soon, and increase the risk of early bursting and later herniation.
(2) *Don't use braided silk*, which increases the risk of sinuses.
(3) Make the sutures just tight enough to bring the edges of the muscles together; *don't strangle them*.
(4) *Avoid closing the peritoneum with non-absorbable sutures*, as there is a higher risk of adhesions.
(5) Take care to pick up the posterior rectus sheath and peritoneum in the lower abdomen, as it might retract laterally and so be difficult to see; otherwise an incisional hernia will result.
(6) *Don't try to close the abdominal wall and skin in a single layer*.
(7) *Don't close the abdomen if bringing the abdominal edges together is a struggle:* the abdominal compartment syndrome will result (11.10). Instead, place a vacuum dressing over the open abdomen (11-20).

DIFFICULTIES CLOSING THE ABDOMEN
(1) Get the patient fully anaesthetized.
(2) Make sure you decompress the bowel (12-4) and make sure a nasogastric tube is in place.
(3) Consider whether you should leave the abdomen open (11.10) rather than closing it.

11.9 After an abdominal operation

POST-OPERATIVE CARE

If you have struggled hard to save a patient in the theatre, it is tragic for him to lose his life in the ward afterwards. If you are working under difficult conditions, post-operative care can be at least as difficult as surgery. You will find an ICU very useful for any ill patient, and particularly for someone who is recovering from a major operation. The staff of even the simplest ICU should be able to check the vital signs, keep an accurate fluid balance, and watch for post-operative bleeding: careful monitoring is not difficult; it just needs care and frequent visits from you. If your nurses are not yet appropriately trained, you will gain much by taking time to explain and teach how to do this, initially doing much yourself. If you don't have an ICU, gather critically ill patients near the nurses' station in an ordinary ward, so that the senior nurse can watch them. The list below of the things she should check is a long one, but most of the checks are quick. Make sure the nurse has an appropriate chart to fill in observations. Above all, try to anticipate complications before they occur.

(a) The recovery position. Nurse the patient on the side in the recovery (lateral) position. Turn him 2hrly.

(b) Monitoring.

If a patient is critically ill, make sure that, during the first few hours, some competent person checks every 15mins:
(1) The level of consciousness.
(2) The pattern of the respiration.
(3) The peripheral circulation; the warmth of the extremities.
(4) The capillary circulation in the nail beds.
(5) The pulse and blood pressure.
(6) The temperature.
(7) The urine output.
(8) The degree of pain (in children, look at their face) and any improvement or worsening of pain.
(9) Any bleeding and discharge from the wound.
(10) Abdominal distension.

The nurses in the ICU must be on the lookout for *any deterioration* and call for urgent help if they notice:
(1) snoring, respiratory obstruction, depression or arrest,
(2) bronchospasm,
(3) aspiration of gastric contents,
(4) rising pulse rate and falling blood pressure,
(5) failure of the nasogastric suction to work properly.

N.B. Nausea is usually due to hypotension.

Later, during recovery, attention can change to:
(1) fluid balance,
(2) coughing and breathing exercises.

(c) IV fluids. Maintenance needs are c.3-4L/day for an adult; on top of this you can assume there is a fluid deficit from operative loss, intestinal ileus, and exposure. So use 2-3L Ringer's lactate or 0·9% saline alternating with 1L 5% dextrose per 24h.

If there is any doubt about the adequacy of fluid replacement, be sure to monitor the urine output. Only a very ill patient and some women need an indwelling catheter; remove it when it is not absolutely necessary. A Paul's tube (condom catheter) is often adequate in men.

If you did not adequately replace the blood lost at operation, the blood will be diluted by the first day, so measure the Hb or the haematocrit, and transfuse as appropriate.

(d) Nasogastric suction will prevent the aspiration of vomit; it will remove gas and fluid and relieve distension (4.9), but will not reduce the duration of ileus.

(e) Bowels. If the patient is taking a high-fibre diet, he will probably have no difficulty with the bowels once any initial ileus has subsided. He is more likely to have difficulty if he is on a low-fibre diet and is not mobilizing. Start oral intake in small, gradually increasing amounts, as soon as post-operative nausea and abdominal distension settles.

If he has passed flatus, but no stool by the 5th day, administer a rectal suppository or enema.

If he does not pass flatus and the abdomen remains distended, suspect abdominal sepsis or a leaking anastomosis.

(f) Pain. Remember, though a wound heals side-to-side, it hurts end-to-end! If there is severe pain, use ½ the standard dose of pethidine or morphine IV initially, and then the other ½ 10mins later if the first was not enough. This is *much more effective and safer* than IM use. (IM drugs are not absorbed rapidly enough.) A useful method is to add further doses to the IV fluids 4hrly. Or, better, run it in continuously with the IV fluids. This makes sure that analgesia is continuous without a need to call the nurses. Morphine 5mg qds is an average dose for a fit adult. Use ½ or ¼ of this if the patient is very sick, thin or malnourished. By 3-5 days he should have no need of injectable opioids, so taper them off, and occasionally, if necessary, replace them by an oral opioid.

N.B. ***If the patient is restless****, it is more likely to be due to hypoxia than pain!* Treat this with oxygen. *Don't use opioids if the respiration is shallow, or the systolic blood pressure <100mm Hg.*

Tilidine oral drops (x1 per year of age up to 10) 4-6hrly are very useful for children.

(g) Other drugs.

(1) *Don't use a hypnotic for 5-7 days*; it will not help while there is pain.
(2) *Don't use an antiemetic without looking for a cause*. It may help if there is an inoperable carcinoma.
(3) *Don't use an antibiotic*, unless there is an established infection.

(h) Ambulation. Encourage movement of the legs in bed. Do all you can to mobilize your patient early. Dependant immobile legs have a higher incidence of deep vein thrombosis than raised ones. This is more likely to occur lying in bed or sitting still in a chair than sitting still in bed.

11.10 Non-respiratory post-operative problems

Many complications can interrupt recovery, but you can prevent most of them. Important problems involve the lungs (11.11). Infections are more likely if there is cachexia, HIV disease (5.6), or diabetes.

In order to recover uneventfully from an abdominal operation, the bowel must start to work soon. The passage of flatus and bowel sounds show that the small bowel is starting to work; the large bowel starts 1-2 days later. If all goes well, eating should start in 2-3 days. This will be delayed after peritonitis, an anastomosis of the stomach or upper small bowel, or because of ileus (12.16), or anorexia from any cause, such as burns or severe sepsis. Unfortunately, the common IV fluids provide little energy and no protein, and you are unlikely to have the necessary solutions of proteins and amino acids for parenteral nutrition. However, if you can provide some nutrition, as described below, it may be crucial. Most patients with sepsis are in a catabolic state and so need *greatly increased* levels of nutrition; if you cannot feed them, they will lose much weight very quickly.

(a) Post-operative bleeding and shock
Bleeding may be obvious but minimal, or hidden but catastrophic. *Don't make the mistake of failing to check for signs of circulatory failure* (3.5, 11.9).

(b) Post-operative vomiting
If there is vomiting immediately after the operation, turn the patient on his side. It may be due to the anaesthetic, especially ether, or to morphine or pethidine. He is likely to recover quickly. If he vomits >8h or copiously at any time, start nasogastric aspiration.

If there is vomiting after 48h, this is likely to be more serious, cause severe fluid loss and may result in tracheal aspiration. It may be due to ileus, post-operative bowel obstruction or, rarely, acute gastric dilatation. If you don't replace the fluids and electrolytes IV, severe hypovolaemia and hypokalaemia will ensue. Start nasogastric drainage to avoid aspiration of vomit.

If there is vomiting with a distended, silent abdomen, this is an ileus (12.16), which may be due to post-operative peritonitis (10.1) which also causes pain, fever, and toxaemia. The nature of the previous operation, such as a pelvic abscess or an injury to the large bowel, usually suggests its site. Later, watch for a localized abdominal collection, especially subphrenic (10.2). If the patient's condition deteriorates, think of the abdominal compartment syndrome.

(c) Post-operative urine output failure
If there is no urine output, or only a little, and the bladder is not distended, look for:
(1) Dehydration.
(2) Hypovolaemia.
(3) Renal tubular necrosis resulting in renal failure, caused by a period of low blood pressure during the operation.

N.B. Some urinary suppression is normal for 24-60h after major surgery, as a normal response to stress.

If there is a little urine of high specific gravity, with obvious dehydration, infuse 1-2L of saline as rapidly as you can. If the urinary output does not improve, check the central venous pressure; only if this shows adequate hydration, use a diuretic such as 0·5-1L of mannitol IV, or 40-80mg of furosemide IV. If this produces a diuresis, there was severe dehydration causing renal shutdown, but which is now recovering. If the kidneys produce no flow, there may be tubular necrosis and renal failure.

CAUTION! Don't overhydrate a child.
(1) Treat him with 30ml/kg of fluid for the first 2h, and repeat it over the next 3-4h if necessary.
(2) Before you diagnose anuria, make sure that the Foley catheter is not blocked and is properly situated in the bladder!

If there is no urine passed, and the bladder is distended (dull to percussion), this is urinary retention. It is common after perineal operations especially in elderly men. Try to initiate micturition by getting the patient to stand by the edge of the bed, and walk about if possible. Open a water tap: the sound of running water may make the urine flow; alternatively, put the patient in a warm bath: the warm water may help him relax. Stimulating the skin of the upper inner thigh may also help. If all this fails, introduce a urinary catheter.

(d) Post-operative fever
Most patients have a mild fever (<37·5˚C) for 1-4 days after a major abdominal operation. This is insignificant.

If there is significant fever post-operatively, consider pulmonary collapse (11.11), pneumonia, wound sepsis (2.10, 11.13), urinary tract infection (especially if you have used a catheter), drug reaction, malaria, further intra-abdominal sepsis either under the diaphragm (10.2) or somewhere else, septicaemia or septic shock, or deep vein thrombosis.

If there is persistent fever, and the general condition is not improving, suspect that there is sepsis somewhere in the abdomen, especially if you operated for peritonitis, there was inadequate lavage of contaminants (pus, faeces or bile) during the operation, or an anastomotic leak. In this case you should re-open the abdomen before 48-72h (12.16). Examine the patient carefully every day. Sepsis after a clean abdominal operation suggests a serious problem, which you should deal with earlier rather than later.

If there is, after 3-5 days, also a raised diaphragm and fluid in the costophrenic angle on a chest radiograph, there is a subphrenic abscess until proved otherwise (10.2).

Get an ultrasound scan (38.2l). If there is also diarrhoea with the passage of mucus, a pelvic abscess (10.3) is probably present. The passage of mucus is a particularly valuable sign.

Avoid 'blind' antibiotic treatment unless the condition is critical. It may merely mask the problem, which will become worse later. Attempt to drain an abscess under ultrasound guidance. Try to get to the abscess extra-peritoneally if at all possible. *Don't contaminate a clean area of the abdomen with pus from an abscess which was walled off before!*

If there is no evidence of peritonitis, no abscess on ultrasound, and you have no good idea of where the sepsis is coming from, *don't launch into a difficult repeat laparotomy.*

If there is obvious peritonitis, sepsis from a source unknown, or details of previous surgery are unsure, perform a re-laparotomy.

(e) Post-operative feeding difficulties

If the return to normal eating is much delayed, considerable weight loss will ensue. Encourage eating of the staple diet, such as rice, maize, or potatoes, and supplement this with nasogastric feeding (4.9), using the high-energy milk feed that you usually provide for malnourished children. Convenient mixes for 1L of feed are:

Dried skim milk	443ml	'Nespray'	86g	Evaporated milk	118g
Sugar	67g	Sugar	67g	Sugar	65g
Oil	52ml	Oil	86ml	Oil	54ml
Water	402ml	Water	811ml	Water	813ml

This provides 1370kcal/L. Using this alone needs at least 2L and preferably 3L od. You can add rice, maize or corn as well if you have a liquidizer. Watch the fluid balance, and add 10mmol K^+ od. Look for hidden sepsis which can destroy the appetite!

If fluid intake by mouth is impossible, pass a small nasogastric tube and start feeding with 200ml of a ¼-strength feed every 3h. Increase this to the limit of nausea and diarrhoea, up to 2-2½L of full-strength feed in 24h.

If there is gross cachexia, and you intend to perform an operation on the stomach or duodenum, fashion a feeding jejunostomy (11.7).

(f) Abdominal compartment syndrome (ACS)

If the abdominal closure is tense because of oedematous or distended bowel *(especially in sepsis or after omphalocoele closure)* or a too tight wound approximation (*e.g.* after an incisional hernia repair), rapidly increasing ascites or haemorrhage, there is congestion of the kidneys, inferior vena cava, diaphragm and mesenteric vessels. This results in further abdominal distension, fall in urine output, pressure on the diaphragm, hypoxia and shock.

ACS is a common life-threatening condition which is often not recognized, and often complicates abdominal sepsis. It can also happen in burns, especially of the abdominal wall, and after laparoscopic insufflation of the abdomen with gas.

You can diagnose ACS by emptying the bladder and then instilling 50ml (or 1ml/kg for children <20kg) of saline or warm water into it through a clamped catheter, and hold the fluid bag up vertically. Let the meniscus settle and wait at least 1min to allow for detrusor muscle relaxation; measure its height above the pubic symphysis with the patient lying supine at end-expiration. It does not give a reliable reading if there is a neurogenic or contracted bladder. A more reliable method, useful for continuous monitoring, is illustrated (11-19).

INTRA-ABDOMINAL PRESSURE MANOMETER

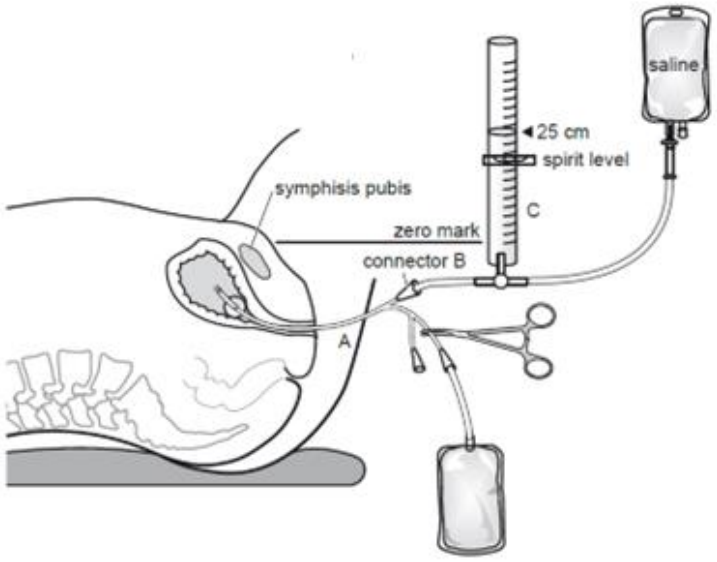


Fig. 11-19 MEASUREMENT OF INTRA-ABDOMINAL PRESSURE.
A, bladder catheter. B, connecting tubing. C, manometer. Make sure the bladder is empty. Prime the circuit with fluid *(the flush solution doesn't need heparin).* Connect the tubing to the urinary drainage tube via a 16G cannula. Clamp the distal outflow, and instil 50ml into the bladder, and turn the 3-way tap so fluid flows into the manometer. Ensure this tube is vertical and open at its top and the zero level is at the level of the mid-axillary line or symphysis pubis (with the patient lying flat), then measure the pressure (in cm H_2O).
N.B. A 2-way catheter avoids the need to connect the pressure tubing to the urinary outlet connections.

If the pressure is >16cm water (12mm Hg), there is intra-abdominal hypertension. Keep the catheter in to monitor the pressure 4hrly. Improve analgesia, sedate the patient, reverse any head-down position, insert a nasogastric tube, empty the rectum by an enema or manually if necessary, remove ascitic fluid by paracentesis if in gross quantities, and administer diuretics IV if the fluid balance is positive. Try metoclopramide or erythromycin to improve gastric emptying.

If the pressure is >27cm water (20mm Hg), ACS is confirmed.

N.B. These measures may not be accurate in the presence of ascites, pregnancy, abdominal packs, obesity, active abdominal contractions or a neurogenic bladder!

VACUUM ABDOMINAL DRESSING

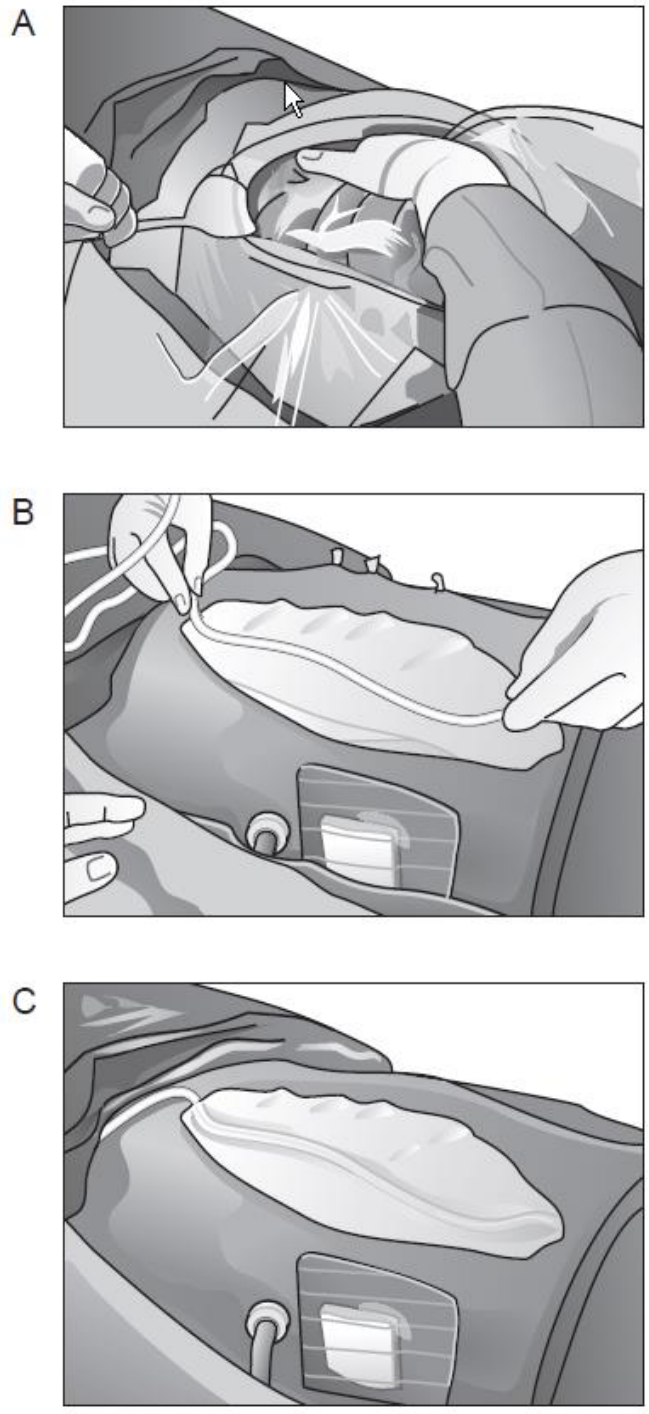


Fig. 11-20 VACUUM ABDOMINAL DRESSING.
A, place a plastic over the bowel, and tuck it 10cm at least under the abdominal wall laterally. B, lay sterile gauze (or sponge) on top of the plastic. C, place 1-2 low-grade suction tubes within the gauze and seal the whole assembly with adhesive. *N.B. The other tube in B,C is an intra-peritoneal drain, separate from the vacuum dressing tube.*

You must, if ACS is confirmed, re-open the abdomen urgently, *and leave it open* (laparostomy). Avoid this scenario by leaving it open in the first place. Do this by placing a sterile plastic (11-20A) over the bowel and tucking this at least 10cm laterally under the abdominal wall edges, adjusting it so that no excess pressure is exerted by the plastic. On top of the plastic, place layers of gauze or sterile sponge (11-20B), and in the midst of these place 1-2 suction tubes (11-20C).

Attach to the tubes low-grade suction (5cm water). Seal the assembly with sticky tape making it airtight. Get an abdominal radiograph to check how many swabs and instruments are left inside.

If possible, keep the patient ventilated mechanically till you perform the 2nd look laparotomy (10.1); however, you don't need to continue ventilation long-term normally. Change the dressings every 48-72h (nitrous oxide is usually all you need for sedation) till you can approximate the wound edges easily and close the abdomen.

Maintain nutrition by nasogastric or jejunostomy feeding and correct the fluid and electrolyte losses by carefully charting them.

N.B. Covering the abdominal defect with a plastic (Bogotà) bag, sutured to the abdominal wall edges, without a suction appliance, is really only useful as a temporary measure because of hypergranulations, sutures cutting out, wound leakage and sepsis. The vacuum suction method is much more effective.

11.11 Respiratory post-operative problems

If the respiratory tract is to function normally, it has to be clear of secretions. Frequently, this clearing mechanism fails, with the result that secretions accumulate, become infected and get into the lung. This may prove fatal. So, you must encourage coughing, to bring up the sputum that might otherwise block the smaller bronchi and cause lung collapse (11.12).

FACILITATING COUGHING IS THE PURPOSE OF MOST CHEST PHYSIOTHERAPY

You can assess Peak Expiratory Flow Rate with a Peak Flow Meter (11-21): this is a simple device which will show how severe the bronchospasm or broncho-constriction is. It is useful if you don't have a pulse oximeter. Measure the patient's best of three efforts at blowing, as hard and quickly as possible, into the meter. He should be sitting or standing to do this, *not lying down.*

Anything which will help mobilization will help the chest. This may not be easy, but any activity is better than lying in bed. Antibiotics are less important, but there may be a need for ampicillin, or chloramphenicol, if the chest infection does not resolve with physiotherapy, or is very severe initially.

PEAK EXPIRATORY FLOW RATE

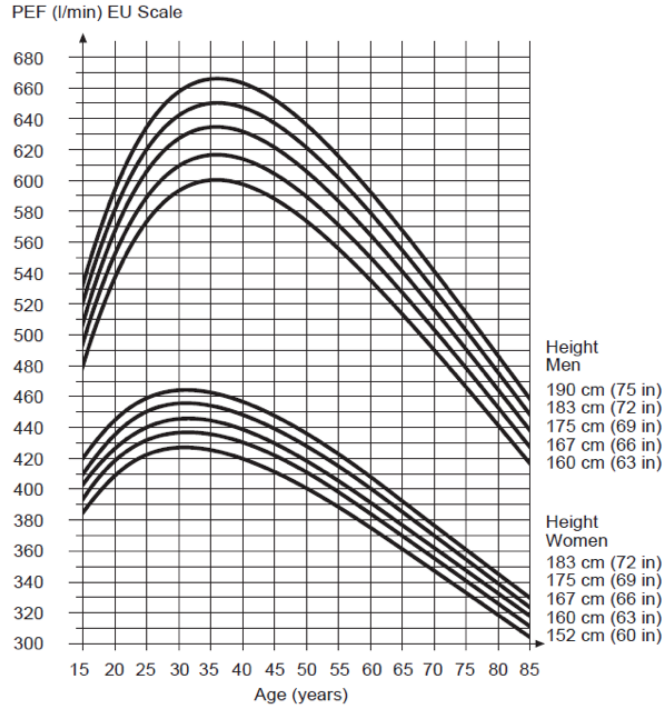


Fig. 11-21 PEAK EXPIRATORY FLOW RATE; NORMAL VALUES.
Measure this pre- and post-operatively, especially if there is asthma, emphysema, or chronic bronchitis. Get the patient to take in a big breath and exhale forcibly into the Peak Flow Meter: use the best of 3 readings.

If coughing remains inadequate and breathing shallow, there are various ways in which you can suck out the sputum. Cricothyroid irrigation, tracheo-bronchial suction, and tracheostomy or 'mini-tracheostomy' (29.15) are heroic measures of last resort but may be surprisingly successful.

(a) Risk factors
Respiratory difficulties are more likely if there is:
(1) HIV disease.
(2) Emphysema or chronic bronchitis.
(3) A painful operation site, particularly in the upper abdomen or chest, which makes coughing painful, and so prevents expectoration of sputum.
(4) Excessive opioid or barbiturate use.
(5) Slow recovery from anaesthesia.
(6) High spinal anaesthesia.
(7) A history of smoking.
(8) Dehydration, which makes sputum thick and more difficult to cough up.
(9) Post-operative immobility as with a fractured femur or paraplegia.
(10) Head injury or polytrauma.
(11) Severe debilitation.
(12) Sickle cell disease when cold or dehydrated, or an Hb <80g/L.

N.B. Babies always have greater risks of respiratory problems.

(b) Complications
If respiration is depressed, and a tracheal tube is still in place, keep the patient in the recovery room until breathing is deep and regular. Anaesthesia may have been very deep, or the patient may be very ill. Attach a self-inflating bag to the tube and inflate the lungs. If this fails, the tube may be blocked with secretions (especially in babies): remove the tube, re-intubate and continue ventilation. *Otherwise, don't remove the tube until spontaneous breathing is adequate*. If the tube has been withdrawn, pull the tongue forward and insert an oropharyngeal airway. If this does not restore normal breathing, attach a mask and a self-inflating bag. If you treat post-operative respiratory depression vigorously, the lungs are less likely to collapse. If you have a ventilator, use it.

If there is cyanosis, wheezing, or an expiratory stridor; **or rapid breathing and tachycardia, or vomit on the lips**, suspect inhalation of gastric contents.
Place the patient in the head-down lateral position. Suction out as much gastric content and secretions as you can. When you have the equipment prepared, intubate the trachea under direct laryngoscopy.
Pass a sterile suction catheter into the trachea and bronchi and aspirate through this. Turn the patient to one side, then the other, and then suck the fluid out again. Repeat this until the breathing is easy and quiet. Or, better, use a bronchoscope, and aspirate through this (29.14). Treat him with oxygen. If respiration is still poor, continue mechanical ventilation.

If there is bronchospasm, treat this with aminophylline 250mg slowly IV.
N.B. Bronchospasm can also be due to the inhalation of vomit (see above).

If there is respiratory failure with cyanosis, treat with oxygen through a face mask with 2 side holes for the air being exhaled. Continue oxygen after tracheal intubation.

(c) Clearing the respiratory tract
The following 3 situations need an antibiotic *and* physiotherapy to clear secretions from the chest.

(1) **If there is a cough, confusion, restlessness, fever, tachycardia, cyanosis, rapid or irregular or grunting breathing, with flaring of the *alae nasi***, there is a serious post-operative lung complication.

(2) **If, in addition**, there is dullness to percussion over the bases of the lungs, usually on the right, with decreased breath sounds and bronchial breathing, low-pitched rhonchi, and radiographs show basal segmental areas of increased density, thick mucus has plugged the smaller bronchi, and caused the lung distal to them to collapse.

(3) **If, in addition to the above signs of lung collapse**, there are mucopurulent sputum, rhonchi, and toxaemia, this is bronchitis, bronchiolitis, or pneumonia.

(d) Cricothyroid irrigation will usually stimulate coughing. Under LA, push a needle and cannula combination ('intracath') on a syringe through the cricothyroid membrane in the midline. Aspirate to make sure that you withdraw air, and then remove the syringe and push the catheter in another 2cm to be sure it is well inside the trachea. Suture it in place, and plug the opening to make sure that air does not go in or out. Instil 2-3ml of saline several times a day to stimulate the cough reflex.

(e) Tracheo-bronchial suction is useful if there is a 'bad chest' and you think that chest complications are likely after surgery. Leave the tracheal tube in for 24-48h, so that you can suck out the chest through it. Vigorous coughing will not be possible, but you will be able to aspirate the chest frequently.
Before you aspirate, turn the patient to one side and instil 5-10ml of saline into the trachea. This will help to liquefy the sputum and will make suction easier. Turn the patient to the other side and repeat it.
Make sure there is a Y-connection on the suction tube. Release your thumb from the side arm intermittently to prevent you aspirating too much air, and making the bronchi collapse.
If you have a flexible bronchoscope with efficient suction, you can pass this through the tracheal tube and aspirate under direct vision.

If you have already removed the tracheal tube that was in place during the operation, and have done everything you can to initiate coughing, consider passing a nasotracheal tube, and sucking out the chest through that.

TRACHEOSTOMY. If other methods of aspiration, including bronchoscopy (29.14), fail, or you need to continue intubation for >5 days, perform a tracheostomy (29.15), and suck out the bronchi through this. If you have bypassed the nose with anything but a mini-tracheostomy tube (see below), humidify the air, if necessary with a steam kettle. It will help facilitate coughing. If you have a steam room, use that for the 1st wk.

'MINI-TRACHEOSTOMY' is the most practical way to suck out the trachea. Use a small (4mm) tube (preferably a disposable 'portex' one). Using LA with adrenaline solution, insert it through the cricothyroid membrane using a guarded scalpel and an introducer. Failing this, use a 4mm paediatric tracheostomy tube and pass a Ch10 suction catheter down it. A tube of this size is not large enough to obstruct the respiratory tract, and there is little bleeding.
You will avoid the complications (particularly stenosis) of the cricothyroid approach using a large tube. It will then be possible to speak, cough, eat, and drink. Use humidified inspired air normally without the need for sedation or anaesthesia. The wound heals quickly with little scarring.

11.12 Respiratory physiotherapy

Some simple physiotherapy will often prevent the complications described in the previous section. If a high-risk patient (11.11) is to have an elective operation, this physiotherapy should start *before* the operation. You may have no physiotherapist, so you may have to learn these skills yourself, and teach them to your nurses and to the relatives.

CHEST PHYSIOTHERAPY CAN BE LIFE-SAVING

PRE-OPERATIVELY, take the patient through the motions of breathing in deeply through the nose and mouth. Either, sit him up at 70º well supported from behind by a back support, and with a bolster to prevent the knees slipping down. Or, lay him on his back with the knees bent. Put your hands on the chest during breathing efforts. Recommend about 6 breaths only at a time, otherwise dizziness may result.

CAUTION! Be sure to explain why these exercises are so necessary.

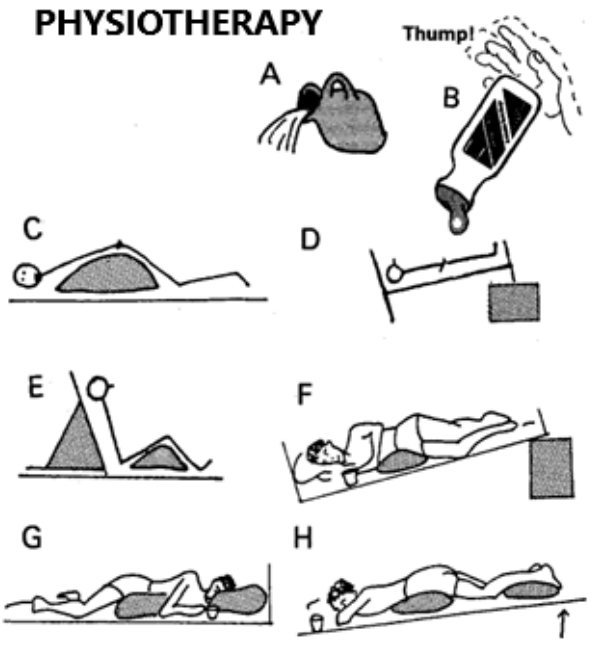


Fig. 11-22 CHEST PHYSIOTHERAPY.
A, if secretions are sufficiently liquid you can pour them out of the chest. B, if they are viscid, you may have to shake them out of the bronchi by percussing the chest in the same way that you can percuss tomato ketchup out of a bottle! C, you can lay a patient with his hips on pillows, so that the hips are higher than the shoulders. D, you can raise the foot of the bed. E, you can sit the patient against a back-rest with pillows under the knees. F, you can raise the foot of the bed and put a pillow under the hips. G, if the patient is too weak to sit up, you can rest him against a pillow and lay him on the side. H, you can use the prone position with a pillow under the hips and the foot of the bed raised. *After Hardinge E, Wilson PMP. A Manual of Basic Physiotherapy, TEAR Fund 1981.*

(a) Post-operative analgesia, when adequate, is a big help. Encourage proper deep breathing, moving about in bed, and getting up as soon as possible. Position is important; *avoid the semi-recumbent 'slumped' position*, because this restricts movement of the diaphragm, and promotes the collapse of the lower lung lobes. Encourage sitting up with a back support. Insist on early mobilization by the 2nd day, if you can, even if there is an IV line, catheter or chest drain to trail around. Encourage the exercises already learnt. An 'incentive spirometer' is very useful.

(b) Coughing. Distinguish between an effective deep productive cough (which is what you want) and a noise in the throat, which is useless. Several short expiratory 'huffs' before coughing will help to loosen the secretions. Suggest taking a deep breath after each cough, and *not to cough continually without pausing*.

If there is an abdominal wound, it is essential to bend the knees, hold the wound, maybe with a pillow, and then to take a deep breath and cough. Give reassurance that the wound will not split open (if your closure is adequate!). If you can, use vibration during coughing.

BRONCHOPULMONARY SEGMENTS

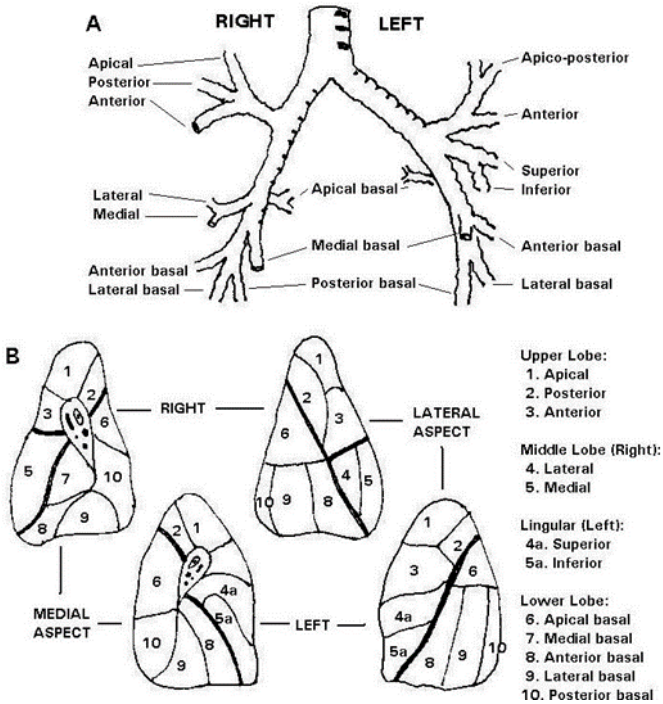


Fig. 11-23 BRONCHOPULMONARY SEGMENTS. A, each branch of the bronchial tree leads to separate segments (B).

(c) Huffing. A 'huff' is a rapid forced expiration without a cough. Huffing, when the lungs are in full inspiration, will dislodge secretions from the larger airways. Huffing, when the lungs are half-full, will dislodge secretions from the smaller airways. So encourage huffing in both phases, with periods of relaxation and abdominal breathing between them. Do this every hour from the day of operation; make checks bd for the 1st 48h.

CAUTION! To be effective, a 'huff' must be long and controlled, using the abdominal wall, and not spasmodic. The noisiest 'huff' is not necessarily the best.

(d) Percussion and vibration. Percuss the thorax over a towel or blanket with your cupped hands for periods of about 1min. Then rapidly shake the chest during expiration. Relax during inspiration, and follow this with some deep breathing. Repeat this 2-3 times.

(e) Postural drainage will be useful if there is much fluid in the bronchi. Listen carefully to the chest, and examine the chest radiograph. Decide where the secretions are worst, and arrange the patient so that this affected part is uppermost, using the appropriate position (11-24). Encourage deep breathing for 10mins, vibrate and slap the chest for 10mins, then repeat the breathing. If there is established collapse or infection, repeat this bd/tds.

If the patient is too ill for the hips to be raised, lay him on his side. If the secretions are viscid, ideally he needs inhalation therapy to 'loosen' them prior to physiotherapy: steam with Friar's balsam or saline with mucolytics from a nebulizer.

POSTURAL DRAINAGE

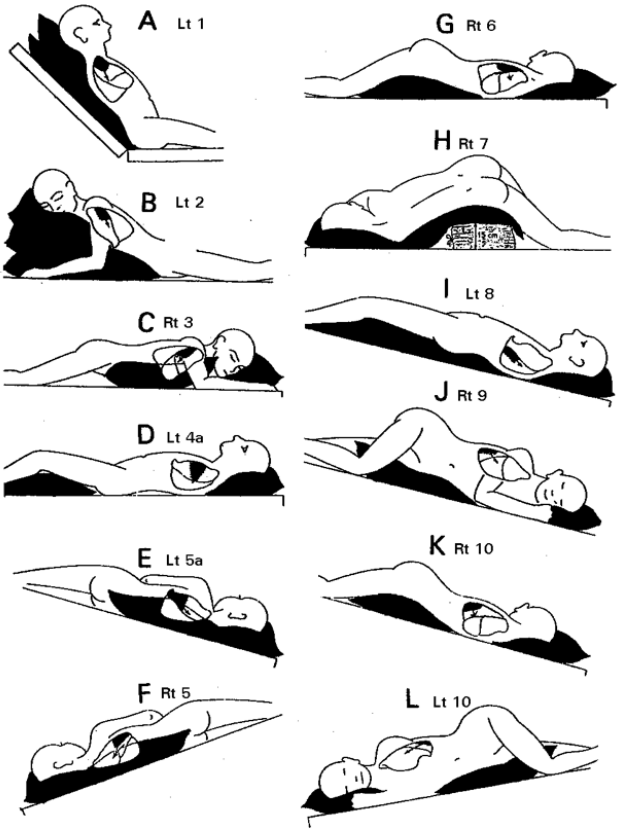


Fig. 11-24 POSTURAL DRAINAGE. Positions which allow gravity to promote the drainage of secretions from particular parts of the lung. Study the chest radiograph, and decide which position will be best. Positions correspond to the following segments: A, left upper apical 1. B, left upper posterior 2. C, right upper anterior 3. D, left superior lingular 4a. E, left inferior lingular 5a. F, right middle medial 5. G, right lower apical 6. H, right lower medial 7. I, left lower anterior 8. J, right lower lateral 9. K, right lower posterior 10. L, left lower posterior 10. *Kindly contributed by Lynne Wilson.*

11.13 Abdominal wound infection

A laparotomy wound usually remains tender for 7-10 days after an operation. If it is abnormally tender and indurated, associated with fever, it is probably infected. Infection may also be elsewhere: in the abdominal cavity, in the chest, or elsewhere. Sometimes the site of infection may be unclear.

Be guided by the severity of the symptoms. More severe anorexia, fever, and malaise should all make you suspect an abdominal abscess, for example. Consider whether a wound infection is a sign of deeper trouble.

If many of your wounds become infected, look at your sterile procedures and leave any but completely clean wounds open (11.8).

(a) Post-operative wound infection

If the wound is red, painful, and tender, and discharges pus, it is infected, so start by removing 1-3 skin sutures on the ward. This will show you the extent of the infection. Take a Gram stain of the pus. If it seems to be deeper, but is still extraperitoneal, use sedation with pethidine, ketamine or diazepam, and press the sides of the wound, and probe suspicious areas with sinus forceps. *Don't open up the deeper layers of all infected wounds from top to bottom, or remove the deeper sutures*. The peritoneum will probably have healed in spite of the infection, but the sutures in the fascial layers will probably pull away. If pus flows adequately, drainage should be adequate. Irrigate the wound with ¼ strength hydrogen peroxide, or hypochlorite solution. It's best to use a 10ml syringe with a 24G needle attached: break off the needle from the plastic hub; the resulting apparatus will produce the ideal spray. Pack it with dry gauze, or gauze soaked in a mild antiseptic, and change this 1-3 times daily. You can speed up the cleansing of a wound by applying a suction dressing; commercial vacuum sponge dressings are available but expensive. You can make your own by simply placing a fenestrated drainage tube, attached to a suction device, between layers of gauze and sponge, and sealing the whole assembly against intact skin with adhesive ('opsite' is best). The seal must be airtight, and you therefore need to make sure the surface is quite dry before trying to stick any adhesive. Use a suction pressure of 50-125mm Hg.

CAUTION!
(1) Make a wide enough opening to release the pus.
(2) Test the glucose and HIV status if the wound infection occurs after clean surgery.
(3) *Don't resort to antibiotics unless there is cellulitis present.*

If the surrounding area is inflamed, there is cellulitis. (6.22). Treat this with IV gentamicin (if you suspect Gram-ve organisms) or cloxacillin (for Gram+ve organisms).

If the wound smells putrid, suspect an anaerobic infection. Treat with oral metronidazole and chloramphenicol. If there is abdominal tenderness nearby, suspect peritonitis and re-open the abdomen; otherwise check for development of a faecal fistula (11.15). Remember if you have inadvertently caught bowel in a suture closing the abdomen, this is how the problem usually first presents.

If you see necrotic muscle or fascia, when you remove skin sutures, debride the dead tissues urgently and lay open the wound widely. Sepsis can spread very quickly and be fatal (6.23).

If the wound is tense, swollen and bruised, with old blood exuding from between the sutures, suspect a haematoma. Under sedation, remove a few of the skin sutures, and wash out old blood and clot with water. Lift out more clots with a swab. Irrigate the wound with hydrogen peroxide, and leave it open. If it is clean after 5 days, consider secondary suturing.

If the wound discharges a little brownish fluid which smells mousey, suspect gas gangrene (6.24). This is commoner than you probably think. Obvious gas in the tissues is uncommon, so that gas gangrene is often missed. Remove all of the skin sutures, make a Gram film of the exudate, and look for Gram+ve bacilli. Treat aggressively with IV benzyl penicillin 10MU qds for 5 days and with metronidazole 1g PR or IV tds. Debride the wound, remove any dead tissue, and make sure you isolate this patient from other patients.

If the wound discharges chronically, there is probably a foreign body *in situ*. This is most likely a non-absorbable suture: infiltrate LA, widen the sinus and use a crochet hook to remove the suture knot. If the discharge persists, there may be a fistula (11.15) or a swab left inside the abdomen! Take a radiograph (most abdominal swabs should have a radio-opaque marker), and re-explore the abdomen carefully. Bowel may be caught in the swab, and need resecting *en bloc* (11-7). If there is neither foreign body nor fistula, check the glucose and HIV status.

11.14 Burst abdomen (Abdominal dehiscence)

An abdominal dehiscence may occur because you should not have closed the abdomen in the first place! A burst abdomen results because either:
(1) your suture breaks,
(2) your knots slip, or
(3) your suture cuts out through unhealthy tissues.

The abdomen is likely to burst if:
(1) You have sutured the fascia with absorbable suture, especially if this is of low quality or out of date, or quickly absorbed, as with sepsis.
(2) Your knotting technique is faulty.

BURST ABDOMEN

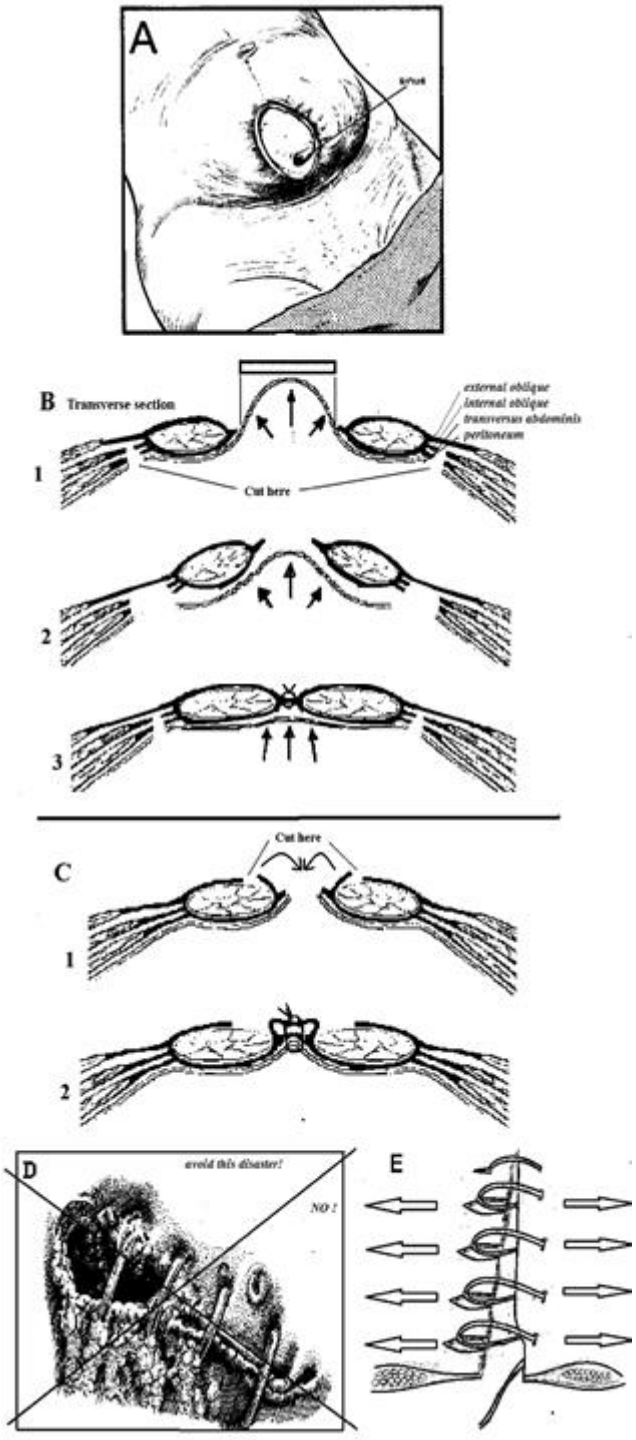


Fig. 11-25 CLOSURE OF A BURST ABDOMEN.
A, measure the defect after debridement, and approximating the wound edges without excessive tension. B & C, incise the *transversus abdominis* and *internal oblique* with diathermy along a line between the mid-axillary and anterior axillary lines. D, recurrent burst abdomen and an intestinal fistula. E, *the tension sutures are not helpful: the wound has broken down.* (*Detail from 11-26; after Esmat ME, New technique in Closure of Burst Abdomen. World J Surg 2006;30(6):1065.)* E, tension sutures cutting out as the intra-abdominal pressure rises.

(3) There is significant abdominal distension due to ileus, intestinal obstruction, or a large tumour.
(4) There is severe intra-abdominal sepsis, such as an infected Caesarean section, typhoid peritonitis, or a perforation of the large bowel.
(5) There is poor healing due to HIV disease, malnutrition, carcinomatosis, uraemia, or obstructive jaundice.
(6) There is a chronic pressure on the wound from prolonged coughing, asthma or chronic obstructive airways disease.

An abdomen which bursts some days after you have sewn it up is a tragedy, because it is often preventable, and because there is a 30% chance of death if subsequent treatment is delayed. Dehiscence is much rarer if:
(1) You suture the abdomen with non-absorbable, such as steel, nylon, or polyethylene.
(2) You close its muscles with interrupted through-and-through sutures, which are not too tight and take wide bites of tissue (11.8).
(3) You use a transverse rather than a midline incision.

DIAGNOSIS. If the wound is painful about a week after the operation, and there is a thin reddish-brown discharge, the abdomen is probably going to burst.

If you can see loops of bowel through the wound, it has already burst! *Initiate treatment before it bursts!* Decompress the bowel by inserting a nasogastric tube, administering a rectal enema. Reduce oedema, if present, with diuretics. Measure the intra-abdominal pressure (11-19). Place a vacuum dressing over the wound.

CLOSURE OF A BURST ABDOMEN
(GRADE 3.3)

If there is a complete dehiscence (with bowel spilling into the bed), examine the patient in theatre under GA unless he is very unfit. In this case, close the skin only under LA if it is not infected, knowing that an incisional hernia will remain. Otherwise, prepare for a laparotomy.

Remove the skin sutures in the area where you suspect the burst. Remove the dressings and gently explore the depths of the wound. Open it down its whole length by removing all the skin sutures. You will soon find out what has happened. If you confirm a burst abdomen, remove all sutures from the fascial layers. Gently separate the parietal peritoneum and the underlying bowel and omentum. *Take care you don't damage bowel.* Check if there is any evidence of intra-abdominal sepsis, and wash out the abdomen, and deal with its cause.

If there is any tension when you try to pull the abdominal wound edges together, measure the width. *Don't try to force the abdomen closed under tension*: an abdominal compartment syndrome will result (11.10).

There are 3 options but unless you have diathermy the first 2 will be too bloody to be safe:

Option 1: Make a long relieving incision *inside* the abdomen longitudinally (11-25B) between the anterior axillary and mid-axillary lines from the costal margin to the iliac crest, preferably with diathermy.

If you go through the *transversus abdominis* and *internal oblique muscles* only, without cutting the *external oblique*, no lateral hernia will form. You will gain 2cm for each side. If you need more, dividing the *external oblique* in addition at a position not directly above the previous incision will give you another 1cm each side; dividing the subcutaneous fascia in addition will give a further 1cm each side. You will therefore be able to close a defect 8cm wide without tension; for a bigger defect, use a vacuum dressing (11.10).

Option 2: Divide the anterior rectus sheath longitudinally in the midline *and* slightly laterally separating it from the muscle bulk (11-25C); then advance both sheaths towards the midline and close them with one continuous #1 non-absorbable suture.

Option 3: Place a vacuum dressing over the open abdomen, leaving it as a laparostomy: this requires you to decompress the bowel, return it to the abdominal cavity, and cover it with omentum onto which you place the dressing and suction drains (11.10).

If there is no tension, and no sepsis present, re-suture the abdominal wall with #1 interrupted steel or monofilament sutures. Suture from within outwards through the peritoneum, posterior rectus sheath, rectus muscle, and anterior rectus sheath, *but not through the skin*. Hold all the sutures out on haemostats until you have placed the last one.

11.15 Intestinal fistula

An intestinal fistula is an abnormal track, usually lined by granulation tissue, between the bowel and the skin. Fistulae are unusual but serious complications of abdominal surgery, and in HIV, tuberculosis, actinomycosis or Crohn's disease arise spontaneously. They may result from a neglected strangulated hernia. Beware of post-operative fistulae:

(1) After you have divided adhesions for intestinal obstruction, especially if you have opened the bowel by mistake and closed it inadequately, or if it is obstructed distally.
(2) After an anastomosis done inaccurately, or in the presence of tension, a poor blood supply, or local disease.
(3) If bowel is caught in the sutures, when you close the abdomen.
(4) After appendicectomy especially when the base of the appendix was inflamed (caecal fistula).
(5) If you use diathermy close to the bowel.

The mortality rate of a high-output fistula (>1000ml/24h) is 70%, and a low-output one (<200ml/24h) 30%. The repair of a fistula is one of the most difficult operations in surgery. *Don't be tempted to operate!*

If intestinal content discharges from the main wound, or the site of a drain post-operatively, there must be a bowel perforation. If the patient says that gas comes out, this confirms it; if you are in any doubt, get the patient to swallow some diluted methylene blue dye, and watch for it to appear in the wound or in the dressings. The speed at which it comes out may give you some idea of whether the perforation is high in the GI tract.

If there is a localized discharge of bowel content, insert a fine soft catheter into the track and inject 10-20ml of water-soluble contrast medium (take some plain abdominal films: a fistulogram) and you may be able to delineate from where the fistula arises, though static films are difficult sometimes to interpret.
Perform an ultrasound scan (38.2l) to detect any fluid collections in the abdomen.

AN INTESTINAL FISTULA

Fig. 11-26 AN INTESTINAL FISTULA. This patient was operated on for obstruction of the small bowel by *ascaris* worms, and a length of it was resected. The anastomosis broke down; a fistula developed. He died a few hours later.
Lesson (1): don't anastomose bowel in the presence of severe sepsis or* ascaris*. (2) don't close the abdomen under tension.
N.B. This child would be better nursed as in 11-11.

SUDHA (25yrs), a young housewife, had an operation in a district hospital for 'appendicitis' through a McBurney incision. Five days after the operation the wound discharged large quantities of pus, and then liquid faeces and gas. She was fed on a low-residue diet, and the skin round the fistulous opening was painted and protected with zinc oxide paste. Absorbent dressings were changed tds and her distal colonic obstruction due to constipated faeces was treated with glycerine suppositories and a plain water enema. The fistula healed in 2wks and she went home.
LESSON Some fistulae will close on non-operative treatment.

TREATMENT is supportive.
Replace fluid and electrolytes, orally, intravenously, or by jejunostomy (11.7). Large quantities of electrolytes as well as calcium, magnesium and phosphates may be necessary.

Maintain nutrition, orally or by jejunostomy. You are unlikely to have IV protein and energy-rich fluids to treat the patient, but you can make up low-residue high-protein, high-calorie feeds to use enterally (50.10). The patient needs c.3-4 times the normal energy intake.

Restrict oral intake initially only whilst you are cleaning up the wound, and then slowly increase fluid intake unless the fistula is so high that fluid pours out directly.

If there is a proximal high-output fistula and you can see or locate the bowel ends using a soft catheter and contrast medium, you can try to initiate feeding through the distal (efferent) loop using a small Foley catheter with the balloon inflated to 5ml only, and at the same time draining proximal intestinal fluid through another Foley catheter in the proximal (afferent) loop.
Unless there are further fistulae distally, you can allow this fluid to pass back into the distal part of the small bowel, thus bypassing the fistula. Antacids or anti-H2 blockers will often be helpful.

Care for the skin, by applying karaya gum or zinc oxide carefully around the fistula so that the liquid intestinal juice, which is full of digestive enzymes, is kept from contact with the skin. At the same time, ensure free drainage either by nursing the patient prone (11-11F), or applying a vacuum dressing with continuous suction (11.13). Milk or magnesium trisilicate applied to the skin will soothe the burning effects of small bowel effluent. Pure honey will help heal excoriated skin.

Control infection with antibiotics and drainage when necessary.

Correct anaemia with ferrous sulphate, vitamin B and folate. Transfuse blood if the Hb is <70g/L.

Keep the distal colon empty, with enemas and glycerine suppositories on alternate days.

An intestinal fistula will close provided there is:
(1) adequate nutritional support.
(2) no foreign body involved.
(3) no untreated inflammatory or malignant bowel disease.
(4) no epithelialization of the track.
(5) no distal obstruction.
Don't be tempted to re-open the abdomen, unless there is frank peritonitis: it will prove to be a disaster worse than the first!

If there is frank peritonitis, re-open the abdomen, exteriorize the bowel, and lavage the peritoneal cavity with copious warm fluid. *Don't try any more surgical heroics*: the chances of your patient surviving are slim. Try to correct his physiology in an intensive care unit.

12 Intestinal obstruction

12.1 The acute abdomen

Any patient needing admission to hospital with acute abdominal pain has 'an acute abdomen', but you have to distinguish who has a serious illness which may be fatal if you don't operate, and who has a self-limiting complaint. You also must distinguish between peritonitis (10.1) and intestinal obstruction. Localized peritonitis may cause obstruction; intestinal obstruction can quickly lead to peritonitis if the bowel blood supply is cut off.

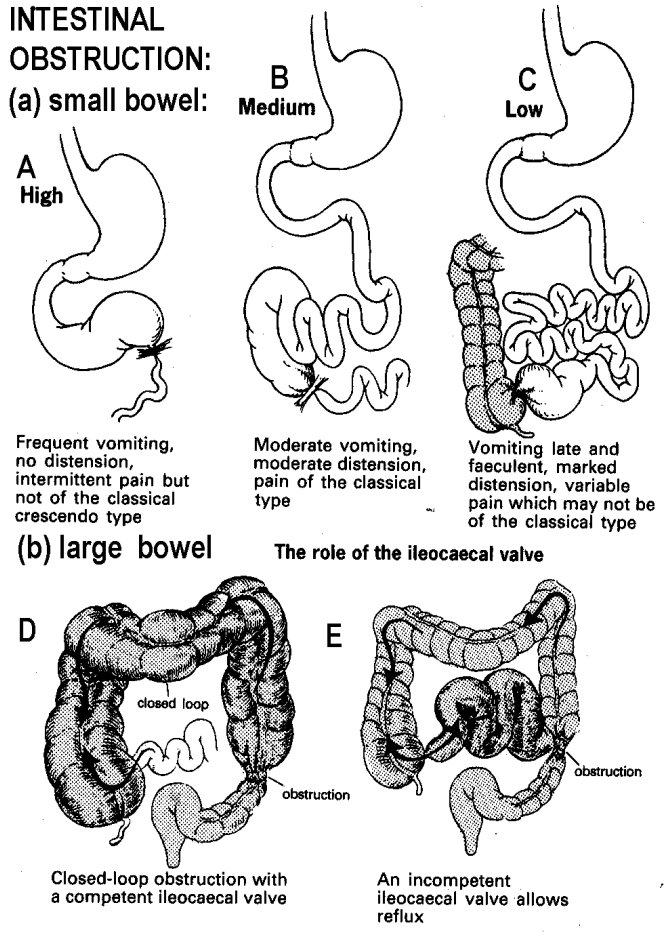


Fig. 12-1 INTESTINAL OBSTRUCTION.
A-C, small bowel obstruction. In A, the obstruction is high, there is frequent vomiting, no distension, and intermittent pain, which is not of the classical type. In B, the obstruction is in the middle of the small bowel. There is moderate vomiting, moderate distension, and intermittent pain of the classical, colicky, crescendo type with free intervals. In C, obstruction is low in the small bowel. Vomiting is late and faeculent, and distension is marked. Pain may or may not be classical. D-E, large bowel obstruction. In D, the ileocaecal valve is competent, and prevents distension spreading to the small bowel, so that there is a closed loop. In E, the valve is incompetent, so there is reflux into the small bowel which distends.
After Dunphy JE, Way LW. Current Surgical Diagnosis and Treatment. Lange 2nd ed 1975 Figs, 33-5, 34-5. with kind permission.

With peritonitis there is an urgency to deal with the source of peritoneal inflammation because of the danger of septicaemia and death; with intestinal obstruction where there is no peritonitis, the urgency is to correct fluid and electrolyte loss first. This allows reasonable time to make a diagnosis.

12.2 Causes of intestinal obstruction

Intestinal obstruction will be one of your major challenges. It is a common abdominal emergency, and in some communities the most common one. Some patients with simple obstruction resolve spontaneously, for example those with ascariasis (often) or tuberculous peritonitis (often) or non-specific adhesions (often if early, less often if late).

When you operate, you may only need to divide adhesions, or massage a ball of *ascaris* from a child's ileum on into the colon. But if you find that a segment of small bowel is gangrenous, you will have to resect it and consider joining the remaining ends. You cannot always safely do this in the presence of sepsis or soiling, especially with large bowel if it is loaded with loose faeces, because such an anastomosis may leak. So you may have to construct a stoma (11.6) or leave the bowel temporarily tied off if the patient is very sick indeed, and join up the bowel at a 2nd look laparotomy (10.1) at 48h when the condition has improved. Unfortunately, a patient with intestinal obstruction often presents late, when he may be severely dehydrated, hypovolaemic, oliguric, and shocked.

The causes of intestinal obstruction vary geographically. Find out the common causes in your area.

(a) Common causes
Incarcerated or irreducible external hernias
(especially inguinal and femoral).
Volvulus of the sigmoid colon.
Ascariasis.
Intussusception.
Adhesions or bands
(following previous surgery, or abdominal sepsis).
Adhesions or fibrosis due to abdominal TB.
Simple constipation (including faecaloma).

(b) Uncommon causes
Volvulus of the small bowel.
Carcinoma of the large bowel.
Carcinomatosis of the peritoneum.
Amoebic granuloma or stenosis.
Intra-abdominal abscess (including retained swab).
Bilharzioma.
Mesenteric thrombosis.
Kaposi sarcoma of the small bowel.
Trichobezoar, phytobezoar (or other foreign body).
Intestinal paracoccidiomycosis.

(c) Rare causes
Primary tumours of the small bowel.
Congenital bands, atresia and malrotation.
Internal abdominal hernias.
Lymphogranuloma.
Abdominal ileal cocoon.
Crohn's disease.
Gallstone ileus.
Diverticulitis.
Radiation enteritis.
Pseudo-obstruction (Ogilvie's syndrome).
Rectal strictures.

WHAT IS THE PATTERN OF INTESTINAL OBSTRUCTION IN YOUR AREA?

12.3 The diagnosis of intestinal obstruction

There are several patterns of intestinal obstruction. They are determined by how the bowel is obstructed, and where it obstructs. Firstly, the obstruction can be simple or strangulated.

(a) Simple obstruction is caused by a mechanical blockage, without impairment of the blood supply of the bowel. It may resolve spontaneously. Operation is usually not urgent, and may be unnecessary. An obstructed bowel dilates above the obstruction, so that it may fill with several litres of fluid and gas. This makes the abdomen swell. Initially, the peristaltic activity of the dilating bowel increases to overcome the obstruction. This causes rushes of hyperperistaltic bowel sounds. Later, as fluid falls from one dilated loop to another, you may hear high-pitched tinkling bowel sounds before the abdomen becomes silent as ileus develops. Inadequate fluid intake combined with the loss of fluid into the lumen of the bowel and by repeated vomiting contribute to fluid depletion, so that dehydration, hypovolaemia, acidosis and shock follow. An adult secretes 7L of gastrointestinal juice in 24h; this fluid is lost in a so-called 'dead space' and so the degree of dehydration is soon serious.

(b) Strangulation obstruction occurs when there is a mechanical blockage and the blood supply to the bowel is impaired. Strangulated hernias and sigmoid volvulus are common causes. About 6h after interruption of its blood supply, the bowel becomes ischaemic and may perforate. If it perforates into the peritoneal cavity, there is spillage of intestinal content, by now heavily infected, resulting in generalized peritonitis which will end in septic shock. If it perforates into a hernial sac, the infection may be more localized. This condition is quickly critical. If you think that peritoneal irritation might be due to strangulation obstruction, operate soon!

Features of obstruction differ according to the levels at which it occurs:
(1) **Small bowel obstruction** is often quite dramatic. The higher the obstruction, the earlier and the worse the vomiting, and the greater the threat to life from electrolyte imbalance, but the less the abdominal distension. Conversely, the lower the obstruction, the greater the distension, the greater the pain, and the later the vomiting.

(2) **Large bowel obstruction** follows a slower course. Because there is more bowel to dilate, there is more abdominal distension, which may be so severe as to interfere with breathing by pushing up the diaphragm. To begin with, only the colon dilates, but the ileocaecal valve usually becomes incompetent (in ⅔ of patients), and allows the dilatation to progress proximally into the small bowel. The signs of dehydration are of slower onset, because the colon can still absorb fluid above the obstruction.

N.B. **Simple constipation** may occur in:
(i) elderly sedentary people, especially if they are taking codeine-based drugs for pain, or many other types of drugs, especially anti-depressants
(ii) shanty-town dwellers eating 'junk food'
(iii) people who like to eat soil (pica), especially if pregnant or with iron-deficiency anaemia, or cassava or guava (especially with the stones) in large quantities, particularly with whole grasshoppers
(iv) Chagas disease (trypanosomiasis)
(v) Hirschsprung's disease. There may be 'spurious diarrhoea' where liquid faeces passes the impacted faeces: obviously giving antidiarrhoeals in this situation makes everything worse!
(vi) Hypothyroidism.

(3) **'Closed-loop obstruction'** *(unusual)* is the result of the ileocaecal valve remaining competent. It is a double obstruction which shuts off a loop (12-1D). It can occur in volvulus especially with an ileosigmoid knot (12-14), and in neglected obstruction of the large bowel. Dilation of the closed loop quickly obstructs its blood supply and rapidly causes gangrene and peritonitis.

(4) **Pseudo-obstruction** (Ogilvie's syndrome), where the large bowel distends alarmingly, may occur after pelvic fractures (especially with retroperitoneal haemorrhage), burns, metabolic disturbances (especially hypokalaemia, uraemia, acidosis and hyperglycaemia), hypoxia, or with opiate or phenothiazine use; there is gas in the rectum unlike in true large bowel obstruction, and bowel sounds are high-pitched.

Common mistakes are:
(i) Not spending enough time, both taking the history and sitting beside the patient watching, palpating, and listening to the abdomen.
(ii) Not recognizing the possibility that obstructed bowel may strangulate, even when the signs of peritoneal irritation are minimal, for example in intussusception.
(iii) Not making proper use of radiographs.
(iv) Operating too early, before adequate rehydration.
(v) Operating too late, after you have allowed the bowel to strangulate.
(vi) Not emptying the stomach with a nasogastric tube.
(vii) Doing a complicated operation when a simpler one would have been life-saving.

(viii) Using poor surgical technique: *open the abdomen with care, dissect dense adhesions gently, make anastomoses carefully, and don't soil the peritoneum with the contents of the obstructed bowel*.
(ix) Not washing out the peritoneal cavity when it is soiled.
(x) Not replacing blood, fluid and electrolytes lost.

(a) History
(1) **Pain** differs in large and small bowel obstruction.

If the pain is periumbilical and colicky, comes in spasms, builds up to a crescendo, and then tapers off, the small bowel is obstructed. Vomiting may relieve it temporarily. Sometimes there are regular pain-free periods at intervals of 2-5mins. This is the classical pain of small bowel obstruction. If peristalsis stops, colic stops, so its disappearance may be a bad sign.

If the pain is below the umbilicus and comes at intervals of 6-10mins, the large bowel is likely to be obstructed.

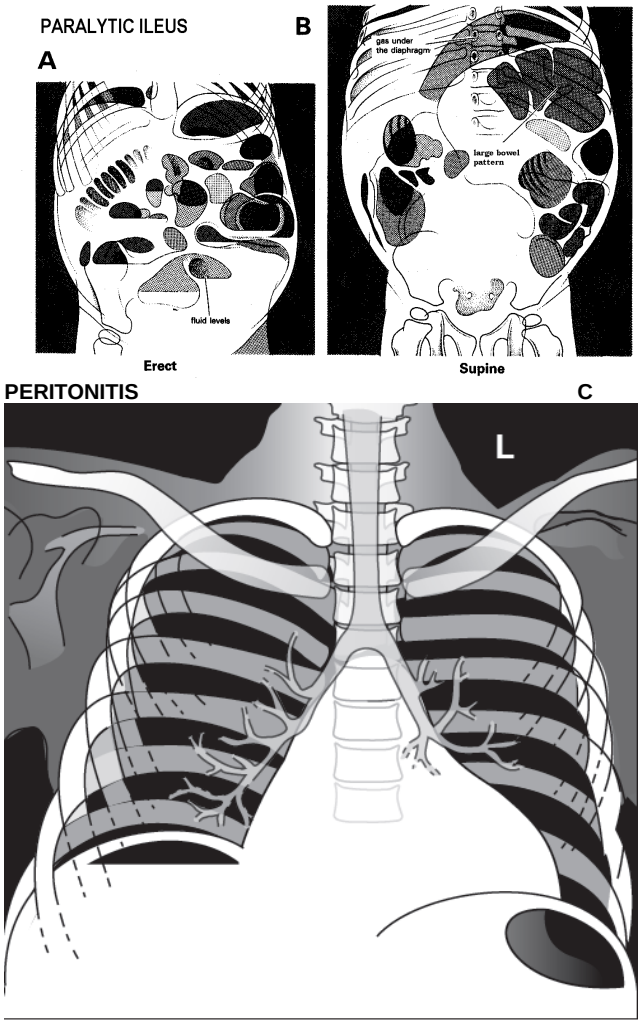


Fig. 12-2 PERITONITIS WITH DISTENDED BOWEL.
A, erect abdominal film showing the multiple fluid levels of small bowel obstruction and gas under the diaphragm. **B, supine film showing difference between small and large bowel shadows. C, erect chest film showing free air under the diaphragm** (better seen than on an erect abdominal film).

If there is no pain, but only 'gurgling and bloating', the obstruction is incomplete in the large bowel or the distal small bowel.

SITA (8yrs) presented with vague abdominal tenderness and few other signs. She was not well, and the only striking sign was a pulse of 148/min. 12h were wasted while she was observed, before a laparotomy was done and 1m of gangrenous bowel was resected.
KRISHNA (45yrs) presented with abdominal distension, colicky pain, and vomiting. She was examined by a medical assistant who noted pain in her right lower quadrant and a 'lymph node' in her right groin, and diagnosed appendicitis. He rang up the doctor, who came in, made a cursory examination, and proceeded with an appendicectomy, using a 'gridiron' incision. Her appendix was normal. Later, she had to have an emergency operation for a strangulated femoral hernia.
LESSONS (1) Strangulation can be difficult to diagnose. Tachycardia is a useful sign. (2) "When acute abdominal pain presents, one maxim I enjoin, pray don't miss that tiny lump, in one or other groin." (*Zachary Cope*)

If the pain is severe and continuous, this suggests strangulation obstruction. There may be both continuous and colicky pain. For example, there may be continuous pain from a strangulated hernia at a hernial site, and colicky central abdominal pain. Nonetheless, if pain was colicky and is now constant and severe, this implies the bowel is in serious trouble.

If pain and fever preceded the symptoms of obstruction, suspect that it may be secondary to abdominal sepsis.

(2) **Vomiting.** The higher the obstruction, the worse this is. If it is high in the small bowel, vomiting is profuse and frequent; if it is low in the large bowel, there may be no vomiting at all. Initially the vomit is yellowish, then becomes green, and after about 3 days of complete obstruction, it becomes faeculent. If paralytic ileus develops, it becomes 'effortless'.

CAUTION! Look at the vomit (you may need to pass a nasogastric tube to be sure): if it is faeculent, the large bowel or lower small bowel are chronically completely obstructed. This means you need to intervene. Vomiting never becomes faeculent if only the upper small bowel is obstructed.

(3) **Abdominal fullness.** The more distal the obstruction, the more the distension. If large bowel obstruction has come on slowly, the complaint may simply be that the 'clothes fit tightly' or that there is much gas.

(4) **Constipation.** If the small bowel is obstructed, the colon may take a day or two to empty, after which 'nothing comes out'. The absence of flatus confirms the diagnosis, but is a late symptom. Constipation may be a major concern in a culture where regular bowel movements occur 2-3 times a day. Pain may be tolerable, but the absence of a decent bowel movement may not. *Beware questions about constipation*: make sure you get answers about the frequency or absence of bowel motions.

(5) **Previous operations or peritoneal sepsis.** Adhesions and bands can follow any operation or septic process in the abdomen. (The scars may be difficult to see, especially in an obese person or after laparoscopic surgery!) In a woman, enquire especially about a past history of PID (23.1).

(b) Examination

(1) **Distension and hyper-resonance.** If there is colic and vomiting, the bowel is obstructed until you have proved otherwise. Distension is not an essential part of the clinical picture. The earliest signs of it are a little fullness in the flanks, or an increased resonance to percussion.

If the percussion note over the abdomen is 'tympanitic', there are distended gas-filled loops of bowel.

If distension is conspicuous and other signs are minimal, suspect large bowel obstruction. If it is extreme, suspect sigmoid volvulus, or Hirschsprung's disease.

If you are not sure if the distension is caused by bowel obstruction or ascites, examine for shifting dullness. Remember that fluid and gas in a distended bowel can cause shifting dullness, but that it is less obvious than with ascites.

If you are not sure if true distension is present or not, particularly in the obese, measure the girth at some fixed place, and see if it increases. Also see if the trousers or skirt fit comfortably.

(2) **Obstructive bowel sounds.** Listen for these at the time pain appears, while you are taking the history. This is essential if you are going to pick up the critical sign of intestinal obstruction during the 30sec during which peristaltic waves make a ladder pattern on the abdominal wall, accompanied by a rush of high-pitched tinkles and splashes. If you miss this opportunity, it may not return for 15mins. So, if the patient loses interest in the conversation, and grimaces with pain, listen quickly. If you hear runs of *borborygmi* (audible rumblings), or a chorus of tinkling high-pitched musical sounds *at the same time that he grimaces with colic*, there is almost certainly obstruction present. *These are very useful early signs*.
N.B. Don't mistake them for the peristaltic rushes of gastroenteritis, or normal hyperactive bowel sounds.

(3) **Visible peristalsis.** In a thin patient, look for waves of peristalsis passing across the abdomen. If he is very thin, this may be normal, especially in a young child.

(4) **A tender mass at one of the hernial orifices.** If you find a painful tender mass, this is an incarcerated or strangulated hernia, until proved otherwise.
Always examine the inguinal and femoral orifices.
CAUTION!
(i) You can easily miss a strangulated femoral or umbilical hernia, especially under an apron of fat in an obese person: it may not be tender or painful.
(ii) Rarely, a hernia may have been reduced 'en masse' (18.3) by the patient or medical personnel!

N.B. ABDOMINAL TENDERNESS is *not* a prominent feature of uncomplicated obstruction. Obvious tenderness over part of the abdomen suggests bowel ischaemia. If there is peritonitis, this means the bowel is definitely necrotic.

(5) **An old laparotomy scar** *(even if very small or even invisible from laparoscopy)* suggests that the cause of an obstruction may well be a band, an adhesion, or an area of stenosis, but *this may not necessarily be the case*.

(6) **A palpable abdominal mass** is unusual, apart from a mass at a hernial orifice. Feel carefully, here are some of the masses you might find:

If, in a child, you feel an ill-defined mobile mass (or masses), usually in the umbilical region, sometimes in the iliac fossae, it is probably a mass of *ascaris* worms.
If there is a large, slightly tender, mobile sausage-shaped mass, some of the bowel may be caught in an intussusception. If the mass is rounder, it may be bowel infarcted due to torsion.
If the mass changes its position from one day to another, and is accompanied by colicky pain, this is probably recurrent intussusception, a mass of *ascaris* worms (12.5), or constipation.
If you feel an ill-defined lump or lumps in the right lower quadrant, this may be ileocaecal tuberculosis or carcinoma. You may also feel more central lumps caused by caseating tuberculous lymph nodes.
If there is a tender indurated mass, suspect that the obstruction is due to intraperitoneal sepsis (10.1), lymphoma or tuberculosis.
If you feel hard impacted compressible masses in the colon and rectally, they are masses of faeces, and may be causing the obstruction (not uncommon in the old and debilitated).
If there are one or more masses especially at the umbilicus and also ascites and cachexia, this is probably disseminated carcinoma.

(7) **Rectal examination** *must never be forgotten!*
If you find fresh blood and mucus on your finger, or on the toilet paper, there is probably an intussusception, a strangulating lesion higher up, or carcinoma of the large bowel. Occasionally, you may feel the tip of the intussusception or see it appear out of the anus. *Don't confuse this with rectal prolapse* (26.8).
If you feel a hard mass of faeces, suspect that constipation may be causing the obstruction. Ask about dietary habits, and causes listed above.
If the rectum is empty and even 'ballooned', you may be dealing with a pseudo-obstruction.
If you feel a tense, tender, possibly fluctuant mass bulging into the pouch of Douglas, it is probably a pelvic abscess. You may feel it more easily bimanually, with your other hand exerting pressure suprapubically (10.3).
If you find a hard mass in the recto-vesical pouch (a 'rectal shelf'), it is probably malignant. Tumour deposits here may be well-defined hard lumps, or a 'shelf' caused by tumour growing into the surrounding tissue.

(c) Has the bowel strangulated?

You may not be certain about this until you perform a laparotomy. Strangulation is easy to diagnose when it is advanced, unless it is so advanced that there is septic shock. Try to diagnose it early.

Individually, the features below are not diagnostic, but the bowel has probably strangulated if several of the following features are present:
(1) Sudden onset of symptoms.
(2) Severe continuous pain: this is the result of bowel ischaemia or irritation of the parietal peritoneum. If there is minimal discomfort and absence of pain between waves of hyperperistalsis, the bowel is probably not strangulated, but only obstructed (unless it is sealed off in a hernial sac or is an intussusception).
(3) A fast pulse: this is perhaps the most reliable sign; if the pulse is <88/min, there is unlikely to be strangulated bowel present.
(4) Fever: this suggests strangulation, or sepsis. *Simple obstruction does not cause fever!*
(5) A low or falling blood pressure.
(6) Localized tenderness, or rebound tenderness; this is a sign of peritoneal irritation, and can be caused by inflammation, blood in the peritoneal cavity, or strangulation. Tenderness may be masked by loops of normal bowel over the strangulated area, so its absence is not significant.
(7) The passage of blood or blood and mucus rectally: this is typical of intussusception, but you may see it whenever the blood supply of the bowel is impaired.
(8) Signs of generalized peritonitis (tenderness, guarding, and absent bowel sounds), prostration, and shock.

(d) Abdominal radiographs in intestinal obstruction
Take films in the erect and supine positions. They can usually give you the diagnosis, its site and chronicity, and sometimes its cause, for example, intussusception (12.7).

While the patient is lying down, take a supine AP film. If he is not well enough to sit up by himself, support him in the sitting position while you take an erect film. This will be more useful than the alternative, which is a lateral decubitus film, taken from the side while he is lying down. Its purpose is to show fluid levels, and maybe gas under the diaphragm.

When you examine the films, first see if there is a distended large bowel shadow, and especially a caecal shadow. If there is, the large bowel is obstructed.

To distinguish large and small bowel shadows, remember that:
(1) Fine folds or partitions (*valvulae conniventes*) extend right across a distended jejunum which is more central in the abdomen.
(2) The ileum has no folds distally, and few proximally.
(3) The caecum is a rounded mass of gas.
(4) The *haustral* markings of obstructed large bowel are rounded and much further apart than the *valvulae conniventes* of the jejunum, and don't cross its full diameter.
(5) The large bowel is more peripheral in the abdomen, whereas the small bowel is more central.

N.B. **Free gas in the peritoneum** is usually a reliable sign of perforation. You will see it better under the diaphragm in an erect chest film, and under the abdominal wall in a lateral supine film. (Free gas may occur from gas-forming organisms in the peritoneal cavity.) On an abdominal film, look for air *both outside and inside* the bowel wall, and air outlining the lateral wall of the liver.

N.B. **Gas in the small bowel** is always abnormal, except:
(1) in the duodenal cap,
(2) in the terminal ileum (rare),
(3) in children <2yrs.

CAUTION! Never administer barium contrast media by mouth in intestinal obstruction.

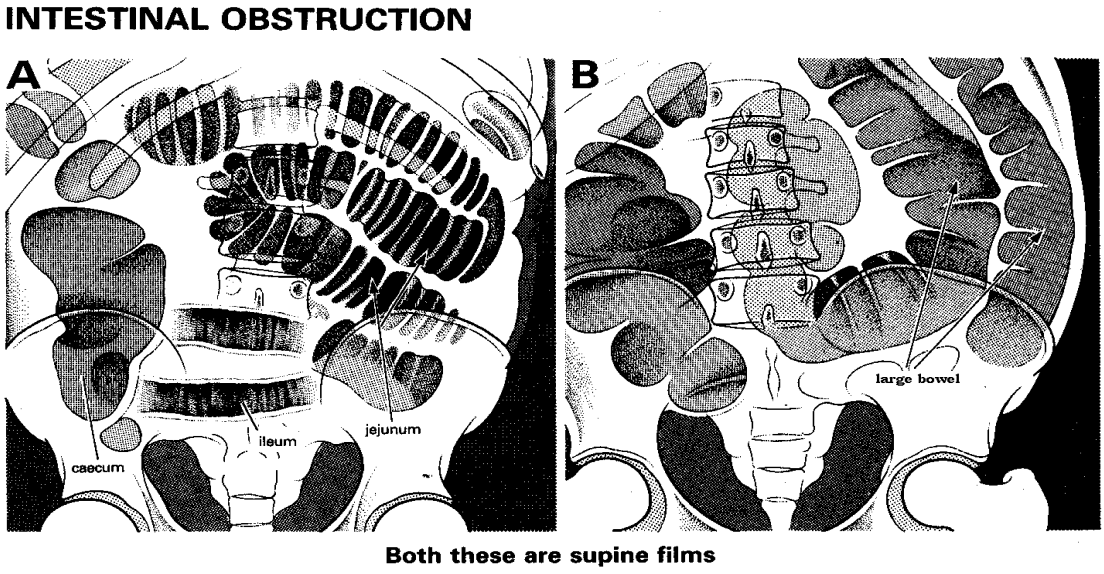


Fig. 12-3 INTESTINAL OBSTRUCTION.
Patient A has distended loops of small bowel. ***Note the different patterns of the jejunum and ileum:*** **the jejunum has *'valvulae conniventes'* (transverse bands across it), whereas the ileum is more featureless. The caecum and ascending colon are distended, but there are no signs of the transverse colon or rectum.** (A barium enema showed a carcinoma just beyond the splenic flexure.)
Patient B's large bowel is distended down to the sigmoid colon, but there is no rectal bubble. This is typical of distal large bowel obstruction; there was a carcinoma of the sigmoid colon. These are supine films, so there are no fluid levels, but the *valvulae* and *haustra* show up well.

(1) **Fluid levels in the small bowel** are always abnormal except where there is no distension. Elsewhere, fluid levels in the small bowel indicate:
(i) mechanical obstruction,
(ii) paralytic ileus, or
(iii) gastroenteritis.
Look for them in erect films. The larger and more numerous they are, the lower and the more advanced the obstruction.

(2) **Gas in the large bowel** is normal, but not if the bowel shadow is hugely distended.

If there are fluid levels in the large bowel, they may be:
(i) normal (if there are only a few), or
(ii) caused by gastroenteritis.

If the large bowel is also distended, there is:
(i) a mechanical obstruction,
(ii) paralytic ileus, or
(iii) some other cause for the dilatation, such as amoebic colitis.

If there are distended loops of large and small bowel irregularly distributed with gas in the rectum, suspect paralytic ileus.

If there is no gas in the caecum (which normally contains some gas), suspect that the small bowel is completely obstructed.

If there are distended loops in the small bowel and minimal air in the colon, suspect partial small bowel obstruction.

If there is gaseous distension of the large bowel with minimal small bowel distension, suspect large bowel obstruction or the Ogilvie syndrome.

If there is much gas in the caecum (which may be huge), the large bowel is obstructed. As the pressure builds up, the small bowel often starts to distend, because the ileocaecal valve is incompetent (in ⅔ of patients).

If you see a mottled opacity in the right lower abdomen, suspect a bezoar (13.12).

If you see a really massive gas bubble, the stomach may be dilated, or there may be volvulus of the sigmoid colon (12.9) or of the caecum and ascending colon (12.12).

If there is gas in the rectum and rectal examination is normal clinically, obstruction is unlikely; but if the large bowel is hugely distended, this suggests pseudo-obstruction.

If the large bowel is relatively empty, and the fluid levels in the erect film pass obliquely upwards from the right iliac fossa to the left hypochondrium, like a stepladder, they suggest volvulus of the small bowel.

If signs are uncertain, take more films a few hours later.

(e) Other investigations
A high Hb or haematocrit is some indication of the severity of dehydration. Urea and electrolyte measurements are very helpful. Expect a potassium deficit.

Ultrasound is usually unhelpful, but if there is a mass, it can show if this is solid, or contains worms, or gives the classic double ring appearance of intussusceptions.

(f) Difficulties in diagnosing intestinal obstruction
If there is excruciating abdominal pain, massive abdominal distension, and circulatory collapse, the possibilities include:
(1) Volvulus of the sigmoid with gangrene.
(2) Ileosigmoid knot (12-14).
(3) Volvulus of the small bowel or caecum.
(4) Perforation of a peptic ulcer presenting late.
(5) Generalized peritonitis leading to ileus.
(6) Typhoid fever with perforation.
(7) Acute pancreatitis.
You may not be able to diagnose which of these there is until you operate. Rapid resuscitation and urgent surgery is necessary, but try to exclude pancreatitis first.

If there are obvious abdominal signs, but the patient looks comparatively well (and he has not been vomiting), suspect large bowel or incomplete small bowel obstruction.

If there are the other signs of obstruction, but loose stools are passed with or without flatus, there may be:
(1) An incomplete large bowel obstruction.
(2) A pelvic abscess.
(3) A Richter's hernia (18.1).

If there is a history of several days of fever, anorexia and localized abdominal pain, followed by colicky pain and the other symptoms of obstruction, suspect that obstruction has followed intraperitoneal sepsis. Distension may mask the abdominal findings, but you may be able to elicit deep tenderness and induration in the right lower quadrant, suprapubically, rectally, or, in a woman, vaginally.

If the abdomen is distended and associated with vomiting but no typical colicky pain of obstruction, suspect ileus rather than obstruction, especially if there is toxaemia and dehydration. Obstruction appears spontaneously, whereas paralytic ileus usually follows some good reason for it, such as local or general peritonitis, a previous operation, or an intraperitoneal injury or haemorrhage.

If signs of obstruction develop after surgery, you will find it difficult to know if the obstruction is mechanical or due to the paralysis caused by ileus (12.16).

12.4 The management of intestinal obstruction

Operation is mandatory for ischaemic bowel; simple mechanical obstruction may resolve without operation, but if it fails to improve after 48h, operate. The detailed indications for operating are listed below. Operate at the optimum moment after you have rehydrated a patient, but *don't operate if the condition is hopeless*.

Rehydrate rapidly over a few hours. If you rehydrate energetically, you should be able to operate within 4h, and certainly within 6h. If you suspect strangulation obstruction, try to operate within 2h, and rehydrate as best you can before doing so. If the patient is conscious with a normal blood pressure and is passing urine, he is probably fit for operation.

At the same time pass a nasogastric tube and drain the fluid and gas from the dilated stomach and upper small bowel. This will stop vomiting, and may reduce the distension. Most importantly, it will reduce the danger of aspiration of stomach contents during induction of anaesthesia.

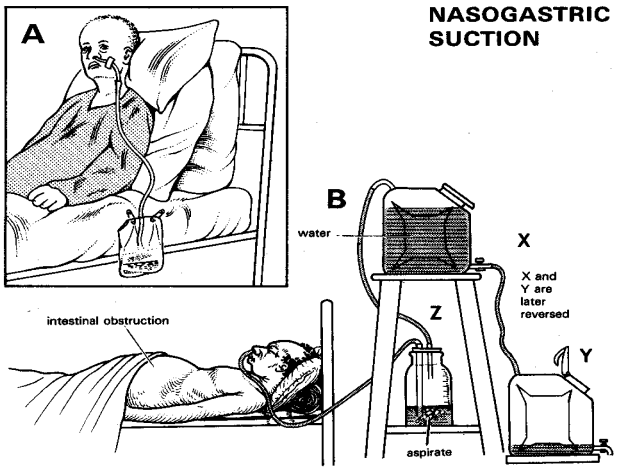


Fig. 12-4 NASOGASTRIC SUCTION.
A, pass a large (Ch16) nasogastric tube and aspirate it with a 20 or 50ml syringe ½hrly. Meanwhile, let it syphon freely into a drip bag beside the bed. Cut off the corner of the drip bag to let the air out. B, if you don't have an electric sucker, you may find this apparatus useful. X,Y are 2 jerry cans with pipes and taps soldered in. Water flows from X to Y, creating a negative pressure in X. When X is empty, reverse X and Y. Z collects the fluid and measures the flow.
After Les Agrégés du Pharo, Techniques Elémentaires pour Médecins Isolés. Diffusion Maloine, 1981 Fig. 168 with kind permission.

When you have resuscitated a patient, he may improve so much that you may wonder if he really needs a laparotomy. If so, try clamping the nasogastric tube to see if the signs and symptoms recur.

N.B. Small bowel obstruction de novo *without previous recent surgery will usually recur because the underlying pathology has not been relieved.*

Your primary task is to save life, so perform an operation which will achieve this. In desperate cases, removing the underlying cause is a secondary consideration, and that may have to wait until later. Sometimes, you can remove the cause quite easily: for example, you may be able to cut some easier adhesions.

Don't embark on complicated operations which need much dissection unless you are experienced and have good back-up. Even then, *always remember that a long operation in an acutely ill patient is not a good idea*.

Open the abdomen with the greatest possible care: you can so easily perforate the bowel and flood the abdomen with small bowel fluid. Distended loops of bowel will bulge through the incision. Deliver them on to the surface, and *don't try to examine the depths of the abdomen until you have done so:* they will continually obscure your field.

Because distended loops of bowel are so difficult to work with, decompress them. Doing so makes distended bowel much easier to handle, makes the abdomen easier to close, reduces the risk of anastomotic leak if you have to resect bowel, and reduces the risk of post-operative vomiting and aspiration. The danger of decompression is that it inevitably contaminates the peritoneum a little, unless you use the retrograde method (via a nasogastric tube). But carefully opening distended bowel outside the abdomen, hanging over its edge, with the proper precautions causes much less contamination than an uncontrolled burst, which is the probable alternative. So, if bowel is greatly distended, decompress it.

IF THE PATIENT IS MORIBUND, RESUSCITATE AGGRESSIVELY FIRST, TO GET A URINE OUTPUT

(a) Pre-operative preparation

(1) **Nasogastric suction.** Pass a nasogastric tube of a suitable size, and aspirate it regularly (4.9). Make sure it reaches the stomach, and be sure it is draining properly. If you are not sure of its position, blow a little air down the tube and listen over the stomach, or use litmus paper to test for aspirated gastric acid content. If you are still unsure, get an abdominal radiograph. Aspirate actively to remove air and fluid before operating. Syphon the fluid into a bag hanging below the bed, and aspirate every 15-30mins with a syringe or suction machine. Empty the stomach thoroughly.

(2) **Resuscitation**
This is critical. If there is severe dehydration, and you fail to resuscitate, death is almost inevitable. If the obstruction has lasted >24h, dehydration is certain, especially if vomiting has been profuse.
Start a fluid balance chart, and rehydration. A 60kg adult will require 4-8L to correct moderate to severe dehydration; you can calculate a total loss of 3L per day of total obstruction and assume the correctable deficit is half of this. Infuse the first half of this correctable deficit as Ringer's lactate or saline and the second half alternating with 5% dextrose. Fluid replacement is more important than potassium replacement. In late cases add 20mmol of K^+ to each litre of fluid after the 1st litre. (Or you can use half-strength Darrow's solution, which has [K^+] of 17mM, instead of 5% dextrose.)
Set up a central venous infusion for critically ill dehydrated patients, if you can do so.

If the patient is thirsty, and the lips and tongue are dry, dehydration is mild: use at least 4L of fluid.

If there are also sunken eyes and loss of skin elasticity, dehydration is moderate: use about 6L.

If there are also oliguria, anuria, hypotension, and clammy extremities, dehydration is severe: use about 8L.

If there is also weakness and disorientation, fluid loss is probably >8L. *Don't be afraid to infuse up to 4L for the first hour*. Set up a CVP line, if you can.

In the elderly or those with cardiac problems, check the lung bases for crepitations, and the jugular venous pressure or the CVP. IV fluid replacement *and* diuretics may be necessary.

If the bowel strangulates, its veins block before its arteries, so that blood is lost into the lumen, so transfuse 1 unit of blood per 50cm of strangulated bowel.

If you have corrected the hypovolaemia as shown by an adequate urine output, or a normal CVP, but there is still hypotension, this is probably due to septic shock.

(3) **Urethral catheterization.** This is necessary for a very ill patient to measure the urine volume hourly. If the patient is not very ill, its risks may outweigh its advantages. If an adult passes 35-60ml/hr, the kidneys are being adequately perfused, and the blood volume is becoming normal. For a man, use of Paul's tubing is often better. *Don't insert a catheter if no one will record the urine output!* However, if there is large bowel obstruction and you plan to operate in the pelvis, it is important that the bladder remain empty to give you more room to operate.

(4) **Antibiotics.** Use perioperative antibiotics (2.9): chloramphenicol 50mg/kg IV qds or gentamicin 3mg/kg IV od *plus* metronidazole 7·5mg/kg tds.

(b) Non-operative treatment of intestinal obstruction

INDICATIONS
(1) A mass of *ascaris* worms (12.5).
(2) Plastic tuberculous peritonitis (16.3).
(3) A localized inflammatory mass, such as an appendix mass, a pyosalpinx, or PID.
(4) A pelvic abscess which can be drained rectally or vaginally (10.3).
(5) Some patients with adhesions (12.6) and incomplete obstruction.
(6) Typhoid fever causing partial mechanical obstruction or ileus (not uncommon).
(7) Constipation or ingestion of foreign material.
(8) Crohn's disease.
(9) *Oesophagostomiasis* ('Dapaong tumour', 'kounkoul').
(10) Pseudo-obstruction.

N.B. Always operate if there is even a suspicion of strangulation obstruction.

METHOD. Continue nasogastric suction and IV infusions. Make careful observations. If you continue to restrict fluids orally for more than a few days, try to add at least 8·5MJ (about 2,000kcal) of energy to the daily intake. If possible, administer this as 50% dextrose into a central vein. When the nasogastric aspirate has reduced, instil 15-30ml gastrografin, if you can, and see if this promotes a bowel action.

If you suspect simple constipation, instil a phosphate or soap-and-water enema. If the faeces are rock hard and fail to move, remove them manually under GA. A 'Murphy skid' (normally used to dislocate the hip) is a useful instrument to help you do this. Advise a short course of laxatives and a high-fibre diet: an instruction sheet is useful to give to patients. Chopped rice husks are ideal. *Don't use liquid paraffin*: aspiration causes severe pneumonitis, and if used with illicit drug 'body packers' it can cause the packets to dissolve and the opiate carried to be rapidly absorbed.

If you suspect *oesophagostomiasis* (especially in Northern Ghana and Togo), use albendazole 10mg/kg od for 5 days. Signs of improvement are:
(1) Reduction in the gastric aspirate to <500ml/day.
(2) Reduction in abdominal distension.
(3) Return of bowel sounds to normal.
(4) Less pain. No tenderness.
(5) Finally, flatus and the passage of stools.

DECOMPRESSION OF LARGE BOWEL OBSTRUCTION (12.9)

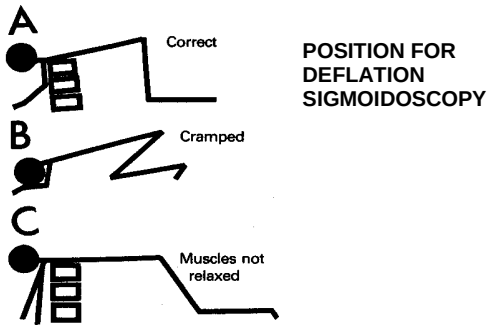


Fig. 12-5 THE KNEE-ELBOW POSITION FOR SIGMOIDOSCOPY FOR SIGMOID VOLVULUS.
A, correct position. B,C, two incorrect positions. *Don't anaesthetize the patient.* *After Shepherd JJ. Management of Sigmoid Volvulus. Tropical Doctor 1971;1(4):174-6 with kind permission.*

INDICATIONS
(1) Sigmoid volvulus without peritonitis.
(2) Pseudo-obstruction.

METHOD: DEFLATION BY SIGMOIDOSCOPY (GRADE 1.2)
Place the patient preferentially into the knee-elbow position (12-5). This is not very socially acceptable, and some elderly patients may not tolerate it, but you are more likely to be successful than with the left lateral position because the weight of fluid in the loop will pull the apex of the sigmoid out straight. Pass the sigmoidoscope (26.1).

If you don't have a sigmoidoscope, or its light does not work, you may succeed in deflating the colon with a soft rubber tube while in this position. The sigmoidoscope usually travels 20-25cm before it reaches the point where

the colon is twisted, but you may not be able to reach this point. If you do, look at the mucosa carefully. If it looks normal through the sigmoidoscope, hold the 'scope firmly, and pass a large (Ch36 or 12mm) well-lubricated soft rubber tube through it. With a gentle rotatory movement, ease the tube past the twist into the high-pressure area of the dilated sigmoid. *Don't use force or you may perforate the bowel!*

If you succeed in deflation, you will be rewarded by much flatus and some loose faeces. Both you and the patient will recognize that you have relieved the obstruction. Withdraw the sigmoidoscope, taking care *not to pull out the flatus tube when you remove the sigmoidoscope!* Using LA, suture the flatus tube to the anal margin, and leave it in place for 2 days. It may continue to discharge liquid faeces, so attach an extension tube to it, and lead this into a bucket beside the bed. If drainage stops, wash out the tube. *Don't leave the tube in for more than 72h*, or it may cause pressure necrosis.

CAUTION!

(1) *Don't anaesthetize the patient or use a heavy sedative*. Pain during or after sigmoidoscopy is a useful indication of trauma or gangrene.

(2) If the sigmoid is gangrenous, *trying to deflate it is dangerous*; you may perforate it.

(3) *Don't pass a sigmoidoscope using force or without seeing where you are going:* you may push it through the colon.

(4) Wear an apron and theatre boots, because a huge quantity of flatus and fluid may rush out. *Extinguish any cigarettes in the vicinity and stand well back!*

If you fail to pass the sigmoidoscope far enough, consider whether there might be a carcinoma. Try the knee-elbow position if you have been using the left lateral position.

If you see any discoloration through the sigmoidoscope, or any blood-stained fluid, or the patient cannot tolerate the procedure, suspect strangulation and prepare for an immediate laparotomy.

If the fluid which runs out is bloody, assume that the sigmoid has an area which is non-viable. Prepare for an immediate laparotomy. (*A smear of blood is not a sufficient indication.*)

METHOD: USE OF NEOSTIGMINE

This is *only* indicated for pseudo-obstruction, so perform a sigmoidoscopy as above to exclude volvulus or other causes of large bowel obstruction. The sigmoidoscopy may help to deflate the colon partially. Administer 2mg neostigmine IV slowly. Monitor the patient carefully. The bowel will usually deflate immediately. Repeat neostigmine after 1hr if the effect is only partially successful.

CAUTION! Don't use neostigmine in asthmatics, epileptics, pregnancy or breastfeeding mothers, or if the blood pressure is low. Have atropine 0·6mg IV ready as an antidote if bradycardia results.

N.B. Fashioning an ileostomy or colostomy is not helpful.

(c) Operative treatment for intestinal obstruction (GRADE 3.4)

(1) **Incision**

A midline incision, ⅓ above the umbilicus and ⅔ below it, is usually best. Start with a 10cm incision and enlarge it up or down as necessary. You will probably find that the posterior rectus sheath and the peritoneum will appear as 2 distinct layers, now that the abdominal wall is distended. Have moist packs (laparotomy pads) ready. Put them into warm water and then wring out most of the fluid. Use them to cover any bowel that bulges out of the wound, and to wall off any fluid that spills.

If there is an old scar, open the abdomen at one end of it to avoid a loop of bowel which may be adherent (11.2). This is safer than making a parallel incision, which may lead to necrosis of the abdominal wall between the 2 incisions.

If there is a strangulated external hernia, make the appropriate incision (18.6,8).

CAUTION!

Open the abdomen with the greatest care (11-2). Use your fingers! Distended loops of bowel will be pressing up against the internal abdominal wall, and the smallest nick of a scalpel will go straight into the bowel. You can so easily cut the thin wall of the distended colon and cause a fatal peritonitis.

Note which parts of the bowel are distended; you will need to know this later, to decide where the obstruction is.

(2) **Handling the bowel**

If it is very distended, decompress it before you do anything else, especially if there are multiple dense adhesions which prevent you lifting out the bowel from the abdominal cavity. If it is less distended, use a moist swab to lift the dilated loops from below gently out on to the surface of the abdomen.

Never pull on the bowel: it will tear! Handle bowel with the greatest care. If you handle it roughly, you will prolong the period of post-operative ileus. Be especially careful of the caecum. It is often greatly thinned, and if it does burst, soiling will be particularly dangerous.

Don't let loops of the bowel get dry: cover them with moist packs. If they are heavily laden with fluid, ask your assistant to support them.

If you make a very superficial cut only into the seromuscular wall of a loop of bowel, leave it alone. Close a deeper injury (you will notice the mucosa pouting out) with a purse-string suture or by sewing it up transversely in two layers, while trying to keep spills to a minimum.

If you soil the abdomen with faeces, suck them out immediately. Irrigate the peritoneal cavity thoroughly with liberal amounts of warm fluid.

(3) **Decompression for intestinal obstruction**
Be safe, and decompress the bowel if there is any risk of rupturing it:
(i) if it gets in your way unduly,
(ii) if dense adhesions prevent you lifting it out of the abdomen, or
(iii) if it prevents you closing the abdomen.

If you have to open the bowel, pack off the remaining abdomen with moist swabs in order to limit contamination with bowel content.
Make sure your suction is connected and working well.

There are 5 ways to decompress the bowel:

(i) RETROGRADE DECOMPRESSION is the method of choice, provided the bowel is not too oedematous and friable. It is useful for the entire small bowel. Start at the jejuno-ileal junction, and milk the contents proximally between your straight index and middle fingers into the stomach. Palpate this from time to time if it gets full, in order to dislodge trapped air and thick fluid blocking the nasogastric suction tube. You may need some firm pressure on the proximal jejunum. When you have emptied enough fluid out of the jejunum, massage the fluid from the ileum into it and repeat the process. As you decompress, ask the anaesthetist to keep aspirating fluid from the stomach.

This is usually the best method, but make sure that suction through a *large bore* nasogastric tube is working properly, or the fluid may spill and your patient may aspirate it! It may prove difficult or impossible if the bowel content is very thick, as in distal small bowel obstruction or large bowel obstruction. *Don't use this method in children where the anaesthetist has put in an uncuffed endotracheal tube.*

(ii) NEEDLE PUNCTURE. Use a specially prepared spinal needle. This will remove gas, but is soon blocked by food particles when you try to remove liquid. A spinal needle, connected to a rubber tube, is especially useful for the sigmoid colon and the caecum, which are often distended only with gas. Its advantage is that there is no need to insert a purse string after removing the needle. Pack off the colon well. Push the needle through a *taenia coli*, and advance it longitudinally between the muscle coats for 3cm. Then angle it inwards through the circular muscle to reach the lumen. Keep its point in the gas and clear of the fluid. If it blocks, pinch the rubber tube connected to the needle, then pinch it again distally. This should provide enough pressure in the needle to free it. If you insert the needle obliquely, there is no need to close the hole, which should not leak.

(iii) POOLE'S SUCTION. Select a site in the bowel, and empty it of its content by manual massage externally, and then place two non-crushing bowel clamps on either side of the emptied portion. Insert a purse-string suture on the antimesenteric border, and make an incision in its centre into the lumen of the bowel, and push the Poole's sucker through. Release the proximal clamp.

The Poole's sucker has a guard with several holes. It blocks less easily than a needle, but the risks of a spill are greater. It always blocks eventually.

Removing it, unblocking it, and reinserting it may be necessary, but is likely to cause a spill. (A Yankauer sucker does not have a trocar, so it is difficult to use without spilling.)

CAUTION! After you have inserted a sucker, *don't remove it unless you have to*. If you have to remove it to clear it, pack off the peritoneal cavity to avoid spillage, and discard any contaminated packs.

Alternatively, you can use a large Foley catheter; insert it (with extra side holes cut close to the balloon) connected to the sucker. Suck the bowel empty. Then blow up the balloon and 'milk' it along the bowel, sucking as you go. If it blocks, inject some saline and start again. Withdraw it, sucking as you go, then close the purse string.

(iv) USE OF THE SAVAGE DECOMPRESSOR. Insert a purse-string suture on the antimesenteric border of the bowel as before, make an enterotomy incision in the centre of this, and push the decompressor with its trocar through. Withdraw the trocar and close the proximal opening of the decompressor with its threaded cap. With your thumb on the vent to control the degree of suction, start sucking out gas and fluid. *Make sure that intestinal fluid, which may come gushing out of the suction vent, pours into a bowl outside the abdomen!* Pass the decompressor proximally and distally, carefully threading the distended loops of bowel over it as you suck. To minimize clogging the holes, remove your finger from the vent from time to time. This will reduce the suction and let the food particles fall away. Or, more effectively, reintroduce the trocar. When you have decompressed enough bowel (there is no need to decompress it all), remove the decompressor, close the purse string, and put it in the 'dirty basin'. Reinforce the purse string with a second layer of sutures, 3 mm beyond the first, going through the seromuscular layer only.

(v) FORMAL DRAINAGE. Mobilize a suitable segment of bowel and make sure it can hang outside the abdomen; pack off the peritoneal cavity and other organs with swabs, and hold the bowel with Babcock forceps. Preferably place two non-crushing bowel clamps across the bowel and make a 1cm enterotomy on the antimesenteric surface between them. Release one clamp in turn and allow the bowel content to drain into large bowls held carefully to catch the fluid, as you massage the fluid from each end towards the hole you have made. Then close the hole in 2 layers (14.3).

This is the best method if you have to resect bowel anyway: drain the bowel contents from the open ends of bowel before anastomosis (11.3).

Measure the fluid you have aspirated to see how much has been lost. Record this in the notes.

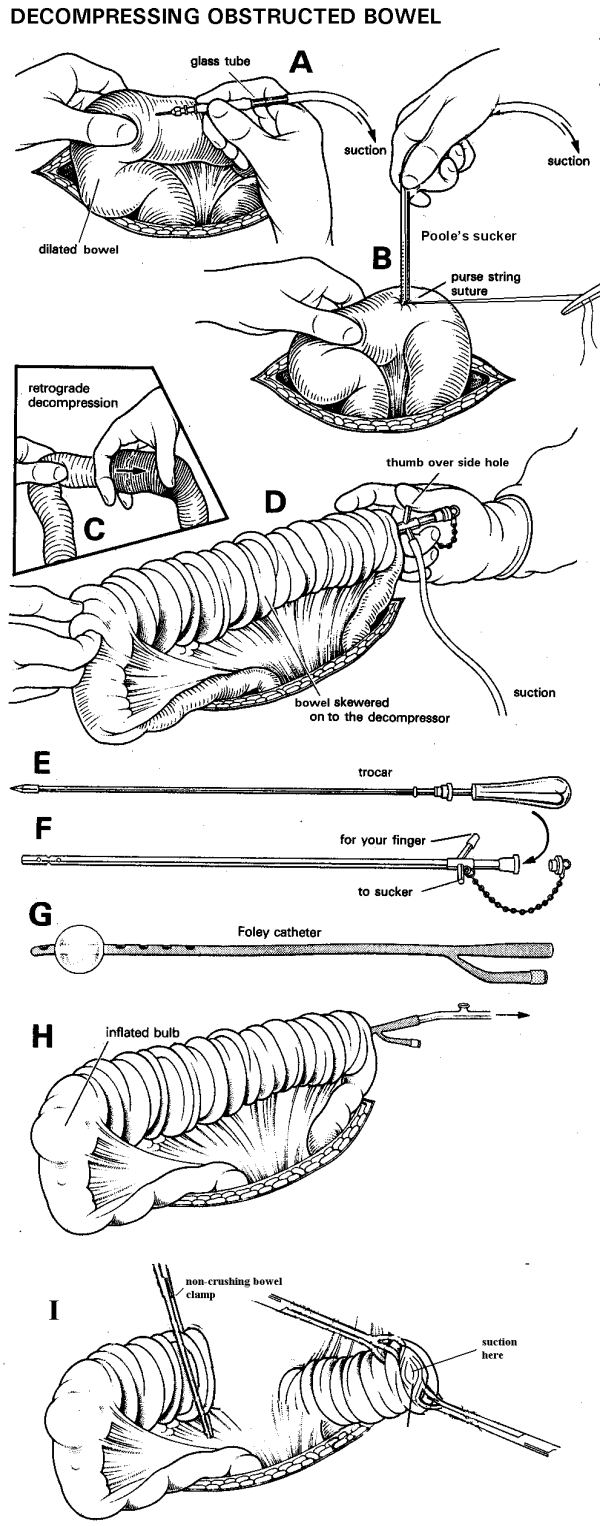


Fig. 12-6 DECOMPRESSING DILATED OBSTRUCTED BOWEL. A, using a needle. Note the glass tube, so that you can see what you are sucking. *(This will only work well if the bowel is distended with air, not faecal matter.)* B, using a Poole's sucker held in with a purse-string suture: *N.B. You must hold the bowel away from the laparotomy wound to avoid dangerous spillage*. C, by retrograde massage between your index and middle finger. D-F, Savage's decompressor. G,H, using a Foley catheter: blow up its bulb after introducing it, then milk the bulb along the bowel. I, decompressing the open bowel, using Babcock forceps to hold it open over the edges of the laparotomy opening, avoiding spillage into the abdominal cavity. *Note the other loop is closed with a non-crushing clamp.* First empty the proximal loop; then clamp this loop and then empty the distal loop, *holding it outside the abdomen.*

(4) **Findings in the abdomen in intestinal obstruction**
Here are some of the many things you might find, either immediately or after a careful search:

If there is straw-coloured fluid in the abdomen, there is probably only a simple obstruction.

If pus is present, there is an inflammatory lesion somewhere.

If loops of the bowel are red and congested, peritonitis is present.

If loops of the bowel are dusky and plum-coloured, they are strangulated.

If a huge purple mass fills the abdomen, it is likely to be a strangulated sigmoid volvulus.

If most of the small bowel is deeply congested and haemorrhagic, it has probably undergone volvulus.

If the fluid is bloody, very dark and foul-smelling, the bowel is probably necrotic, or recently perforated.

If much of the small bowel is purple or black, but is not twisted, there is mesenteric vascular thrombosis.

(5) **Finding the cause of intestinal obstruction**
First decide if the obstruction is proximal or distal to the caecum. Your task will be easier if you decompress the bowel and then lift as many of its loops on to the abdominal wall as you can. Protect them by wrapping them in a moist pack or in a sterile plastic bag.

If the caecum is distended when you open the abdomen, the obstruction is distal to it, so feel the upper rectum and sigmoid. Then raise the left side of the incision and feel the descending colon. Then feel the splenic flexure, the transverse colon, the hepatic flexure, and the ascending colon. You may need to extend the incision to feel the splenic flexure properly. Be very careful *not to rupture the caecum* especially if it is >12cm diameter.

If the caecum is collapsed, the obstruction must be in the small bowel. First look for a strangulated hernia by palpating the hernial orifices from inside the abdomen; you should of course have examined them earlier from outside. If these are clear, ask your assistant to retract the right side of the lower end of the wound. Pick up the last loop of the ileum, start at the ileocaecal junction, and run the small bowel through your fingers, loop by loop, and then return it to the abdomen. Try to handle only collapsed bowel distal to the obstruction, and not fragile distended bowel proximal to it. The place where collapsed bowel meets distended bowel is the site of the obstruction.

If you cannot find a collapsed loop, withdraw the distended loops and explore the pelvis and right iliac fossa.

If you find a loop which feels 'tethered', and you cannot lift it into view, it is probably the site of the obstruction. Expose this area well, by appropriate retraction, by packing bowel away, and by lengthening the incision. If the bowel is firmly stuck in the pelvis, *don't try to dissect it out when you can't see properly*: it is then best to pinch it firmly and extract it with your fingers, knowing you will have to resect that portion. *Be sure you have decompressed the proximal bowel before you do this!*

If the obstruction is difficult to find, remember that it is more likely to be in the small bowel.

If you are not sure if a piece of bowel is large or small, remember that large bowel has *taenia coli* running over its surface.

If you don't know which piece of bowel is proximal and which is distal, pass your hand down to the root of the mesentery, and remember that it runs obliquely downwards from left to right. Follow the bowel to its end.

If you really are lost as to which way the bowel goes, you have no alternative except to deliver the obstructed loops until you reach the duodenum proximally, or the obstructed focus distally. Check if the bowel is viable (11.3, 11-6).

CAUTION! Don't try to rely on the standard differences between ileum and jejunum. Obstructed bowel loses some of its characteristic features.

If you cannot find or release the cause of the obstruction, and yet the bowel is grossly distended, decompress it preferably as near proximal to the obstruction as possible. Search the length of the bowel again, including in the pelvis, for missed pathology. Could this be a pseudo-obstruction? Or a Chagas megacolon? Or a toxic megacolon from *amoebiasis* or ulcerative colitis?

If you find a mass within the bowel, but not attached to its wall, it is probably a foreign body (gallstone, bezoar, or other ingested foreign body): make a transverse enterotomy and remove the obstruction.

SPECIAL METHODS. See elsewhere for: obstruction due to bands and adhesions (12.6), inguinal hernias (18.6), femoral hernias (18.7), other hernias (18.10 to 18.14), *ascariasis* (12.5), intussusception (12.7), volvulus of the small bowel (12.8), sigmoid volvulus (12.9), volvulus of the caecum (12.12), and abdominal tuberculosis (16.1).

(6) **Closing the abdomen after intestinal obstruction**
Before you close the abdomen, have a last look at the bowel, check the anastomosis (if you have made one), and look for bleeding, and at the length of the proximal bowel (if you have decompressed this) for damage or leaks. Wash the abdominal cavity thoroughly. Handle the bowel carefully.

Now turn your attention to closing up: do this with particular care as a 'burst abdomen' is a major risk (11.14). Distension may also recur, hopefully only temporarily.

Remember to bring the omentum down over the bowel to separate it from the abdominal wall; this will often prevent adhesions, and is especially important if you have made an anastomosis. Close the abdomen by Everett's method (11.8).

If the abdomen is difficult to close, decompress the small bowel into the stomach, and again empty it by aspiration through the nasogastric tube. If necessary, use the 'fish' (11.8). If this fails, leave the incision open, and fix a vacuum dressing in place (11.10).

If you have had to resect bowel, or the peritoneum has been soiled, wash out the peritoneal cavity with warm sterile water.

If there has been significant soiling, leave the skin edges unsutured for delayed primary closure (11.8).

(7) **Post-operative care for intestinal obstruction**
Continue nasogastric suction until flatus is passed, the distension is becoming less, the bowel sounds are returning, and the nasogastric aspiration is ≤500ml daily of light-green fluid, which is the normal gastric secretion. (If the aspirate is persistently large, but flatus is being passed, withdraw the drain 10cm: it might have slipped into the duodenum.) Commence free oral intake when faeces has been passed.

Continue to keep an accurate fluid balance chart. Measure the urine output, and when necessary the CVP. An adult in the tropics loses at least 3L of fluid a day (skin 1L, lungs 0·5L, urine 1·5L). Replace this with 1L of 0·9% saline and 2L 5% dextrose. In a hot humid environment increase these volumes by 50% after the first 24-48h. Monitor the urine output: this should be at least 1·5L by the 3rd post-operative day.

Replace the fluid you aspirate from the stomach in addition. You can usually replace it with IV 0·9% saline or Ringer's lactate.

As soon as a post-operative diuresis starts (at 24-60h), replace the potassium loss. Basic needs are about 40mmol/24h. But if IV fluids are still needed after 48h, up to 80mmol of potassium a day may be necessary, depending on the volume of secretions lost. Replace this loss either as a 1M solution added to the IV fluids, or as Ringer's lactate ($[K^+]$=4mM), or as Darrow's solution ($[K^+]$=34mM) or as half-strength Darrow's ($[K^+]$=17mM).

(8) **Difficulties with intestinal obstruction**
If you don't know what to do about an obstruction, and the situation looks very complex, consider bypassing the obstruction by anastomosing a distended to a collapsed loop. Or, if you cannot do this, bring out the proximal loop of bowel as a stoma.

If the large or small bowel is not viable, but you are not sure how to make a safe anastomosis, exteriorize the bowel. Bring the non-viable bowel out through a stab wound which is big enough to accommodate it.

Suture its margins, at a point where it is healthy, to the skin of the wound, so that it won't slip back inside. Close the laparotomy wound carefully. The stoma will be of rather generous proportions, sticking out of a short wound in the flank. Then cut off the non-viable bowel about 3cm from the skin to form a double-barrelled stoma. You will have to replace orally the large volumes of small bowel fluid lost from an ileostomy or jejunostomy (11-12).

If the small bowel is not viable to within 5cm of the caecum, it is not safe to resect bowel without mobilizing the caecum, including this and making an ileocaecal resection. If you cannot do this, perform an end-to-side ileocolic anastomosis and close the distal ileal stump, or make an ileostomy (11-12).

If obstruction is clinically present, and yet you cannot find any cause for the obstruction, the only useful thing to do is to decompress the bowel. There may be just spasm or a pseudo-obstruction.

If post-operatively, the bowel sounds don't return, the fluid you aspirate does not decrease, and the abdomen becomes more distended, paralytic ileus is developing. Consider an anastomotic leak (12.16).

If there is diarrhoea post-operatively, this is common after any operation to relieve intestinal obstruction: it is a sign of recovery and usually clears up spontaneously if there is no persistent fever. Measure the stools and replace the volume accurately IV with Ringer's lactate or 0·9% saline with added potassium.

12.5 *Ascaris* obstruction

Obstruction of the bowel by *ascaris* worms is the classical indication for non-operative treatment. Heavy infestations can obstruct the bowel, usually in the distal jejunum and proximal ileum, partly or completely. The children of impoverished shanty-towns are most heavily infected, but in only a few of them is the infestation so heavy that it obstructs the bowel. The number of worms a child has is directly proportional to the number of ova he has swallowed. So the prevalence of *ascaris* obstruction is a sensitive indicator of very poor hygienic conditions indeed. Sadly, the environment of many cities is deteriorating, and *ascaris* obstruction is becoming more common.

A child between 2-14yrs, or occasionally a young adult, usually has several mild attacks of central abdominal pain and vomiting, before the small bowel finally obstructs. Often, he vomits worms, or they may come out of the nose, but this by itself is unimportant unless he becomes unconscious.

If obstruction is partial, as it usually is when it is caused by a bolus of living worms, non-operative treatment commonly succeeds. Even if a solid mass of tightly packed dead worms obstructs the bowel completely, you can usually treat this non-operatively.

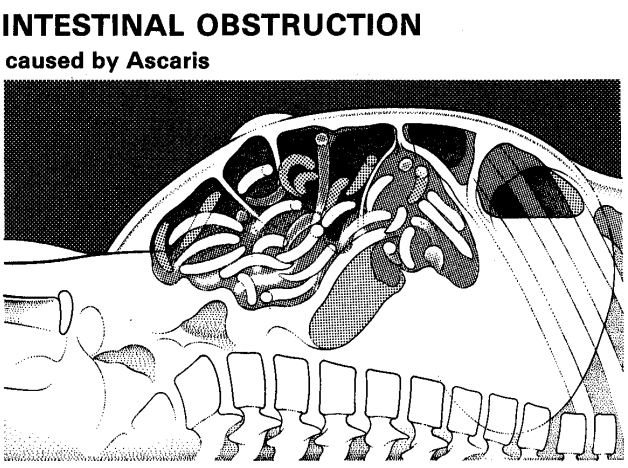


Fig. 12-7 INTESTINAL OBSTRUCTION caused by *ascaris* worms. This is a lateral radiograph in the supine position. Note the fluid levels and gas-filled coils of bowel. Rarely are worms seen quite as clearly as this! Typically, they are coiled in a mass, like 'Medusa's head'. *Kindly contributed by John Maina.*

Complete obstruction commonly follows an attempt to 'de-worm' a heavily infested child. It paralyzes the worms, and so makes them even more likely to form a ball and obstruct the bowel. So wait to 'de-worm' a child until the obstruction has passed. *Don't operate if you can avoid it*, but a child may deteriorate rapidly from volvulus (a closed loop obstruction) or a perforation. At operation, try not to open or resect healthy bowel. Instead, try to milk the worms through the small bowel into the large bowel, from where they will be expelled naturally. The danger of anastomosing small bowel is that worms find their way through the anastomosis out of the bowel causing a leak; the patient with the fistula (11-26) had bowel resected for *ascaris* obstruction. *Ascaris* worms occasionally obstruct the biliary tract and cause jaundice (15.6), or the appendix and cause appendicitis. Sometimes, they block drainage tubes. They may also be aspirated into the bronchi!

(a) History. Enquire about:
(1) recent attacks of colicky abdominal pain.
(2) vomiting worms, or passing them rectally or nasally.

(b) Examination. The child is unwell. Distension is mild to moderate. There may be visible peristalsis. Feel for a mobile irregular mass in the centre of the abdomen, 5-10cm in diameter, firm but not hard, and only moderately tender. This feels indeed like a mass of worms, and there may be more than one mass. It may change in position and you may feel the worms wriggling under your hand. If the abdomen is very distended, the mass will be difficult to feel. Signs of peritoneal irritation are absent, unless there is bowel perforation or a complication.
N.B. Examining stools for ova may not contribute to the diagnosis in a community where most children have worms.

(c) Abdominal radiographs show multiple fluid levels on an erect film, and you may see the worms (12-7). They may not be the cause of the symptoms. Radiographs are not necessary if you can make the diagnosis clinically.

(d) Ultrasound will demonstrate a mass filled with worms which you can see moving. Ascites is a bad sign.

DIFFERENTIAL DIAGNOSIS includes the other common causes of intestinal obstruction in childhood.
Suggesting intussusceptions: a more regular sausage-shaped mass, the passage of blood and mucus rectally, and tenderness which is more acute.
Suggesting an appendix abscess causing obstruction: the mass is not mobile, tenderness is more acute, a swinging temperature and toxaemia.
Suggesting Hirschsprung's disease: longstanding gaseous abdominal distension with chronic constipation.
N.B. Tapeworms don't cause obstruction.

(e) Non-operative treatment for *ascaris* obstruction
INDICATIONS. The child's general condition is good, the colic is intermittent, and the vomiting is mild. There are no signs of peritoneal irritation.

METHOD. Introduce 15-30ml of gastrografin through the nasogastric tube and clamp it for 4h: this often dehydrates and disentangles the worms.
CAUTION!
(1) *Don't try to 'de-worm' a child with partial or complete obstruction*. Wait until the obstruction has gone.
(2) *Don't use purgatives*: they may precipitate intussusception or volvulus.

(f) Operative treatment for *ascaris* obstruction
LAPAROTOMY FOR *ASCARIS* OBSTRUCTION (GRADE 3.4)

INDICATIONS. *A laparotomy is not often needed*.
Absolute indications are:
(1) Signs of perforation, which is caused by pressure necrosis from the obstructed mass of worms, which may lead to migration of a worm into the peritoneal cavity.
(2) Signs of peritonitis associated with intussusception, volvulus, appendicitis or, rarely, diverticulitis of a Meckel loop.
(3) Persistent abdominal pain with a tender palpable mass.
(4) Jaundice which you think might be caused by a worm in the bile duct (15.6).

Relative indications are:
(1) Toxaemia out of proportion to the severity of obstruction.
(2) Persistence of a worm mass at the same site, or its fixity.
(3) Rectal bleeding especially associated with abdominal pain.
(4) Increasing bowel distension or increasing evidence of free intra-peritoneal fluid.

PREPARATION. Correct any dehydration present. Insert a nasogastric tube.

INCISION. Make a transverse or midline incision and inspect the bowel.

If the mass of worms has thinned, devitalized, or eroded the bowel, or it is intussuscepted or twisted, resect it.

Decompress the remaining bowel and milk out all the worms you can feel. Instil piperazine intraluminally along the whole length of bowel and perform an end-to-end anastomosis. Try to cover your enterotomy closure or anastomosis with omentum in order to prevent any remaining live *ascaris* worms migrating through.
CAUTION! If you have difficulty, *don't be tempted to perform a bypass operation above the level of the worms*.

If there is severe contamination, leave the skin open (11.8).

If the bowel is healthy and you find a ball of worms blocking it, try, if possible, to break up the ball and milk the worms avoiding tearing the serosa, through to the caecum, where they will be safely expelled. If they are in the terminal ileum, this should be easy. If they are more proximal, *don't try to milk them up into the stomach*: they may then climb the oesophagus and be aspirated into the bronchus.

(g) Post-operative 'de-worming'
Don't 'de-worm' the child until 48-72h after all signs of obstruction have gone, and there are no palpable masses of worms. Then use a single dose of piperazine citrate 4g, which will paralyze the worms so that they pass rectally. Or, use mebendazole 100mg bd for 3 days.

(h) Difficulties with *ascaris* obstruction
If you cannot milk the worms downwards, and the wall of the bowel is healthy, try injecting 15-30ml of gastrografin into the lumen of the bowel proximally: this might break up the mass satisfactorily. Otherwise, isolate the mass carefully with abdominal packs. Resect the affected portion of bowel; then try to remove all the remaining worms in the bowel by milking them down through the open bowel ends. Most of them will probably be in the upper small bowel. If you can remove all of them, there will be no chance of them working their way through a suture line later. Nonetheless, make a careful anastomosis (11.3).

If you have difficulty milking all the worms out of the retroperitoneal duodenum or the rest of the bowel, construct an enterostomy (11.6). When bowel function has re-commenced, instil gastrografin into the bowel lumen. When you are satisfied that all the worms have been expelled, close the stoma. If you are unable to construct an enterostomy, leave a nasogastric tube in place till signs of obstruction have gone and then use piperazine as above; beware of worms migrating proximally and down into the bronchus! Check the airway by a laryngoscopy at the time of extubation to see if there are any worms present.

CAUTION! Don't be tempted to make a bowel anastomosis if many worms are still present in the bowel.

If you find a mass or fistula associated with worms, an *ascaris* has perforated through the bowel; often you won't be able to find the hole, and the worms will have caused an infected inflammatory mass. The worms may still be alive: remove them and drain the abscess. This is preferable to doing a resection.

If a worm has migrated into the biliary tree, try to manipulate it out. You won't often be able to do this and may have to explore the common bile duct (15.6).

N.B. Anisakis worms arrive in the human intestinal tract through the eating of raw fish. They are dead once they reach the small bowel. They can cause obstruction like the *ascaris* worm, but once a mass of them causes obstruction, unless you can flush them out with gastrografin, remove them *via* an enterotomy.

12.6 Obstruction by bands and adhesions

Bands and adhesions sometimes form in the peritoneal cavity and obstruct the bowel from outside. They are the result of some focus of inflammation being slowly converted into fibrous tissue, and can follow:
(1) A previous abdominal operation, which may be followed by obstruction soon afterwards (12.16), or later, as described below. You can reduce the probability of this happening by not using powder in surgical gloves, handling tissues gently, and pulling the omentum down over the bowel, and particularly the site of an anastomosis. This will reduce the chances of the bowel sticking to the abdominal wall. Close the abdomen after a laparotomy leaving out the peritoneal layer, using non-absorbable sutures (11.8).
(2) Abdominal sepsis of any kind, especially local peritonitis from PID (23.1), or an appendix abscess. In communities where there is much PID, obstruction due to adhesions is common, and is apt to recur, so that a woman who has had one attack is likely to have another.
(3) Abdominal tuberculosis (16.3) or Crohn's disease which mimics it precisely but is rare.
(4) A congenital anomaly: such bands are unusual and present in early childhood.

If a loop of bowel has stuck to the parietal peritoneum at the site of an old scar, you can usually free it without too much difficulty, but even this can be dangerous because you can easily damage it.

If PID has caused massive adhesions that have resulted in loops of bowel firmly stuck in the pelvis, releasing them may be very difficult. As you will soon learn, freeing them is an art.

Obstruction due to adhesions is less likely to strangulate than some other kinds of obstruction, and is more likely to be incomplete, self-limiting, and recurrent, so you *may* be able to treat it non-operatively, if you are sure of the diagnosis!

(a) Non-operative treatment for adhesions

INDICATIONS
The patient's condition remains good, there are active bowel sounds and minimal tenderness.

Administer 100ml of gastrografin via the nasogastric tube and 6h later, repeat an abdominal radiograph. If the contrast passes into the colon, the obstruction is no longer complete. Continue a conservative approach and ensure there is insignificant nasogastric output, no abdominal tenderness, nor pain, and passage of flatus.

METHOD (12.4)
CAUTION! Abandon the non-operative method if there is no steady improvement. Generally, adhesion obstruction will not resolve if occurring >1yr after the initial abdominal problem.
N.B. Don't operate for pain alone without signs of obstruction: more adhesions will inevitably result. Beware the 'Münchhausen' patient (who shops from doctor to doctor) with many abdominal scars!

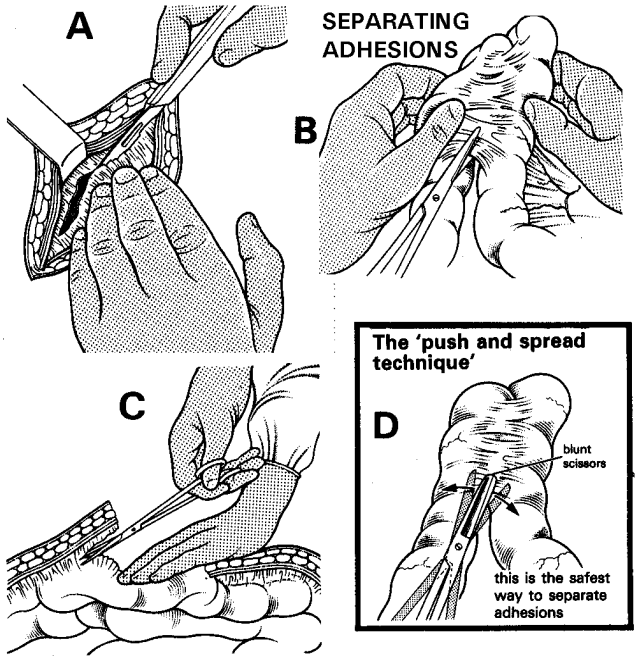


Fig. 12-8 SEPARATING ADHESIONS.
The great danger is that you may perforate the bowel: A, on entering the abdomen. B, on cutting adhesions between 2 loops of bowel. C, when freeing adhesions between the bowel and the abdominal wall, or when closing the abdomen in the presence of obstructed bowel. D, the safest way to separate adhesions is to use the 'push and spread technique' (4-9); preferably use Metzenbaum's or McIndoe's scissors, which are not so blunt as those shown here.

(b) Operative treatment for adhesions

LAPAROTOMY FOR ADHESION OBSTRUCTION (GRADE 3.4)
INDICATIONS
(1) Where non-operative treatment has failed, or there are signs of peritonitis.
(2) Where adhesions formed >1yr beforehand. But remember the risk of re-obstruction is c.20% after a laparotomy to break down adhesions.
(3) Where adhesions may not be the cause of obstruction at all.

INCISION
Open the abdomen with great care. Always dissect under direct vision: so get good exposure, and keep the field dry. Don't use diathermy close to the bowel wall: it too easily causes necrosis.

If there is a previous midline or paramedian incision, excise the scar and re-open the abdomen through it, unless this is difficult. Start above or below it in an area which is free of adhesions. Put a finger into the incision and explore the deep surface of the old scar.

Work slowly with a sharp scalpel and detach the adherent bowel from under it. *Don't make a midline incision parallel to a previous paramedian incision*, because the intervening skin may necrose. If you have to enter the abdomen through the site of multiple adhesions, dissect them away with the utmost care and patience. If the bowel has completely stuck to the abdominal wall, be prepared to excise a piece of the adherent peritoneum when necessary, rather than damage the bowel.

If there is a transverse or oblique incision, re-open this incision.

FREEING THE ADHESIONS
Look for the site of the obstruction, which may be a band with a knuckle or loop of bowel caught under it. This has a 95% chance of being in the small bowel and a 75% chance of being in the ileum. Use the 'push and spread technique' with blunt-tipped Metzenbaum's or McIndoe's scissors (4-9B, 12-8D). Use the outer sides of the blades to spread the tissues. If you work carefully, you can define tissues when they are matted together, by opening up tissue planes, and without injuring anything. You will see what is bowel and what is an adhesion, and will be able to cut in greater safety. Work away at one site and then at another until the adherent loops unravel. Go very gently and *be patient!*

Alternatively, use the 'pinching technique'. Pinch your index finger and thumb together between two loops of adherent bowel. *Don't pull on the bowel*: it may rupture; rather, try to lift it out from underneath. Grip the bowel firmly with moist gauze, and release it periodically, to help you to identify what you are cutting, and to control bleeding. When you have divided a band, you will want to know if the trapped bowel is viable or not: do this using the criteria described (11.3, 11-7). If you can squeeze bowel contents past a kink in the bowel, you can probably leave it safely. Make sure you decompress the bowel proximally (12.4).

If the obstructed part of the bowel is non-viable or strictured, resect this portion and make an end-to-end anastomosis (11.3), remembering to decompress the bowel content before you do so.

Don't necessarily try to divide every adhesion you find. Freeing adhesions can go on indefinitely, and can be dangerous. If there are adhesions between loops which are not causing obstruction, leave them alone.

CAUTION! Work slowly and carefully. Making a hole in the bowel wall increases greatly the post-operative morbidity, especially the risk of a fistula (11.15) and further adhesions.

DIFFICULTIES WITH INTESTINAL ADHESIONS
If bleeding obstructs your view, apply gentle pressure with a warm moist pack. Leave it alone for a few minutes, and dissect somewhere else. Place a small figure-of-8 suture on a bleeding point if bleeding persists: *don't use diathermy!*

If you strip the serosa with some of the muscle layer, leave it. Apply a warm pack, and suture any bleeding points.

If distended loops of bowel obscure your vision, and you cannot release these, it is safer formally to decompress the bowel (12.4) instead of tearing it accidentally.

If you do open the bowel, apply non-crushing bowel clamps either side of the perforation, clean up any spillage and pack around the affected bowel. You can use this opportunity to decompress it of its contents (12.4).

If the opened bowel is still stuck, free it completely before trying to clamp it, otherwise you may cause more damage. Then finish releasing all the adhesions you need to free up. Then close any perforation transversely carefully in two layers.

If the edges of the defect are ragged, trim them neatly, so that you only use full-thickness bowel for closure: make sure that there is no obstruction distal to the point of repair! If there is, a fistula is sure to form.

If you make more than one hole in the bowel, aim to resect the affected portion as one piece if possible and make a formal anastomosis, unless you will have to sacrifice too much bowel.

If there is much soiling, make a temporary enterostomy (11.6) and have a second look 48h later.

If loops of bowel are firmly stuck down in the pelvis, usually in a female from PID or from TB, try carefully to pinch them off the pelvic wall from behind. If you fail, bypass them with an entero-enterostomy (11.4). This is a safe way out of a difficult problem, provided that too long a length of small bowel is not bypassed. Choose an easily accessible loop of bowel proximal to the obstruction, and anastomose it side-to-side with a collapsed loop distally. Some of the absorptive surface of the bowel will be lost, but this will be life-saving. If necessary, another operation can be done later when the condition has improved: you may well then find the adhesions are much less of a problem.

12.7 Intussusception

Telescoping of the bowel into itself takes several forms:
(1) Ileocaecal, ileocolic and ileorectal, which occur in children, especially with *ascaris and coeliac disease*, but also in adults,
(2) Caeco-colic,
(3) Colo-colic,
(4) Ileo-ileal, generally occurring in adults as a result of lymphadenopathy, especially with HIV disease, TB, amoebiasis, schistosomiasis or polypoid tumours.

The relative frequency of these varieties differs considerably from one area to another. It may be the result of intestinal tuberculosis, and occurs more frequently at Islamic festivals in periods of fasting and feasting.

The danger of any intussusception is that the bowel may strangulate: firstly the inner part (*intussusceptum*), but later also the outer part (*intussuscipiens*). However, the signs of peritoneal irritation are initially absent, because the gangrenous inner part is covered at first by the normal outer part. Occasionally, intussusception may occur in a reverse direction.

The child is usually between 6mths and 8yrs. He suddenly cries or screams for a few seconds every 10-30mins. This then stops as unexpectedly as it began. When it restarts, he draws up his knees in spasms of colicky pain. He vomits, and may pass bloody mucus stools. *Don't confuse this with dysentery!* You can usually feel a sausage-shaped abdominal mass in the line of the transverse or descending colon, above and to the left of the umbilicus, with its concavity directed towards the umbilicus. The right lower quadrant may feel rather empty. If the abdomen is much distended, the mass is not so easy to feel. Rarely, it is hidden under the right costal margin, or is in the pelvis, where you may be able to feel it bimanually. Sometimes, the telescoping bowel presents at the anus, or you may feel it rectally, and see blood and mucus on your finger afterwards.

If you notice a mass at the anus, be careful to distinguish an intussusception from a rectal prolapse (26.8). Occasionally, a small intussusception reduces itself.

In an adult, you rarely make the diagnosis pre-operatively; any type of intussusception is found: the colo-colic type will produce signs of *large* bowel obstruction, whilst the ileocolic or ileo-ileal types signs of *small* bowel obstruction.

N.B. Beware of confusing intussusception with dysentery!

COLIC, ABDOMINAL MASS, AND DIARRHOEA? THINK OF INTUSSUSCEPTION!

(a) Abdominal radiographs. You will see the ordinary signs of any small or large bowel obstruction: a dilated bowel with fluid levels. Very rarely will you see any specific features. A barium contrast enema is rarely needed to make the diagnosis.

(b) Ultrasound will demonstrate a typically crescent-shaped mass (pseudo-kidney) in the distended bowel lumen.

(c) Non-operative reduction using air

If, and only if, the history is <24h, and there is no sign of tenderness or gross abdominal distension, and no free gas seen on a radiograph, you can try to reduce an ileocaecal or ileocolic intussusception by an air enema.

Resuscitate the child with IV fluids, and insert a nasogastric tube, leaving its end draining freely into a kidney dish below the level of the trunk. Use ketamine anaesthesia. Insert a Ch20 or 22 Foley catheter into the rectum and inflate its balloon fully within the rectum. Strap the buttocks together.

Fig. 12-9 AIR ENEMA REDUCTION OF INTUSSUSCEPTION.
A, Foley catheter inserted into the rectum. B, mechanical (aneroid) sphygmomanometer bulb and gauge attached. C, nasogastric tube draining freely into a kidney dish (placed below the level of the trunk).

Palpate the abdomen to locate the intussusception mass, or locate it by ultrasound (38.2). Attach a mechanical sphygmomanometer to the end of the Foley catheter and insufflate air into the rectum up to a maximum pressure of 120mm Hg. Follow the passage of air proximally in the bowel by palpation or ultrasound. When this is complete, confirm disappearance of the mass and continuous free flow of air through the nasogastric tube into the kidney dish. Deflate the balloon of the Foley catheter and remove it; feel that the abdomen is soft. You should notice some air escaping via the anus.

N.B. Don't try to reduce it with hydrostatic pressure using a saline or gastrografin enema unless you have an experienced radiologist available; never use barium.

If the mass remains, or there is no continuous free flow of air in the nasogastric tube, you can try again.

If the abdomen suddenly swells, or no air escapes from the anus, because of passage of air into the peritoneal cavity, administer IV gentamicin and metronidazole, and perform an immediate laparotomy.

(d) Operative reduction

LAPAROTOMY FOR INTUSSUSCEPTION (GRADE 3.4)

INDICATIONS. Any intussusception >24h old, which does not spontaneously resolve, or which cannot be reduced by an air enema, needs a laparotomy.

INCISION. Make a transverse supra-umbilical incision in a child (or a midline incision in an adult), and feel for the mass. Retract the edges of the wound and try to lift out the mass. Look at it to see which way the intussusception goes: backwards or forwards.

METHOD

If the outer layer of the intussusception looks viable, try to reduce it by manipulation. If the intussusception has not gone beyond the splenic flexure, manual reduction should not be too difficult. But if it has reached the sigmoid colon, or if it has been present >24h, it is likely to be non-viable.

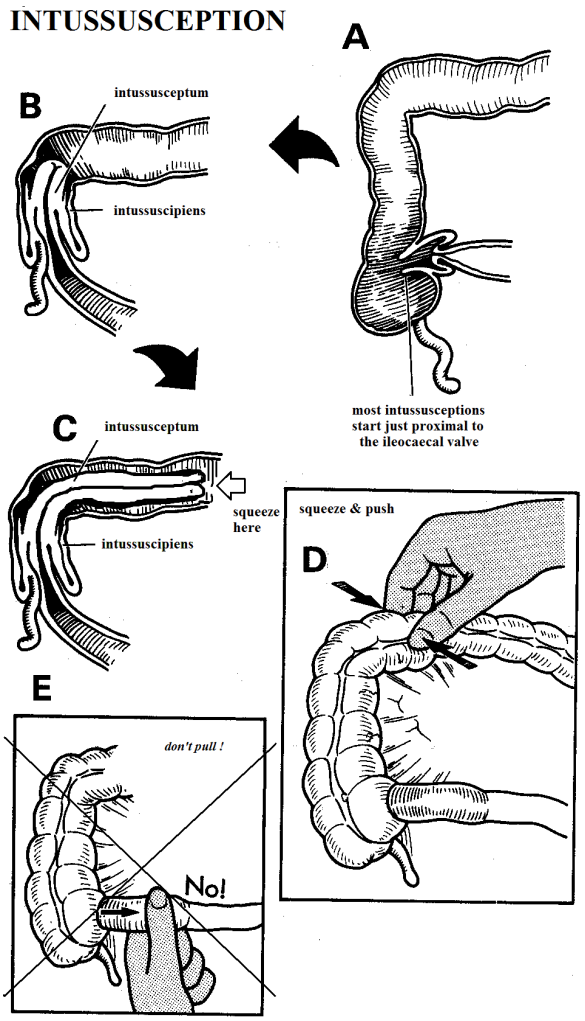


Fig. 12-10 INTUSSUSCEPTION.
A-C, stages in the development of the common ileocolic intussusception in children. D, squeeze the colon that contains the leading edge of the intussusception. E, *don't try to reduce an intussusception by pulling*. *Partly after Ravitch MM, Paediatric Surgery Yearbook Medical 1979 Fig 93-3 with kind permission.*

Using a thick, moist gauze swab between the thumb and index finger of your hand, apply *gentle* pressure to the part of the bowel which contains the leading edge of the intussusception. Reduce it from its apex proximally by pushing. Use the gauze to transmit the pressure to as wide an area of the bowel as you can. Squeeze it gently, so as to make the mass go proximally. Be patient, and change the position of your squeezing hand as necessary. The intussusception will usually reduce itself quickly.

Manual reduction will be most difficult near the end, and the seromuscular layers of the bowel usually split. Persist up to a point. Abandon reduction if:

(1) splitting becomes deep.
(2) you cannot reduce the intussusception any further.
(3) you see a purplish or necrotic area of bowel emerging.

If you split the serous and muscular coats of the last few centimetres of the bowel as you reduce it, don't worry. This usually happens. Provided the mucosa is intact and the bowel is not gangrenous, it will heal.

An area of residual thickened bowel is common and *not* an indication for resection. *Don't perform an appendicectomy*, unless it is inevitably included in the bowel resection.

CAUTION!

(1) Complete all the reduction by squeezing.
(2) *Don't pull the proximal end.*
(3) Reduce the last dimple, or the intussusception may recur.
(4) Make sure the apex is viable, because this is the part which is most likely to become gangrenous.

You will often need to mobilize the ascending colon: stand on the left side and ask an assistant to retract the right side of the wound, so as to expose the caecum and ascending colon. Use a pair of long blunt-tipped dissecting scissors to incise the peritoneal layer 2cm lateral to the ascending colon. Free the colon using the 'push and spread technique' (4-9). Put a moist pack over the colon and draw it towards you, so as to stretch the peritoneum in the right paracolic gutter. As you incise the peritoneum, draw the entire colon medially, from the caecum to the hepatic flexure. Use a 'swab on a stick' to push away any structure which sticks to its posterior surface, especially the duodenum and the ureter, which runs downwards about 5cm medially to the colon, and which you should identify and preserve.

If, after manual reduction, any part of the terminal ileum, caecum, or colon is not viable, resect it and exteriorize the bowel or make an anastomosis. The danger is that death from peritonitis may ensue if you fail to remove all non-viable bowel.

If there is a gangrenous *intussusceptum* protruding from the anus, tie it off tightly and amputate it before opening the abdomen. You will then be able to reduce the remaining intussuscepted bowel easily from inside, and perform a resection of the affected portion with the ligature on.

Most cases in children will involve the ileum. It is rare that the intussusception only affects small bowel. *Don't resect terminal ileum and leave an anastomosis within 5cm of the caecum.*

Commonly you will need to perform an:

(1) ILEOCAECAL RESECTION (GRADE 3.4)

As you lift the caecum and ascending colon medially, you will see the ileocolic vessels which supply them. Hold up the colon and try to see them against the light. Make windows in the peritoneum on the medial side of the colon, and clamp the branches of these vessels, one by one, 3cm medial to the wall of the colon. Insert two haemostats through each window and cut between them, leaving a cuff of tissue distal to the proximal haemostat. Then tie the vessels held in each haemostat with 2/0 or 3/0 suture, depending on the size of the child. Tie them twice on the proximal side for safety.

If you cannot find the blood vessels because strangulation has altered the anatomy, lift up the colon and apply haemostats to the mesentery close to the wall of the colon. Cut between them and the colon, until it is completely free.

Apply haemostats to the mesentery of the ileum 2cm from the bowel, and cut between them until you reach healthy bowel supplied by a visibly pulsating vessel. Raise the greater omentum towards the head, and use scissors to separate the filmy adhesions between it and the hepatic flexure.

Mobilize the hepatic flexure under direct vision. Cut peritoneum only and draw the flexure downwards and medially. Free the colon from the duodenum with a 'swab on a stick'.

You should now be able to lift the strangulated bowel out of the wound, free of all its peritoneal, mesenteric, and vascular attachments. As you lift it up, make sure that there is healthy bowel above skin level at both ends. Occasionally, you will have to mobilize the descending colon and splenic flexure.

Do this in the same way as for the opposite side, but take especial care not to damage the spleen, pancreas or stomach when you free the splenic attachments of the colon, which may be quite high under the rib cage.

Place non-crushing clamps across the bowel 2cm proximal and distal to the non-viable segment, and crushing clamps next to the ends of the non-viable segment of bowel; this will leave healthy portions of bowel between the crushing and non-crushing clamps. By drawing a knife along the crushing bowel clamps (on the side where the non-crushing clamps are), amputate the non-viable bowel. Decompress the bowel contents from the proximal end by suction or by drainage into a bowl after packing away the abdomen.

You now have the choice of either exteriorizing the bowel or doing an anastomosis.

(2) EXTERIORIZATION is a messy but life-saving procedure. By doing this, you may avoid contaminating the peritoneal cavity and improve the chances of survival. When you have done this, the patient will find himself with a temporary ileostomy and colostomy, but you will have saved his life. You will however have to replace the quantities of fluid lost from the stoma, and try later to get this closed.

Examine the proximal and distal ends of the strangulated bowel to find parts which you are sure are healthy. Protect the area with carefully applied towels. Make the proximal ileostomy and distal colostomy using a separate incision for the bowel and thread them through (11.6), next to each other as a type of ‘double-barrelled’ stoma (11-12D).

CAUTION!
(i) Check again that viable bowel extends 2cm above the skin.
(ii) Make sure that there is no tension on the ileum or colon inside the abdomen.

Manage the ileostomy by fitting a standard drainable ileostomy or makeshift (11-15) bag. Protect the skin with zinc oxide cream, barrier cream, or karya gum powder. Use codeine phosphate orally to slow down peristalsis, so that a semisolid stool forms. If the effluent is copious and very liquid, nurse the patient in a prone position with the hips and chest supported on several pillows so as to allow the contents of the ileum to discharge by gravity (11-11). Replace the fluid and electrolyte losses orally.

(3) ANASTOMOSIS may be technically more demanding, but will save the child the fearsome problems of fluid and electrolyte loss, and re-operation. Make a careful end-to-end anastomosis preferably with one layer of long-lasting absorbable sutures for a small child (11.3, 11-7), taking care not to twist the mesentery of the bowel. Close the mesenteric defect with a continuous suture.

If a distal colo-colic intussusception requires resection, perform a Hartmann’s operation (12.9).

12.8 Small bowel volvulus

This is seen at all ages, particularly in young men, often after a heavy meal after a prolonged period of fasting. The small bowel spontaneously rotates on its mesentery, or on a band 5-10cm from the ileocaecal valve, which tethers it to the posterior abdominal wall. As it rotates, it traps large volumes of blood and fluid. Most of the small bowel may rotate, apart from its top and bottom ends, or only a smaller part. Occasionally it is due to a congenital malrotation. Sometimes, an adhesion to a loop of small bowel starts the twist, or it occurs as a result of a mass of *ascaris* or *anisakis* worms impacted in the terminal ileum, or the patient may have a primary sigmoid volvulus, and loops of the small bowel may twist around this (12-14), producing an ileosigmoid knot (compound sigmoid volvulus).

Volvulus of the small bowel is a sudden deadly illness in which the bowel rapidly becomes ischaemic. As the mesenteric vessels occlude, the bowel strangulates and there is sudden severe diffuse abdominal pain. A typical history is of sudden abdominal colic, distension and vomiting, coming on after a large evening meal. Early on, the patient does not look particularly ill although he may have a tachycardia: his abdomen may be fairly relaxed and not particularly tender at this stage. You may feel an ill-defined mass, but high-pitched bowel sounds and radiographs showing a few distended loops of small bowel with a fluid level may be the only signs of a dangerous volvulus. A notable feature is the speed with which the abdomen distends. The pain persists and is followed by signs of peritonitis.

In theory, treatment is easy: untwist the bowel. One of your difficulties will be to make the diagnosis, when all you see at laparotomy are distended loops of small bowel.

Manipulating distended bowel is dangerous, whether or not it is strangulated. If a loop ruptures, survival is in doubt because of septic flooding of the peritoneal cavity that results. There is about a 30% chance of death when bowel is gangrenous but in a survivor, volvulus rarely recurs.

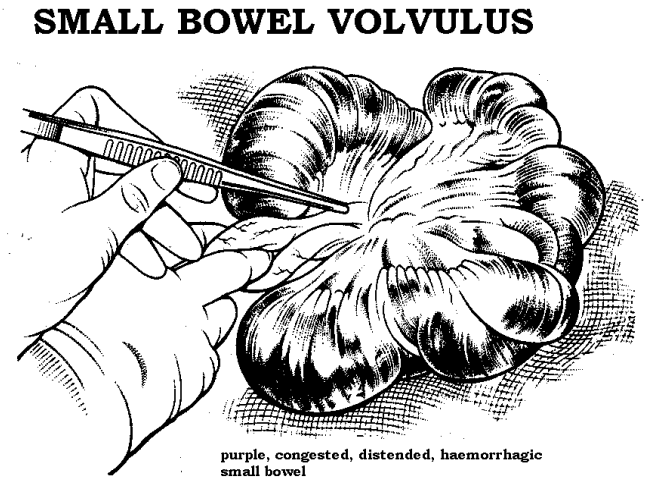


Fig. 12-11 VOLVULUS OF THE SMALL BOWEL is a sudden deadly illness in which the symptoms of obstruction progress rapidly to those of strangulation. *Kindly contributed by Gerald Hankins.*

(a) Abdominal radiographs early on show a few loops of distended small bowel with one or two fluid levels on erect films; later you will see the regular horizontal step-ladder pattern, and many fluid levels, although by this time the patient is usually moribund.

CAUTION! When a strangulated closed loop is distended with blood, there may be no fluid levels, so that the radiographs appear normal.

(b) Operative intervention

LAPAROTOMY FOR SMALL BOWEL VOLVULUS (GRADE 3.4)

Make a midline incision. You will find purple, congested, haemorrhagic, distended small bowel full of food and fluid. A collapsed caecum shows that the obstruction is in the small bowel.

Try to reach the base of the mesentery. Approach this by first putting your hand down into the pelvis, and then up along the posterior border of the abdominal wall. Often, the whole of the small bowel is twisted, except the first few centimetres of the jejunum and the terminal ileum. Rotate the whole mass until the volvulus is undone. If you find a band near the ileocaecal valve, dividing it may help you to reduce the volvulus. *Take great care doing this so you don't inadvertently puncture the bowel and spill large volumes of bowel contents!*

Deliver the bowel, untwist it, and cover it with warm moist abdominal swabs. Assess its viability.

If you have difficulty untwisting the bowel, decompress it first. Introduce the suction nozzle into a distended loop through a purse-string suture, and decompress it proximally and distally. Do this routinely if the bowel is obviously non-viable.

If the bowel is viable, decompress it by massaging the fluid proximally (12.4).

If you are not sure if the bowel is viable or not, assess it (11-6). Wait for at least 10mins before you make a decision.

If the bowel is not viable, and the gangrenous section ends well proximal to the ileocaecal valve, resect it and perform an end-to-end ileal anastomosis after decompressing the bowel (11.3). If the bowel is gangrenous down to the caecum, perform an ileocaecal resection (12.7).

If there are *ascaris* worms, resect any non-viable bowel, decompress the remainder and remove all palpable worms, irrigate the bowel with piperazine, and perform a secure end-to-end anastomosis, covered by omentum (12.5).

CAUTION! Be sure to select healthy bowel for the anastomosis, with obviously visible pulsations in the vessels that supply it: a serious and sometimes fatal complication is a leak due to necrosis of the bowel at the site of the anastomosis.

Continue nasogastric suction and IV fluids post-operatively till flatus is passed. You may need to transfuse blood.

12.9 Sigmoid volvulus

A long sigmoid colon distended with the gas of a high-fibre diet is more liable to twist on its mesentery. This is the commonest cause of large bowel obstruction in much of the world, particularly in the African continent, and is sufficiently characteristic to allow you to diagnose it easily. Occasionally, it resolves spontaneously. If sigmoid volvulus persists, ischaemia results and the colon becomes gangrenous and may perforate. Sigmoid volvulus is however less dangerous and more common than small bowel volvulus (12.8).

In a patient, normally male, with his first episode of volvulus, the colon may not be thickened, and the blood vessels thin. However, usually there have been many undiagnosed episodes of volvulus; the long mesentery is scarred, its vessels are enlarged, and the bowel wall is thickened as in Chagas disease (12.13). Occasionally, in younger adults, the small bowel is pulled round with the sigmoid and an ileosigmoid knot (compound sigmoid volvulus) (12-14) results.

Classically there is initially difficulty passing flatus. This is followed over a few days by increasing gaseous abdominal distension, tympanitic (like a drum), but without much pain or tenderness. There may be so great distension that breathing is severely impaired.

Vomiting is unusual, except when the colon presses severely on the stomach. The general condition is usually good: drinking is possible and dehydration not severe. The contrast between the satisfactory general state and the huge abdomen is striking.

If the twist in the sigmoid is >180º, symptoms and signs will be more acute, with severe colicky pain, perhaps some bloody loose stool, abdominal distension, prostration and circulatory collapse. Likewise a patient whose volvulus persists will show similar signs of peritonitis.

(a) Abdominal radiographs are usually immediately diagnostic: in the erect film, there is a huge appearance of gas like an inverted 'U' ('the coffee bean sign') reaching from the pelvis to the upper abdomen, inclining right or left, often with smaller fluid levels proximal to the loop (12-12A). A supine film may show three dense curved lines converging on the left sacroiliac joint. The middle line is the most constant, and is caused by two walls of the distended loop lying pressed together (12-12B).

(b) Differential diagnosis
(1) Carcinomatous obstruction of the left colon or rectum.
(2) Caecal volvulus.
(3) Megacolon.
(4) Neglected short segment Hirschsprung's disease.
(5) Pseudo-obstruction.

Suggesting carcinoma of the colorectum: a change from a normal bowel habit to constipation over a much longer period; a smoothly distended abdomen without obvious coils of colon; palpation of a rectal tumour; radiographs showing caecal distension, and not the characteristic signs of sigmoid volvulus.

Suggesting a caecal volvulus: radiographs show a huge appearance of gas centrally in the abdomen unlike the appearances in 12-12A,B.

Suggesting megacolon: long history of constipation with no acute signs.

Suggesting pseudo-obstruction: gas in the rectum.

MANAGEMENT
If the bowel is viable, you should be able to relieve obstruction by decompression with a flatus tube passed through a sigmoidoscope (12.4). This will relieve the immediate symptoms, but it is not sufficient treatment, because the volvulus has a >30% chance of recurring. After a 2nd attack, it has a 60% chance of doing so. If you succeed in relieving the volvulus, prepare for a definitive procedure. It will recur if the interval is too long: so give adequate warning. If you proceed to sigmoid colectomy, recommence oral fluids and provide bowel preparation with magnesium sulphate (or other laxatives) and rectal washouts on the 3rd and 4th days. Start gentamicin with metronidazole perioperatively. On the 5th day perform a laparotomy to resect the sigmoid colon. If the colon is nicely deflated, and you are experienced, you may be able to do this neatly through a left iliac fossa incision.

N.B. The main danger in using a sigmoidoscope is that you may perforate a gangrenous loop of bowel and cause catastrophic spillage of faecal material into the peritoneal cavity. This will depend on the acuteness of onset and delay in presentation.

(c) Operative intervention
EMERGENCY LAPAROTOMY FOR SIGMOID VOLVULUS (GRADE 3.4)

INDICATIONS
(1) Failure to reduce the volvulus with a flatus tube.
(2) The presence of bloody diarrhoea or infracted mucosa on sigmoidoscopy.
(3) Perforation of the colon at sigmoidoscopy or by passage of a flatus tube.
(4) Signs of peritonitis.
(5) (Elective procedure after successful deflation)

RESUSCITATION. Resuscitate vigorously. There may be large volumes of fluid lost into the sigmoid. If there is a compound volvulus, 2 or even 4 units of blood may be needed.

If respiration is very laboured because of enormous abdominal distension, you can buy time by puncturing the colon percutaneously and introducing a Foley catheter into the colon to decompress it, provided you have already started IV fluid resuscitation.

METHOD. Use the Lloyd-Davies stirrups. Catheterize the bladder. Add gentamicin 80mg and metronidazole 500mg IV when you start the operation if they have not been given already. Make a generous midline incision. You will see an enormously distended loop of colon. Gently lift it out of the abdomen from below.

CAUTION! Open the tense distended abdomen with the greatest care: it is easy to perforate the bloated sigmoid! Pack the sigmoid off well and decompress it (12-6B).

If the sigmoid loop is of normal colour, gently introduce the rectal tube into it. Ask your (suitably clothed) assistant to get under the drapes and pass it further up the rectum. As he does this, guide it manually past the twist. The loop will deflate and allow you to untwist it. Get your assistant to suture the tube to the anus so that it acts as an internal splint.
Alternatively, find the pedicle and see which way it is twisted. Using both hands, try to untwist it. This will be safe provided it is not gangrenous. The loop seldom rotates by more than 360º. If you succeed in untwisting it, flatus will discharge through the rectal tube. If you cannot find the pedicle and don't know which way it is twisted, twist it first one way and then the other.

What you should do next depends on your experience:
(1) If you are not at all experienced, deflation alone without resection will be wiser. The problem of the patient returning to have an interval resection is, however, a very real one. You can reduce this risk (but not abolish it) by fashioning a temporary tube colostomy to fix the colon to the abdominal wall.

(2) If an anastomosis is out of the question, you may perform a mesosigmoidoplasty. *Fixing the colon to the lateral abdominal wall by means of a colopexy results in too many cases of recurrence to be worthwhile.*
(3) If you are experienced, resect the sigmoid colon loop and perform an end-to-end anastomosis.

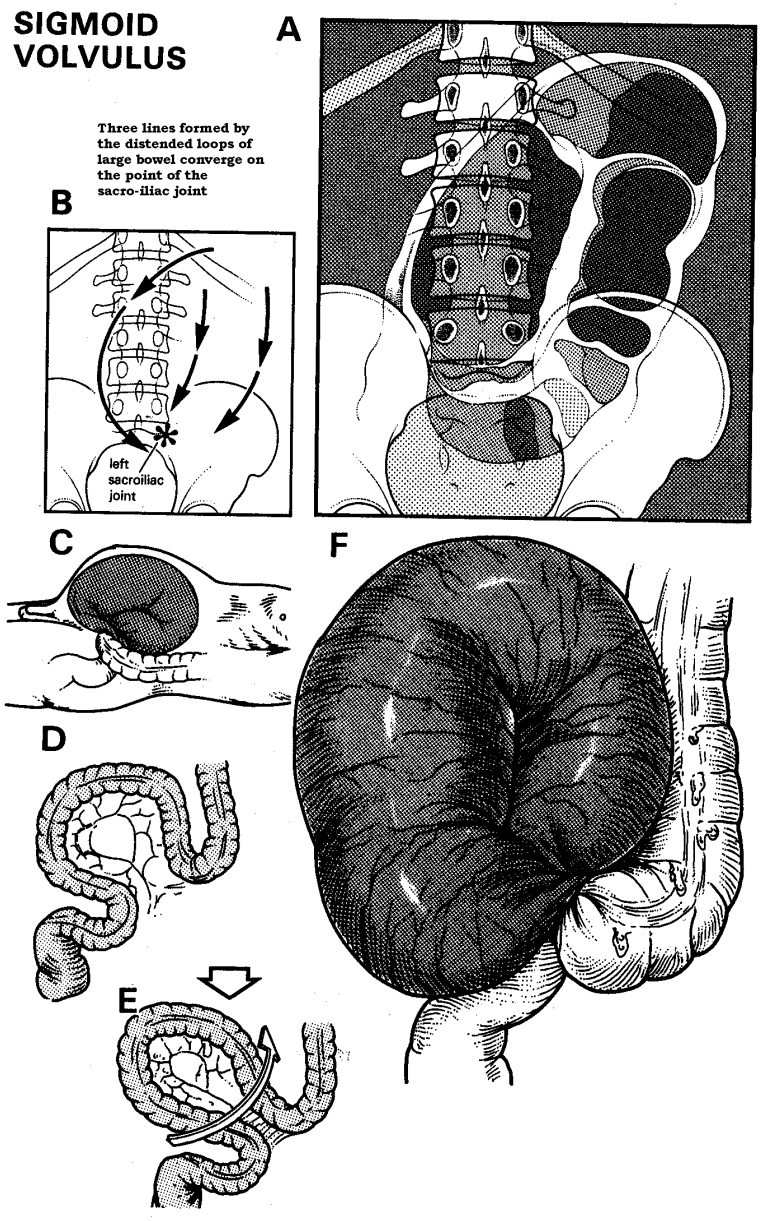


Fig. 12-12 SIGMOID VOLVULUS.
A, supine radiograph showing a huge distended inverted loop of sigmoid. B, diagrammatic version of A to show three lines formed by the walls of the sigmoid converging on the left sacroiliac joint. C, abdominal distension caused by sigmoid volvulus. D-F, show the mechanism of sigmoid volvulus.
Partly adapted from drawings by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

If you are not sure if the colon is viable or not, proceed with resection if you can. *Don't leave non-viable colon in the abdomen!*

In all these operations you will have to mobilize some of the descending colon by incising the peritoneum 2cm lateral to it, followed by blunt dissection.

If the loop is obviously gangrenous, assume that the area of the twist is likely to be even more unhealthy. Pack it off (it may pop like a balloon). Clamp the bowel proximal and distal to the twist. Very cautiously decompress it (12-6B), making sure you drain bowel content outside the abdomen.

Don't try to untwist the bowel. You will have to resect the colon. You need to decide either to perform a Hartmann's procedure (12-13C), or alternatively perform an anastomosis if you are very experienced, the patient is in good condition and there is minimal soiling of the peritoneal cavity. If you fear that the anastomosis may leak (which is still a possibility in the presence of gross soiling, even if your anastomosis is immaculate), it is best to fashion a proximal defunctioning loop colostomy (11.6) to minimize complications should a leak occur.

Exteriorizing the whole segment of dubious or necrotic bowel is difficult and rarely possible.

(1) MESOSIGMOIDOPLASTY (GRADE 3.3)
This may seem a lesser procedure, but runs the risk of recurrence, and *is not easy to perform*. It is only indicated where an anastomosis is out of the question. It aims to shorten the long mesosigmoid.

Lift up the distended sigmoid loop, and divide its mesentery on both sides preserving the most peripheral and most central vascular arcades. Then close the longitudinal defect, thus created, transversely on each side of the mesentery with a continuous suture, taking care only to pick up the peritoneal surface (12-13E).

(2) HARTMANN'S OPERATION (GRADE 3.4)
Consider carefully if you cannot *safely* do the easier operation of sigmoid colectomy. Reversal of a Hartmann's operation (12.10) is difficult and may prove impossible, so your patient may be left with a permanent colostomy. An anastomotic leak complicating reversal of a Hartmann's operation will mean re-establishing the colostomy, almost certainly permanently.
Mobilize enough of the descending colon to bring healthy bowel out to the surface as a colostomy. You will have to go higher than you think initially: *don't allow any tension on the bowel*. Ask your assistant to retract the left side of the abdominal wall, so as to expose the junction of the descending and sigmoid colon. Incise the peritoneum in the left paracolic gutter (12-13A), and carefully displace the mobilized colon medially and upwards. Draw the whole loop of sigmoid colon out of the abdomen, so that you can transilluminate the mesocolon.
CAUTION! Remember that the inferior mesenteric vessels and ureter may take a looping course near the sigmoid colon (12-13A).

Shine a laterally placed light behind the bowel to reveal the mesenteric vessels and divide them well out towards the bowel wall, so that you avoid injuring the left ureter or the superior rectal vessels.

Unless you have special small bowel clamps which can pass through the opening, you are liable to spill bowel content at this stage; it is best to tie a strong ligature round the end of the bowel (tight enough to prevent spillage of faeces, but not too tight to cause ischaemia).

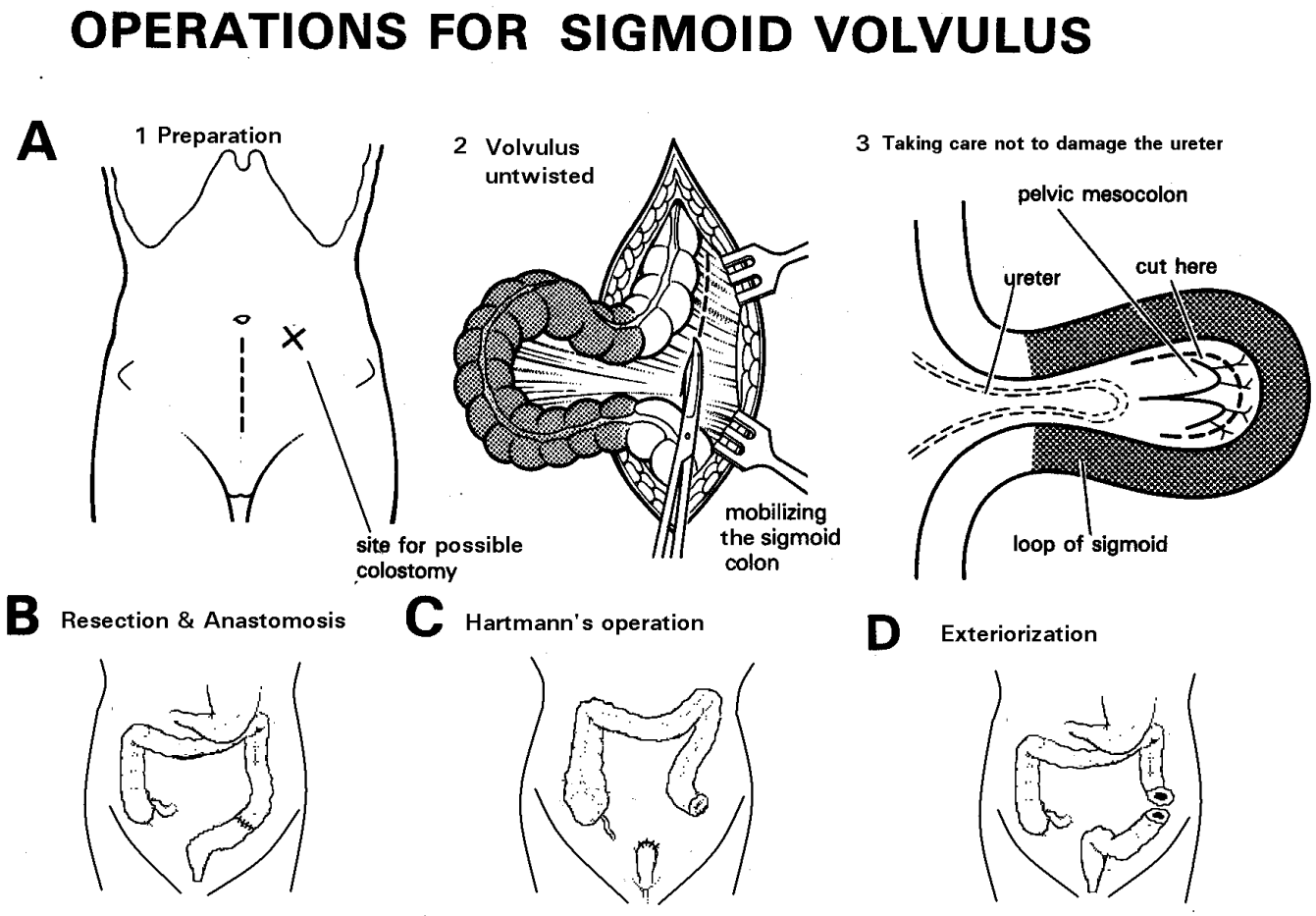


Fig. 12-13 OPERATIONS FOR SIGMOID VOLVULUS.
A, preparation: (1) site for a pelvic colostomy through a small opening ⅓ way between the umbilicus and the left anterior superior iliac spine. (2) mobilize the descending colon. (3) the ureter is usually on the posterior abdominal wall, but it may run close to the sigmoid, so avoid cutting it by dividing the sigmoid mesocolon close to the bowel. B, resection and anastomosis. C, Hartmann's operation. D, exteriorization of bowel (*rarely possible and only if there is enough healthy bowel distally to reach skin level*). E, mesosigmoidoplasty (1) divide the mesosigmoid mesentery, leaving the outermost vascular arcade intact, (2) close the mesentery transversely.
E, after Bach O, Rudloff U, Post S, Modification of mesosigmoidoplasty for nongangrenous sigmoid volvulus World J Surg 2003; 27(12):1329-32.

Resect the twisted sigmoid colon between the bowel clamps, making sure there is viable colon at the point of division. Close the rectal stump, starting at one end with a continuous suture of 2/0 long-acting absorbable, and then bury this suture with another continuous non-absorbable suture. Leave one end of this suture long: this will make finding the stump easier, when you come to perform a re-anastomosis.

Mobilize enough of the descending colon to bring healthy bowel to the surface as a terminal colostomy. Make the opening at a point in the left iliac fossa ⅓ from the umbilicus to the antero-superior iliac spine, and draw the end of the descending colon through.

The exteriorized bowel must lie comfortably; if it doesn't, mobilize more of the descending colon.

While your assistant retracts the abdominal wall to the left, close the space between the colostomy and the parietal peritoneum if there will be a significant delay before you can arrange to re-anastomose the bowel, because this is a space into which loops of bowel can herniate and obstruct. Do this with 3 or more interrupted sutures between the parietal peritoneum and the seromuscular layer of the colon.

Have a final look at the colostomy from within, to make sure the bowel is lying nicely. Then wash out the abdomen with warm fluid and close it (11.8).

Fashion the stoma (11.6) and check the width of the colostomy lumen with a finger. Apply a colostomy device.

(3) EXTERIORIZATION (GRADE 3.5)
Proceed as for the Hartmann's operation. Carry the dissection back to the point where the proximal and distal colon are viable. Bring the sigmoid colon outside the abdomen, through a separate incision. Wash out the abdomen with warm fluid and close the main laparotomy incision (11.8).
Place a small crushing clamp just beyond the two ends where the colon is not viable. Apply non-crushing clamps proximally and distally, and divide healthy bowel between the crushing and non-crushing clamps, and remove the gangrenous loop. If possible, make a colostomy (11-14), and suture the everted colon mucosa to the skin.

N.B. In practice, the distal end is usually too short to pull out as a stoma, so you will be forced to perform a Hartmann's operation.

(4) SIGMOID COLECTOMY (GRADE 3.4)
Resect the redundant sigmoid, taking care not to rupture the large mesenteric vessels: if the colon is viable, *be careful not to remove too much bowel distally* because this will make your anastomosis more difficult. Make sure the proximal colon is mobilized well enough to reach the distal colon.
Make a small opening in the redundant sigmoid loop that you will resect after untwisting it, and decompress the bowel proximally by emptying its content into a large bowl: make sure the bowel hangs nicely outside the abdomen so as not to cause spillage, and place a non-crushing clamp distally. Make an end-to-end anastomosis in two layers (11.3). Check for leaks by placing a non-crushing bowel clamp proximal to the anastomosis, and filling the pelvis with water; then blow air or irrigate diluted methylene blue dye through the rectum with a bladder syringe.
Fashioning a defunctioning proximal transverse colostomy does not prevent the column of faeces distal passing the anastomosis, so a proximal descending colostomy is better; the security of your anastomosis, however, is more likely to depend on accurate technique, a properly cleaned bowel and suitable suture material. Long-lasting absorbable sutures are ideal, and it is then probably not absolutely necessary to have a whistle-clean bowel, but cleaning the bowel of faecal content is very worthwhile. If you only have catgut and silk, however, you can try to get the bowel absolutely clean by an on-table washout by passing fluid through from the appendix or caecum, via a Foley catheter, out of the proximal resected end of the colon, attached to wide anaesthetic tubing. This is, however, messy unless you are very careful; *if you still have improperly prepared bowel, you should avoid an anastomosis using catgut*.

(d) Difficulties with sigmoid volvulus
If a loop of ileum is twisted in with the sigmoid colon (ileosigmoid knot, *compound sigmoid volvulus*), you may not be able to untwist both loops. Puncture and deflate the colon, and then clamp it before you unravel the knot. If both loops are gangrenous, resect them *before you try to unravel the knot*. Anastomose the small bowel end-to-end, and deal with the large bowel depending on experience as above.
If the lower limit of the gangrene on the ileum is close to the ileocaecal valve, you may need to resect the ileocaecal segment as for intussusception (12.7).

If you decide to make a stoma, fashion an ileostomy rather than a colostomy distal to a small bowel anastomosis. Rarely, one loop is viable: if it is the small bowel, leave it alone; if it is the sigmoid, perform a resection.

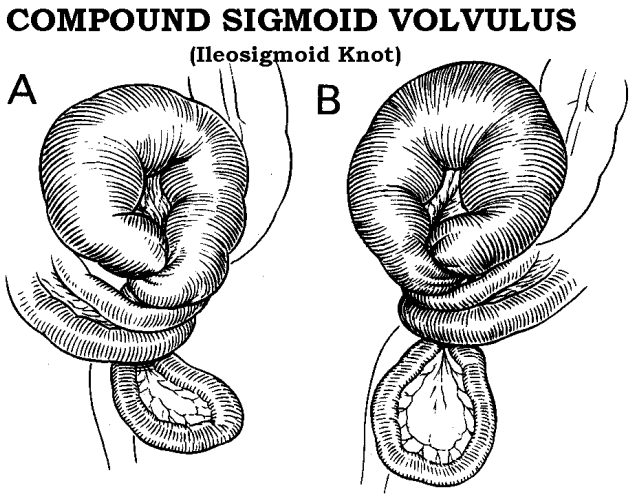


Fig. 12-14 COMPOUND SIGMOID VOLVULUS (ileosigmoid knot) is found where simple sigmoid volvulus is common. A loop of the ileum is twisted with the sigmoid. The twist in the bowel may be left-handed (A) or right-handed (B). If you cannot untwist it, you will have to deflate it and resect it. *Don't try to untwist it if its circulation is impaired*.

12.10 Reversing Hartmann's operation
(GRADE 3.5)

This is one of the more difficult operations described. *Never let the patient or his relatives persuade you to do this operation if you are inexperienced: the penalty is the death of the patient and your reputation!* There may be very dense adhesions in the pelvis making the operation extremely difficult for you and hazardous for the patient. Even if you are confident with fashioning a low anastomosis, this may prevent you from proceeding. So, think very carefully before you promise to reverse a Hartmann's operation. (This procedure exteriorizes the proximal colon as an end-colostomy, closing the rectal stump with the anal canal left open.)

You will have to divide the adhesions, find the rectal stump, mobilize the proximal colon, open the rectal stump, and bring the proximal colon down to meet it, and make a neat leak-proof anastomosis!

TIMING. As soon as your patient has recovered from the operation, is eating and can have bowel preparation; *the classic wait of 6-12wks does not necessarily reduce the adhesions*.

PREPARATION. Administer magnesium sulphate (Epsom salts) 10g orally (or other available laxatives) the evening before the operation, and again early in the morning on the day of operation to clear the bowel. Meanwhile provide plenty of oral fluids to avoid dehydration.

Start metronidazole 400mg tds, and restrict intake to oral fluids only on the evening before operation. On the day before the operation, wash out the proximal loop, and administer an enema up the rectal stump. Cross-match 2 units of blood. Add gentamicin 240mg IV when you start the operation.

METHOD. Use the supine position and put the legs in Lloyd-Davies stirrups. Catheterize the bladder. Tilt the head down to give you better access to the pelvis. Excise the previous wound. Using scissors and gentle blunt dissection, carefully separate any adhesions between the bowel and the abdominal wall, and between loops of bowel.

Pack off the small bowel inside the upper abdomen (a three-bladed abdominal or universal Dennis-Browne retractor is useful for this, 4-5). Infiltrate lignocaine with adrenaline (1:200000) around the colostomy to reduce bleeding. Hook up the bladder to the pubic symphysis with a temporary stay suture.

Start by finding the distal rectal stump: if you fail to do so, you will not have disturbed the proximal colostomy, and can close up, and try again later. Put a long blunt instrument into the rectum through the anus to help identify the stump.

Then start to dissect it out (the non-absorbable suture placed earlier will also help to find it). Dissect across the top and about 5cm down each side (12-15B). Remove the instrument in the rectal stump and cut it across 1cm from its blind end, so a clean bowel lumen becomes visible. *Don't be tempted to leave a swab in the distal rectum*: you may forget it or be unable to retrieve it post-operatively.

Make an elliptical incision around the colostomy; dissect down to the rectus sheath. Control bleeding carefully so you can see where you are. Free the bowel from the anterior and posterior rectus sheaths; a finger inside the stoma will help show you where to dissect without cutting into the bowel lumen (11.6). If there are adhesions to the stoma site, free them from the proximal colon from inside the abdomen; apply a non-crushing clamp across it from inside just adjacent to the abdominal wall, and amputate it.

Mobilize the proximal descending colon by incising its lateral peritoneal reflection, and using a 'swab on a stick' to peel the bowel away from its attachments (12-15A).

Mobilize it sufficiently, so that it reaches the distal stump without tension. This may mean mobilizing the splenic flexure.

Perform an end-to-end anastomosis. Put stay sutures at the left and right edges, holding both ends together. Start the anastomosis at the back (posteriorly) in the middle *instead of the antimesenteric border* with two separate long-acting absorbable or silk sutures, placing the knots outside the bowel wall, and leaving the ends left long (12-15C).

Continue these as a continuous seromuscular Lembert suture and work round on the outside towards the left and right sides in turn. Starting at the same point inside the lumen in the middle posteriorly, insert an all-coats over-and-over inner layer long-acting absorbable suture, working round towards left and right as before (12-15D), and continue as an inverting Connell suture finishing at the front (anteriorly). Then complete the outer layer seromuscular suture to bury the inner layer.

Check the soundness of the anastomosis and the size of the lumen by pinching it between your thumb and finger (11-7Q). You can further check the anastomosis by filling the pelvis with water and blowing air up through the anus with a bladder syringe. There is a leak if you see bubbles in the water!

Remove the stay suture hitching up the bladder. Close the pelvic peritoneum over the anastomosis and close any defect through which a loop of small bowel might prolapse, preventing its fall into the pelvis.

DIFFICULTIES REVERSING HARTMANN'S OPERATION

If you cannot bring the ends of the bowel together easily, mobilize more of the descending colon proximally by incising the lateral peritoneal reflexion further, and raising more of the mesentery. You can always bring the bowel ends together if you mobilize enough mesentery. You may, rarely, need to mobilize the splenic flexure; make sure you fix the colon to the posterior peritoneum to prevent it twisting on an axis from the transverse colon to the rectum.

If the ends of the bowel are different sizes (the proximal end is usually bigger), place the sutures on the wider end further apart from each other, so that you meet at the same place on each lumen.

If the lumen is too narrow, or there is a 'dog ear' at the anastomosis, undo it and start again. *If you leave it, obstruction or a leak are inevitable*.

If there is a small leak, evidenced by bubbles from insufflations of the anus, at the anastomosis, identify the defect carefully, and invaginate it. Re-check if there is still a leak. If so, try again, but if there remains a leak, undo the anastomosis and start again.

If the ends of the bowel bleed, press them firmly for up to 5mins. If there is a bleeding vessel beside the bowel, clip and tie it.

If you cannot get access to the rectal stump to make an anastomosis by hand, extend the incision, make sure you have good abdominal relaxation, the small bowel is packed out of the way, and get another assistant to retract.

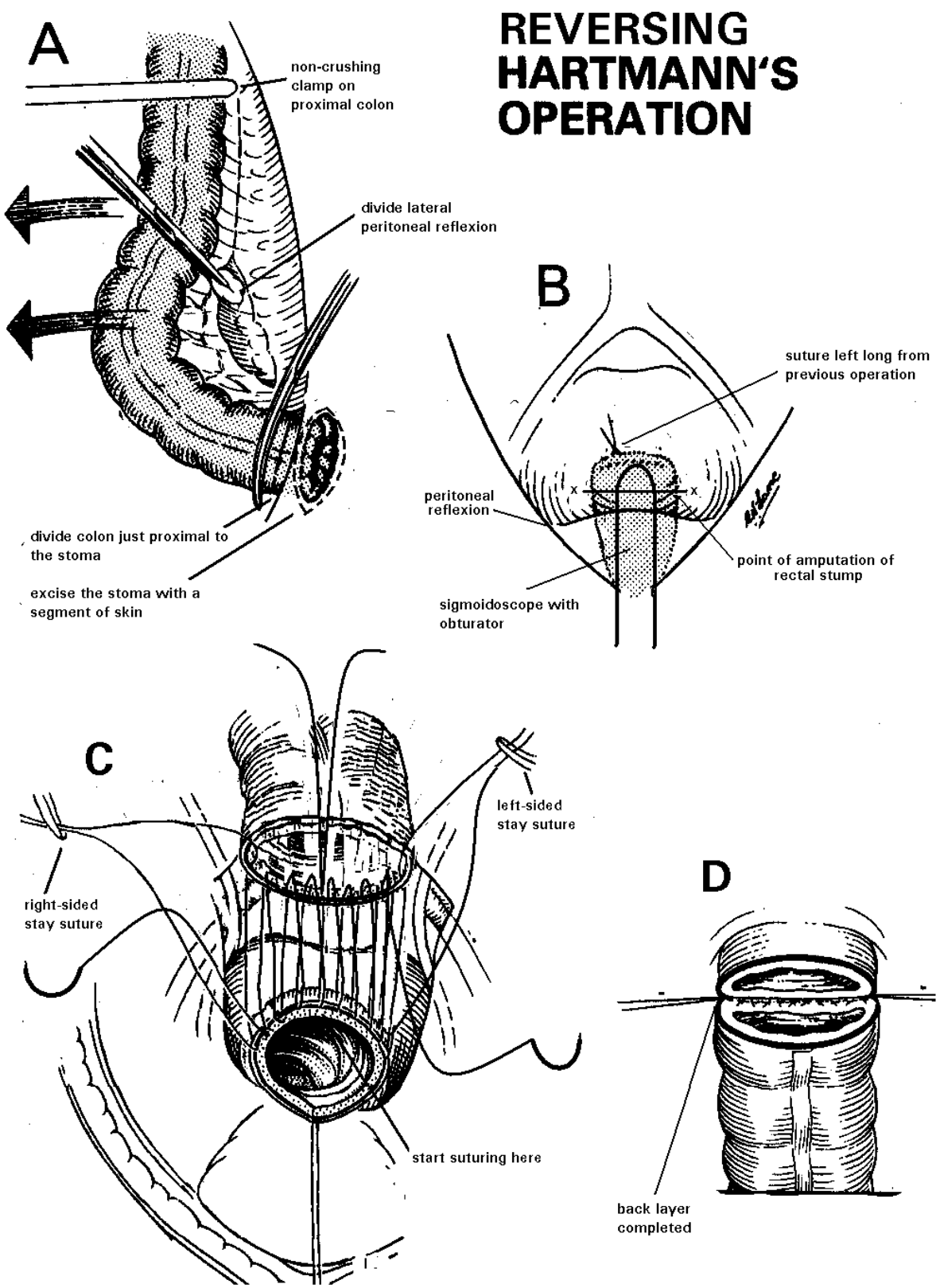


Fig. 12-15 REVERSING HARTMANN'S OPERATION.
The distal bowel is usually deeper in the pelvis, even than shown here. **A, mobilize the colon. B, free the rectal stump and cut off the top of the rectal stump. C, place the seromuscular (Lembert) sutures that will draw the 2 ends of the bowel together. D, pull tight the sutures placed in C. Note that the 2 ends have been left long.**

If you have an ANASTOMOSIS STAPLE GUN (4.10), it is especially useful here: make sure the bowel ends are clear of excess fatty tissue. Insert the instrument through the anus and make it come out lateral to the rectal closure rather than out in the middle of the closed lumen. Make sure that a monofilament purse string holds the rectal end snugly round the shaft of the instrument above the cartridge. Introduce the colonic end over the top of the anvil and secure it tightly below with another purse string. Screw the instrument so that cartridge and anvil come close together and a mark on the instrument appears to show they are close enough. Then release the safety catch and fire the instrument in one clean movement. Open the gun slightly to separate anvil and cartridge, and twist and turn the instrument gently to remove it from the anus. Examine the cartridge: if you find 2 complete doughnuts of bowel, the anastomosis is complete; if not, reinforce it by hand, which should now be possible as the rectum will be drawn up.

12.11 Colorectal carcinoma

Carcinoma of the large bowel is usually a slow-growing adenocarcinoma. It may project into the lumen like a cauliflower, or form a stricture (long or short), or an ulcer (which may not penetrate the bowel for about 18 months). It invades locally, spreads to the regional nodes or the liver (usually late), or through to the peritoneal cavity (late and uncommon). Tumours of the rectum spread upwards, whilst those of the anal canal (26.7) spread to the inguinal nodes.

Colorectal carcinoma is related to a low-fibre and high-fat diet, which is increasingly common in many affluent parts of the world but also in shanty-towns. Another factor is exposure to organochlorine pesticides. Endemic *schistosoma mansoni* predisposes to rectal carcinoma, and multiple familial polyposis likewise.

The patient is usually male, >45yrs, but is occasionally a young adult, who presents with:
(1) Blood and mucus mixed within the stools.
(2) An alteration in bowel habit.
(3) A sense of incomplete defecation (tenesmus).
(4) Colicky abdominal pain (incomplete obstruction).
(5) Intestinal obstruction.
(6) A fixed mass.
(7) An anorectal, recto-vesical or recto-vaginal faecal fistula which appears spontaneously.

You are most likely to meet carcinoma of the large bowel when you operate for obstruction, and have to relieve it. This is why this topic is described here, although you may find a colorectal carcinoma without obstruction. Carcinoma of the rectum usually presents late, because it causes little pain in the early stages. You can feel most rectal carcinomas with your finger – either as a firm raised plaque, or an ulcer with hard rolled edges, leaving blood on your glove afterwards.

Don't mistake it for a fissure or haemorrhoids! You can detect probably c.50% of colon carcinomas with a simple rigid sigmoidoscope.

(a) History and examination
Symptoms have usually been present for several months. Look for:
(1) Signs of loss of weight, anaemia and jaundice.
(2) A primary mass, ascites, a hard craggy liver with metastatic tumour, or a hard umbilical (Sister Joseph's) nodule.
(3) A rectal mass.
Get an idea of the site of the lesion by the symptoms:
Suggesting a lesion in the right colon: anaemia, a mass, caecal pain, weight loss, small bowel obstruction.
Suggesting a lesion in the left colon: colicky abdominal pain, alteration of bowel habit (diarrhoea alternating with constipation), blood mixed with stools, large bowel obstruction.
Suggesting a lesion in the rectum: rectal bleeding, diarrhoea, a feeling of incomplete evacuation.

(b) Assessment
SIGMOIDOSCOPY (GRADE 1.2)
If you feel a rectal mass, examine the lesion under GA in the lithotomy position (26-4B). Feel if the tumour is mobile or fixed. Take a biopsy from its edge: not all rectal strictures are malignant! Pass a sigmoidoscope (26.1) to look for other tumours: *most adenocarcinomas of the colorectum are accessible to the finger or sigmoidoscope!*

(c) Radiographs
If the above investigations are negative and you still suspect a carcinoma, if there is no colonoscope to hand, perform a barium enema. This is not easy, but it can be done in a district hospital. Use barium and air phase contrast (38.1). Avoid a barium enema when there is complete or partial obstruction. Get a chest radiograph to exclude pulmonary metastases.

(d) Ultrasound is very useful to detect liver metastases (38.2b). If there is a palpable mass, it might give details of its nature, size, and involvement of other structures.

(e) Differential diagnoses include:
(1) Other causes of blood in the stools (haemorrhoids, amoebiasis, and dysentery) (26.4).
(2) Other causes of altered bowel habit (bowel infections, poor food supply, upper abdominal malignancy).
(3) Other causes of acute-on-chronic obstruction (sigmoid volvulus, amoebic stricture, Chagas disease).
(4) Other causes of rectosigmoid strictures (amoebiasis, lymphogranuloma venereum (especially in women), schistosomiasis, herbal enemas).

(f) Management depends on how skilled you are and:
(1) Confirmation of malignancy.
(2) Where the tumour is.
(3) Its staging.
(4) If the bowel is obstructed or not.

(g) Preparation. If there is no obstruction, you will be able to perform a planned elective operation. Try to empty the colon first.

Enemas only clear the distal part, so you can use magnesium sulphate (Epsom salts) 10g (or other available laxatives) the evening before operation, and again early in the morning on the day of operation to clear the bowel.

N.B. Evidence now suggests that total bowel preparation may be unhelpful, although an obviously full colon must increase the risk of infection if there is spillage. This spillage will be worse, though, if the bowel content is liquid. It may just be simpler to restrict the diet to yoghurt and foods of low residue 1wk pre-operatively. If you use bowel preparation, you must replace fluid lost by osmosis into the bowel by plenty of oral fluids.

Start metronidazole 400 mg tds, and restrict to oral fluids only on the evening before operation. Cross-match 2 units of blood. Add gentamicin 120mg IV timed 30-60mins before you start the operation.

(h) Operation

LAPAROTOMY FOR COLORECTAL CARCINOMA (GRADE 3.4)

Make a long midline incision, or a long transverse subumbilical incision. Palpate the tumour for staging. It is a firm mass involving the colon; an inflammatory mass may look and feel the same, so keep an open mind! A tumour can be mobile, tethered to surrounding structures, or fixed.

It may be unresectable if it is fixed to the pelvic wall, the abdominal wall, or the bladder. *You will have to assess how readily you can resect the tumour: a more experienced surgeon may manage a more extensive resection, but the prognosis may not necessarily be improved thereby.* If chemotherapy (cisplatin, fluorouracil and folinic acid) is available, it may shrink an unresectable tumour and make it resectable.

It is inoperable if there are palpable masses in the liver, widespread metastatic mesenteric lymphadenopathy, or malignant ascites (or of course metastases elsewhere outside the abdomen).

If the bowel is obstructed (much the most likely occasion on which you will meet this tumour), to relieve the obstruction you can fashion a proximal defunctioning colostomy, or perform a resection and colostomy, or a resection and anastomosis.

CAUTION! The contents of the large bowel are always loaded with bacteria, so when you have to operate in an emergency for obstruction, avoid contaminating the peritoneal cavity, and try to decompress the proximal colon as much as you can.

If the tumour is proximal to the mid-transverse colon (12-16A):

(1) **If the tumour is resectable**, resect the caecum; this is major surgery (12-16G). If the ileum is not the same size as the colon, you can make a cut on the antimesenteric border to enlarge it (11-6). When you anastomose the ileum to the transverse colon, remember to save as much ileum as you can, because its last few centimetres are the site of absorption of vitamin B12.

N.B. Don't fashion a colo-caecal anastomosis: the resection is inadequate and will not have a good blood supply.

LARGE BOWEL CARCINOMA

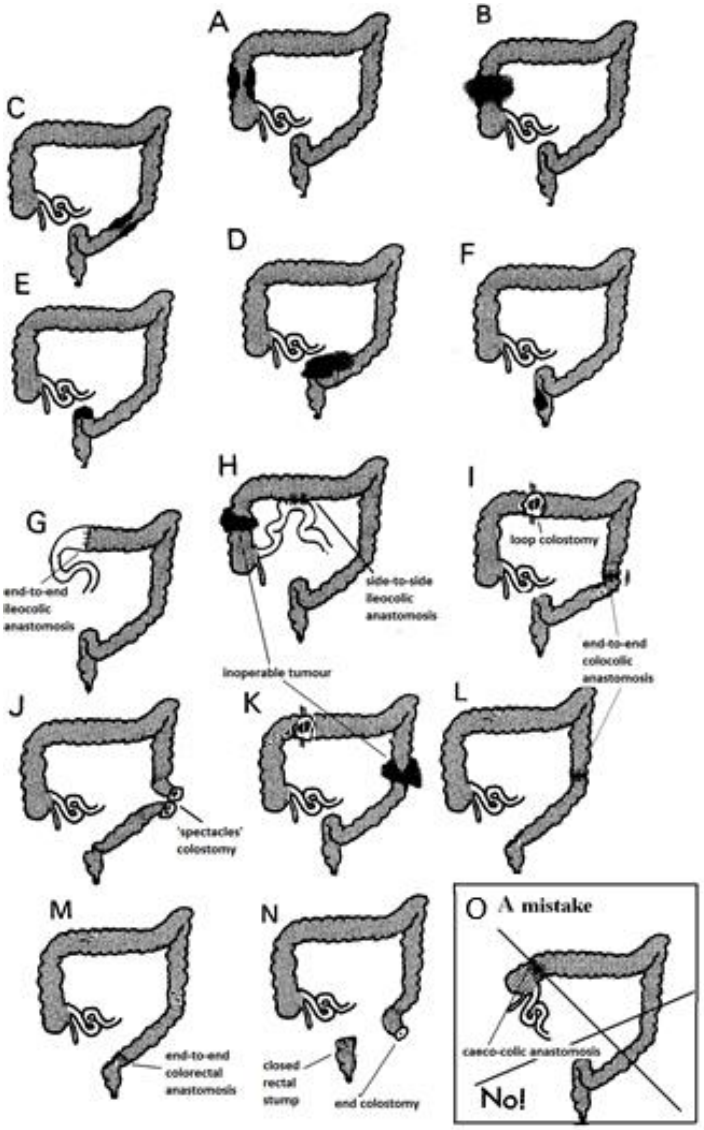


Fig. 12-16 LARGE BOWEL CARCINOMA.
Carcinomas in various sites: A, ascending colon operable. B, inoperable. C, descending/sigmoid colon operable. D, inoperable. E, upper rectum. F, lower rectum.
Solutions: G, right hemicolectomy with an end-to-end ileocolic anastomosis. H, side-to-side ileocolic bypass anastomosis. I, excision of tumour of the left colon with protective loop colostomy. J, excision of tumour of the left colon with adjacent colostomy. K, colostomy to relieve obstruction by tumour of the descending colon. L, primary resection with colo-colic anastomosis. M, primary resection with colorectal anastomosis. N, Hartmann's operation.
***N.B. don't fashion a colo-caecal anastomosis* (O).**

(2) **If the tumour is not resectable** (12-16B), bypass the obstruction with a side-to-side ILEOTRANSVERSE ANASTOMOSIS (GRADE 3.4) (11-10, 12-16H).

(3) **If the colon is obstructed**:
(i) If the condition is good and you are experienced, perform a right hemicolectomy, as in (1).
(ii) If the condition is poor or you are inexperienced, make a bypass, as in (2). This might thereby mean missing the opportunity of a curative resection, however.

If the tumour is between the mid-transverse colon and the sigmoid colon (12-16C):

(1) **If the tumour is resectable**, resect the involved bowel with its associated mesentery, leaving a margin at least 5cm proximal to the tumour. Perform an end-to-end anastomosis (12-16M). The bowel ends must have a good blood supply. If not, resect more bowel, but not more mesentery. The extent of this operation varies from being a LIMITED COLECTOMY (GRADE 3.2) to being a LEFT HEMICOLECTOMY (GRADE 3.5). Try to remove the tumour and its lymphatic drainage area to make a satisfactory oncologic resection, but *don't compromise the patient's survival by being too heroic!* If necessary, try to get a more experienced colleague to perform a wider resection if that would be possible.
Beware of the left ureter, which is easily reflected with the descending and the sigmoid mesentery (12-13A) and *the spleen*, if you need to mobilize the splenic flexure. Make the anastomosis in 2 layers, as for small bowel, preferably with a non-absorbable suture for the outer layer. *Make sure the anastomosis is not under tension*.

(2) **If the tumour is not resectable** (12-16D), bypass the obstruction with a side-to-side COLO-COLIC ANASTOMOSIS (GRADE 3.4) or make a defunctioning right transverse loop colostomy (11-14, 12-16I).

(3) **If the colon is obstructed**:
(i) If the condition is good and you are experienced, proceed as (1) provided the bowel ends have a good blood supply and you can satisfactorily clean the bowel.
(ii) If not, as this is rarely possible, resect the bowel and bring the two cut ends out as a double-barrelled colostomy (11-12D, 12-16J). Close this electively later.

If the tumour is in the sigmoid or upper rectum (12-16E):

(1) **If the tumour is resectable**, resect the SIGMOID (GRADE 3.4) or upper rectum, leaving 5cm margins clear of the tumour, as for a sigmoid volvulus (12.9). *Beware of the left ureter*. You may need to mobilize the proximal colon to make sure it reaches the distal end without tension. Perform an end-to-end anastomosis (12-16M). If access to the anastomosis is difficult, start it at the back (posteriorly) in the middle with 2 separate sutures knotted together and work round towards the left and right sides in turn, finishing at the front (anteriorly). If you cannot make a safe anastomosis, bail out and fashion a colostomy and mucous fistula or perform a Hartmann's operation (12.9).

If you have made an anastomosis which you think may leak, make a defunctioning proximal colostomy in addition. This won't have any effect on the risk of an anastomotic leakage, but will make it less dangerous for the patient.

(2) **If the tumour is not resectable** (12-16D), make a defunctioning right transverse loop colostomy (11-13): this is preferable to a sigmoid colostomy which may later be encroached by spreading tumour.

(3) **If the colon is obstructed**:
(i) Resect as above in (1) and make a colostomy proximally and a mucous fistula distally, or
(ii) Perform a Hartmann's operation: resect the sigmoid, bring out the proximal bowel as an end-colostomy, and close the rectal stump (12.9, 12-16N). If you are confident with bowel anastomoses, you can clean out the bowel by decompressing it proximally, or doing an intra-operative washout (12.9), and make a primary anastomosis; make sure you test it afterwards by filling the pelvis with water and blowing air up the rectum (12.10).

If the tumour is in the middle or lower rectum (12-16E):

(1) **If the tumour is resectable**, you should not have proceeded unless you can perform an anterior or abdomino-perineal resection of the rectum, or, better, a rectal resection with a colonic pull-through and colo-anal anastomosis. Biopsy the tumour through a proctoscope or sigmoidoscope if this was not already done. If the tumour is stenosing and likely to obstruct, make a sigmoid loop colostomy (11.6).

(2) **If the tumour is not resectable**, make a sigmoid loop colostomy (11.6) unless you think he will die before he becomes obstructed. Radio- or chemotherapy (as before) may shrink the tumour and make it resectable.

(3) **If the colon is obstructed**, make a transverse colostomy, if you think he could have definitive surgery later, or a sigmoid loop colostomy if this is unlikely.

CAUTION! **If there are liver metastases or a fixed tumour**, think hard before you make a colostomy. The patient may live a few more months, but dying with a colostomy will be miserable, especially if colostomy care is poor. If the tumour is not resectable, it is better to perform a bypass operation, an ileotransverse or colo-colic anastomosis. This is possible for lesions of the ascending, transverse, or descending colon, but not the distal sigmoid or the rectum.

If a bypass is impossible, however, a colostomy is better than dying in obstruction.

(i) Lavage. When the operation is over, wash out the peritoneal cavity with warm fluid; *don't insert drains.*

12.12 Caecal volvulus

Rarely, the caecum, ascending colon and ileum may all twist. This can only happen if they are all free to rotate as the result of a rare anomaly of the mesentery. This is more common during pregnancy. Twisting causes sudden severe pain and vomiting. The abdomen distends and becomes tender centrally and in the right lower quadrant. Signs of strangulation develop quickly.

(a) Abdominal radiographs show a huge appearance of gas which is not where the caecum should be, but is central, or even in the left upper quadrant where it may mimic the stomach. Unlike a sigmoid volvulus (12-12B), this radiological appearance does not have 2 limbs descending into the pelvis.

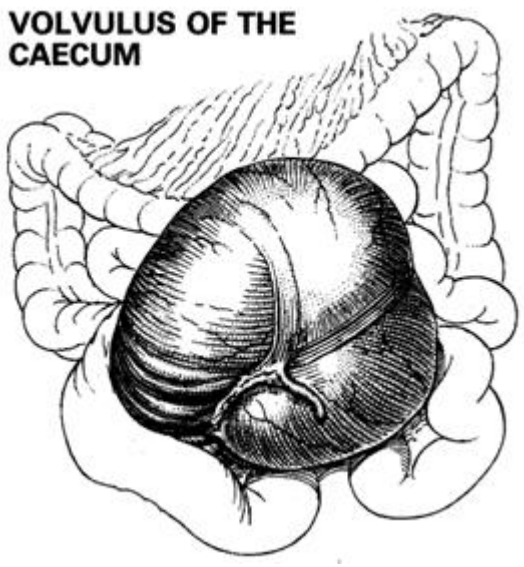


Fig. 12-17 VOLVULUS OF THE CAECUM can only happen if the caecum, ascending colon and ileum are all free to rotate as a result of a rare anomaly of the mesentery.
Adapted from a drawing by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

(b) Operation
Make a midline laparotomy incision. A huge drum-like structure seems to fill the entire abdominal cavity. Decompress it (12-6). When you inspect the right lower quadrant, you will find that the caecum is not in its normal place. Untwist the caecum.

If it is viable, ask your assistant to retract the right side of the abdominal incision. Anchor the caecum to the peritoneum to the right of it with several seromuscular non-absorbable sutures, passed through its taenia. *Don't complicate this procedure by fashioning a caecostomy, or adding an appendicectomy, which may contaminate a previously clean operation.*

If it is not viable, and you are skilled, perform a right hemicolectomy (12.11). If you are less skilled, exteriorize it, as for an ileocolic intussusception (12.7).

12.13 Chagas megacolon

In South America, almost 20% of the adult rural population may be infected by the parasite *trypanosoma cruzi*, which is deposited in insect faeces on the skin and introduced into the circulation by scratching. The parasite is carried by a bug, *triatoma infestans*, which lives in cracks in walls and in thatch, and can be killed by insecticide spraying.

A neurotoxin released and inflammatory reactions cause destruction of the Meissner and Auerbach neural plexuses in the colon, resulting in uncoordinated peristalsis especially in the distal colon; this is exactly the same picture as in Hirschsprung's disease in the baby.

The result is tenesmus, progressive constipation, difficulty evacuating flatus, abdominal distension and spurious diarrhoea. Faecal loading may result in sigmoid volvulus (12.9). The usual picture is a megacolon. *Trypanosoma* may also affect the heart and oesophagus, giving a picture like achalasia (30.6). HIV may cause reactivation of dormant infection, so you should screen for HIV.

(a) Blood films. Fix thin capillary blood films in methanol and stain both thin and thick films by Giemsa, immersing the thick films beforehand for 1sec in 0·5% aq. methylene blue. Microfilariae can be seen at x100 or x400 magnification. (More sophisticated haemagglutination or immuno-fluorescence tests are also available.)

(b) Abdominal radiographs. Faecal loading results in dilation of the colon, initially distally. Appearances of sigmoid volvulus may develop.

(c) Management. Advice on hygiene and high-fibre diet is all that is needed for early cases; enemas may need saline drip irrigation in addition to soften faecalomas. You may have to effect a manual evacuation.

You are unlikely to offer a definitive surgical solution, which involves either a low anterior resection of the rectosigmoid, or an abdomino-perineal pull-through resection with delayed anastomosis.

12.14 Mesenteric thrombosis

If the mesenteric blood supply is compromised, patches or whole segments of small bowel may become ischaemic. Chronic arterial insufficiency can produce longstanding discomfort ('intestinal angina'), but there is usually a dramatic acute vascular event.

If this is arterial from an embolus, the result is sudden ischaemia of the small bowel which rapidly becomes necrotic.

If the thrombosis is venous, there is infarction of bowel but because of the vascular arches this may be incomplete and patchy. Venous thrombosis occurs especially in HIV disease, in *angiostrongylus costaricensis* infestation (seen

in Central and South America), in *clostridium perfringens* infection (14.4) and after radiation damage.

The result is, in both cases, peritonitis as bowel organisms translocate through the ischaemic bowel wall. This may be difficult to distinguish from pancreatitis and amylase levels may be raised.

(a) Abdominal radiographs
Featureless bowel gives no clue towards the diagnosis initially, but thickened bowel wall with air within the wall develops later; occasionally you can see air in the intrahepatic portal veins!

(b) Management. Often you only discover the problem at laparotomy.

If bowel is frankly necrotic, resect the affected portion.

If the bowel is suspicious, apply warm packs, and if after 5mins the bowel remains suspicious, lavage, close the abdomen and plan an elective re-laparotomy after 48h. Administer heparin. A defunctioning ileostomy is no real benefit.

If there is patchy necrosis, resect affected portions, but try to limit the number of anastomoses to 2 or 3 at maximum; you may have to sacrifice normal bowel to do this. If you are uncertain about the blood supply around an anastomosis, make an ileostomy or plan a re-laparotomy after 48h.

If there is an obvious embolus in a mesenteric artery, try to remove it using a Fogarty embolectomy balloon catheter after isolating the artery segment with bulldog clamps, if the bowel is still viable. If you fail, resect the non-viable bowel.

12.15 Other causes of intestinal obstruction

You are unlikely to make the diagnosis of rarer causes of intestinal obstruction before operation; even to the experienced surgeon, the abdomen is full of surprises. Here are some guidelines:

If there is widespread carcinoma, avoid a stoma. Try to perform a bypass operation (12.11).

If you find an inflammatory swelling in the caecum or colorectum, it may be an amoeboma (14.5), a bilharzioma, actinomycosis, paracoccidiomycosis (14.1), Crohn's disease (14.2) or TB (16.3). You may not be able to make a diagnosis without histology, so take a biopsy. If there is incomplete obstruction, continue nasogastric drainage and IV fluids post-operatively, providing medical treatment for whatever is most likely in your region. If there is complete obstruction, perform a colostomy or bypass, take a biopsy of inflammatory tissue taking care not to perforate bowel, and start medical treatment.

If an intra-abdominal abscess is causing obstruction, drain the abscess: this will usually be enough to relieve the obstruction. If there is a retained swab, it may have eroded into bowel. Remove it carefully, and examine the bowel very carefully for perforations; if you find it is damaged, *don't try to repair holes* but perform a formal resection.

If you feel a solid object at the point where the distended loops join the collapsed ones, decompress the obstructed bowel proximally and apply non-crushing clamps to the empty segment. If you can easily move the solid object to another site in the bowel where the mucosa will not have been ulcerated, do so. Isolate the segment with packs and make a longitudinal incision in its antimesenteric border. Remove the foreign body and repair the bowel transversely. If it is a gallstone, it has come through a fistula from an inflamed gallbladder. Extract the stone, look for a second one if the first is faceted, and leave the gallbladder alone.

If it is a food bolus that has impacted in the small bowel especially if there is a gastrojejunostomy (13.7), try to break it up and massage down into the caecum. If you fail, perform an enterotomy as above. Sometimes narcotics smugglers ingest packets of drugs and these obstruct. Be careful not to spill their contents as sudden absorption of cocaine, for example, may be fatal.

If you find a tumour in the small bowel causing obstruction, look for other such tumours (especially purplish Kaposi sarcoma lesions). Resect the affected portion of small bowel.

If there are many inflammatory adhesions between loops of bowel, *don't try to resect bowel*. This may be Crohn's disease. Take a biopsy of a node to check for TB. Continue nasogastric suction and IV fluids till the inflammation settles.

If there is atresia of the jejunum in a neonate (33.2), much of the proximal bowel is diseased, and it is inevitably hugely distended compared with the distal bowel. Check for more areas of atresia distally by injecting saline into the distal bowel. Resect as much proximal bowel as possible and perform an end-to-back anastomosis, opening the distal bowel on its antimesenteric border.

If an internal hernia is obstructing the bowel, it will probably be of the closed loop variety. You can usually divide the obstructing structure quite safely, but be careful with a hernia into the recess formed by the paraduodenal fold at the duodeno-jejunal flexure, because you can easily cut the inferior mesenteric vein. If bowel is strangulating through a hole in the mesentery, *don't cut the neck of the constricting ring*, or it will probably bleed severely. Instead, decompress the distended loop (12-6), withdraw it, and close the defect in the mesentery, carefully avoiding its blood vessels.

If you find the ileum encased in a membrane in a woman, carefully open this 'cocoon' and free the bowel. *Don't perform an ileocaecal resection.*

If there is an inflammatory mass or abscess around colonic diverticular disease, drain it. If inflammation is

severe or extensive, or there is evidence of perforation or spillage (you will be able to smell it!), perform a proximal defunctioning colostomy, and leave a drain. *Don't try to resect the inflamed colon, unless you are experienced!*

If there is radiation damage to bowel, *it will not hold sutures well*. Bypass the obstruction.

If there is a submucosal haematoma in the ileum, and the patient was taking anticoagulants, administer vitamin K 10mg IM stat and leave the haematoma alone. Rest the bowel by nasogastric suction.

12.16 Ileus and obstruction following abdominal surgery

After a laparatomy, the normal muscular action of the bowel is usually absent for 6-72h. The return of normal bowel sounds is a sign that the bowel is starting to work properly again. The presence of a nasogastric tube and the use of opioids inhibit the return of bowel action, which is stimulated by early nutrition, mobilization out of bed, and the use of epidural analgesics.

The bowel may fail to function as a result of:
(1) Paralytic ileus, which is a prolongation of the normal post-operative inactivity of the bowel. This is the commonest cause, especially after an operation for abdominal sepsis.
(2) Persistent sepsis, either inadequately dealt with or from a new cause.
(3) Hypokalaemia or hypo-albuminaemia.
(4) Mechanical obstruction due to adhesions or, more rarely, intussusception or an internal hernia.
(5) Constipation as a result of long immobility in bed.

Distinguishing between these causes is difficult because:
(a) post-operative obstruction may cause little or no pain;
(b) a recent abdominal incision makes careful abdominal palpation more difficult.

If there are no bowel sounds in the abdomen and it steadily distends after an abdominal operation, make meticulous observations of the vital signs.

Administer an enema if there is faecal residue in the rectum. Mobilize the patient. Encourage him to chew gum. Observe for signs of peritonitis: unless there is deterioration, treat symptomatically for ileus and obstruction, and *don't re-operate*. This will allow an inflammatory mass time to resolve.

However, you should re-operate if there are signs of peritoneal irritation (which could be due to a leaking anastomosis, iatrogenic bowel damage, haemorrhage or new infection), or some mechanical obstruction unrelated to the original operation.

An ultrasound may detect a localized abscess (38.2l), but this will usually take 5 days to form.

Don't wait too long; if there is no improvement within 48-72h of a laparotomy for serious sepsis, perform a second-look laparotomy (10.1), especially if the patient is HIV+ve. You will be able to wash out further sepsis, clean out any cavities you have missed, check for viability of bowel or leak from anastomoses.

NJOROGE (10yrs) had a splenectomy for a ruptured spleen. On the 3rd post-operative day he was clearly not well. He had obstructive bowel sounds, some colicky pain, and a moderate amount of fluid was coming up the nasogastric tube. He was immediately operated on and an intussusception was found. LESSON *Don't wait too long before you re-open an abdomen*; be guided by the whole clinical picture. Early mechanical obstruction such as this is rare; ileus is more usual.

DISTINGUISH BETWEEN POST-OPERATIVE BOWEL OBSTRUCTION AND PARALYTIC ILEUS

After a messy operation with much pus, bleeding or spillage, expect ileus with *absent* bowel sounds. After a clean operation, severe ileus is unlikely; if present, it therefore points to a serious problem. Mechanical obstruction results in *increased* bowel sounds. Ileus tends to occur *earlier* and mechanical obstruction *later*.

Examine the patient frequently, asking these questions: Has he any pain? Is he passing any flatus? Is abdominal distension increasing or decreasing? How much nasogastric fluid is being aspirated? Have the bowel sounds returned? Does he have signs of peritonitis? Is there pyrexia, tachycardia, tachypnoea? Is the general condition deteriorating? *Frequent re-assessment of a patient is more valuable than any single symptom, sign or test.*

Typically, absent bowel sounds indicate ileus, and 'tinkling' ones indicate mechanical obstruction: these are late signs, however. If there is little pain, and radiographs show gas-filled loops with fluid levels all through the large and small bowel, ileus is more likely.

A patient who has passed flatus, and even stool, who then starts to distend and vomit, is more likely to have a significant problem. Unless frank signs of obstruction ensue, you should be able to treat him conservatively with nasogastric suction. If you can, try a gastrografin (*not barium*) challenge to see if there is a leak, and if contrast reaches the rectum.

Assess if the case is one where small bowel obstruction is likely, *viz.* multiple adhesions, not all released; hernia orifices unchecked; appendicectomy phlegmon or stump remaining; or where a re-laparotomy is going to be very difficult, *e.g.* a 'frozen abdomen' due to adhesions or radiation damage; carcinomatosis.

If distension progresses from Day 1 and is still present on Day 5 but the abdomen is not tender, there is probably a simple ileus. The normal post-operative muscular inactivity usually starts to resolve after 72h, but may last 7-14 days or more in the presence of infection, metabolic imbalance, impaired renal function or severe general illness.

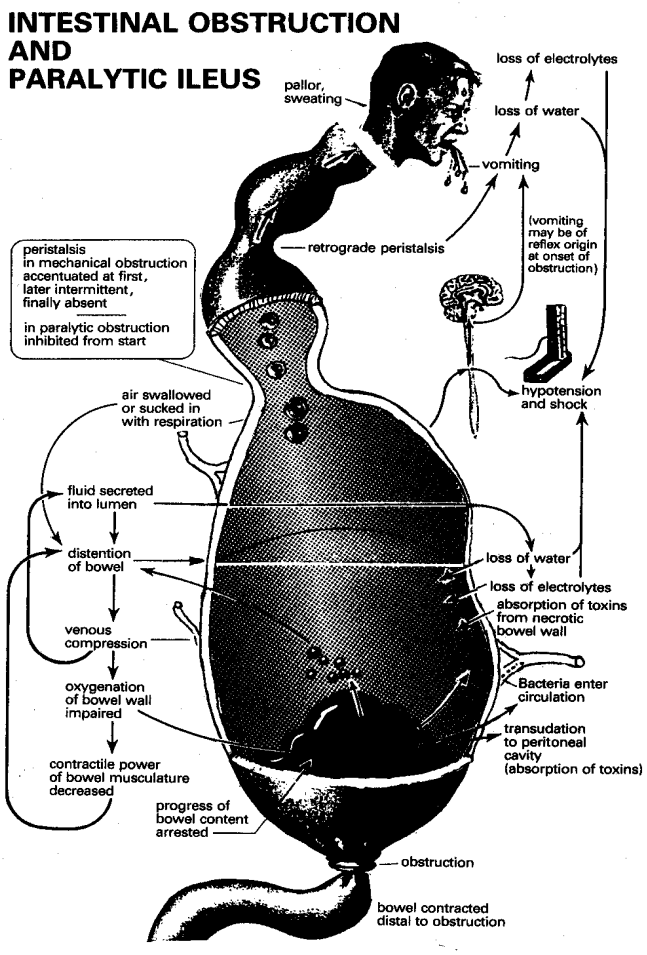

INTESTINAL OBSTRUCTION AND PARALYTIC ILEUS
pallor, sweating
loss of electrolytes
loss of water
vomiting
(vomiting may be of reflex origin at onset of obstruction)
retrograde peristalsis
peristalsis in mechanical obstruction accentuated at first, later intermittent, finally absent
in paralytic obstruction inhibited from start
air swallowed or sucked in with respiration
hypotension and shock
fluid secreted into lumen
distention of bowel
loss of water
loss of electrolytes
absorption of toxins from necrotic bowel wall
venous compression
Bacteria enter circulation
oxygenation of bowel wall impaired
transudation to peritoneal cavity (absorption of toxins)
contractile power of bowel musculature decreased
progress of bowel content arrested
obstruction
bowel contracted distal to obstruction

Fig. 12-18 INTESTINAL OBSTRUCTION AND PARALYTIC ILEUS. The passage of intestinal contents down the bowel can be prevented by a mechanical obstruction, or by a functional disturbance of the motility of the bowel (paralytic ileus). The physiological effects are much the same in both and are shown here.
Adapted from a drawing by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

If the recovery was good until Day 5, and then distension starts, there is probably a mechanical obstruction, especially if there is colic, 'tinkling' bowel sounds, distension, vomiting, but no fever. The tinkling bowel sounds may be intermittent, so you may have to listen for a long time.

If, at Day 5, the abdomen is silent and painless, with fever and a raised white count, this is probably an ileus. Suspect residual septic fluid collections and get an ultrasound scan (38.2l). Aspirate fluid and test for bile with a urine dipstick: if present, this strongly suggests a bowel leak, needing an urgent re-laparotomy.

If normal bowel function starts, and then stops again, or there is vomiting or distension, or you aspirate progressively more fluid, even >3L/day, suspect mechanical obstruction. If at the same time there is diarrhoea, there may be a pelvic abscess or, uncommonly, staphylococcal enterocolitis, or a partial obstruction, which allows some fluid to pass and obstructs the rest. Maintaining the fluid balance will be difficult.

If you have excluded enterocolitis, and ultrasound scans suggest fluid collections (38.2l), re-open the abdomen.

If there is no flatus for some hours when previously present, colicky pain, or radiographs show distended small bowel and collapsed large bowel, no fever and tinkling bowel sounds, suspect mechanical obstruction.

(a) Non-operative treatment
Continue nasogastric suction, administer IV 0·9% saline and replace electrolytes (11.9). Hypokalaemia aggravates ileus, so take care to add supplements to replace the potassium lost in the intestinal secretions. About 40mmol/day at least is needed.

(b) Operation
RE-LAPAROTOMY FOR POST-OPERATIVE BOWEL OBSTRUCTION (GRADE 3.4)

Proceed as for obstruction due to adhesions (12.6). If you do decide to re-open the abdomen, do so *very carefully*, so as not to make more damage in the bowel and create a situation far worse than before.

Take great care not to exert traction on previous anastomoses. Always decompress distended small bowel before you re-close the abdomen.

If you find much sepsis, wash out the abdomen thoroughly and look for a bowel leak.

If this is in the small bowel or is a small leak in the large bowel, close it with interrupted non-absorbable sutures, and exteriorize that portion of bowel, or fashion an enterostomy.

If this is in the proximal jejunum, introduce a feeding tube in the distal part of the bowel. You can also put a tube inside the bowel proximal to the leak, and drain this into the bowel distal to the leak: make sure you anchor this tube firmly, and make it pass outside the abdomen so you can monitor what is passing through it.

If there is a large leak in the colon, resect the affected portion, close the distal stump and bring the proximal colon out as an end-colostomy (like a Hartmann's operation: 12.9).

If there is minimal contamination within 48h of the previous operation, you will be justified in repairing the leak with interrupted transverse invaginating sutures.

13 The stomach & duodenum

13.1 Peptic ulcer

Indications for surgery on a peptic ulcer in the stomach or duodenum include:
(1) Closing a perforation.
(2) Performing a gastrojejunostomy or pyloroplasty if the pylorus stenoses.
(3) Stopping bleeding.
(4) Performing an elective truncal vagotomy and pyloroplasty or gastrojejunostomy if there is a chronic disabling duodenal ulcer which has resisted medical treatment.

Peptic ulcers are a common cause of epigastric pain in most parts of the world. The underlying cause may well be *Helicobacter pylori.* You will need to take a careful history to diagnose and manage peptic ulcer disease. This can be difficult, so enquire how the patients in your community express their ulcer symptoms. They are unlikely to give you a clear history that their pain is relieved by food, or by antacids, for example, and their physical signs may be minimal. So, in spite of the limitations of the history, it may be the only way you have of making the diagnosis. When a patient presents with the surgical complications of peptic ulcer disease, you may have to enquire carefully to find out that there have been any previous ulcer symptoms.

The decision to abandon medical for surgical treatment will often depend on the social circumstances; omeprazole, cimetidine and antacids may cost more than the patient's salary if symptoms are chronic, so operation may be a reasonable cost-effective alternative.

Don't forget that tuberculosis and burns can cause chronic gastric or duodenal ulcers, often leading to fibrosis and stricturing.

(a) History
Is there heartburn, dyspepsia, haematemesis or epigastric pain? If so, how long for, and has it recently got worse? Does it have the features of peptic ulcer pain: epigastric, dull, boring, worse at night and when the stomach is empty; relieved by food, milk, antacids, vomiting, and belching; and aggravated by coffee, alcohol, and smoking? The periodicity of the symptoms is important at first. Is there any reason for stress, in the family or at work? Is there weight loss? Or black tarry stools?

(b) Examination
Tenderness in the epigastrium may be the only physical sign. Look for other signs suggesting other diagnoses: tenderness over the gallbladder (cholecystitis), hepatomegaly (cirrhosis/hepatoma), oral candidiasis (oesophageal candidiasis), pancreatitis and epigastric hernia.

(c) Medical treatment
No smoking, no alcohol, and frequent small meals may help the symptoms. Treatment with cimetidine 400mg bd or ranitidine 150mg bd for 4wks will cure 70% of duodenal ulcers. More effective are proton-pump inhibitors (PPI) such as omeprazole 20mg od for 2wks. Extend this for 6wks for gastric ulcers, and 8wks for NSAID-induced ulcers.

Treating with magnesium or aluminium compounds in addition will reduce the absorption of antihistamines and so is not logical. Dietary restrictions are unnecessary. However, Gaviscon, a mixture of magnesium carbonate and aluminium hydroxide with gum and alginate, is quite soothing. Bismuth compounds are often useful, as they 'coat' the mucosal surface, allowing it to heal.

If *helicobacter* is common (it usually is), a week's course of ranitidine 400mg or PPI, amoxicillin 1g, clarithromycin 500mg bd or metronidazole 400mg bd, maybe with bismuth, will eradicate it in c.90% and may be worth administering 'blind'. (Unfortunately, though, in some places, *e.g.* India, there may now be resistance to metronidazole.) Remember a breath or stool test may be negative unless you stop proton-pump inhibitors 2wks beforehand! Don't treat *helicobacter*, though, unless there are severe symptoms.

Alternatively, misoprostol 200µg bd up to qds will help especially in the elderly who need NSAIDs.

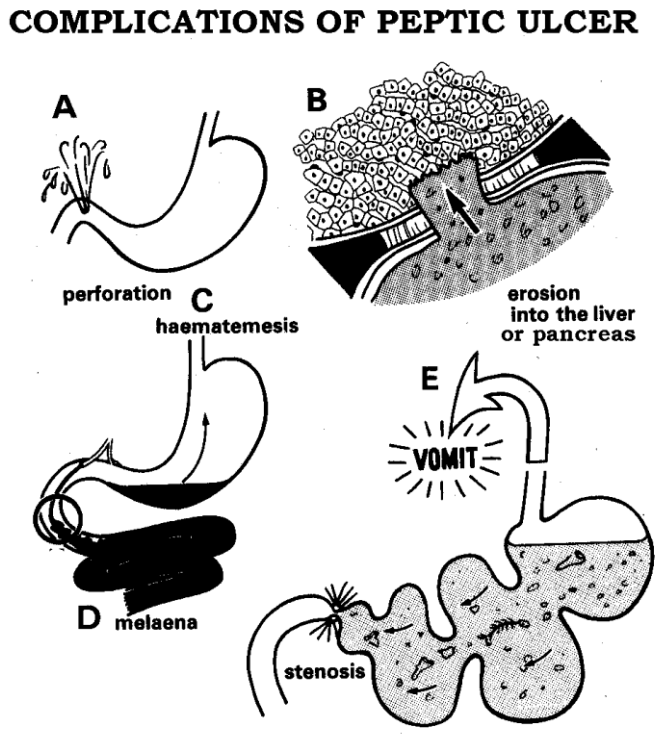


Fig. 13-1 COMPLICATIONS OF PEPTIC ULCERATION.
A, anterior perforation of a duodenal ulcer. B, penetration into the liver or pancreas. C,D, haematemesis and melaena. E, pyloric obstruction. *Note the hyperperistalsis and undigested food in the stomach.*

13.2 Oesophagogastroduodenoscopy (OGD)

You may be fortunate enough to have an upper GI endoscope; if you are, it is such a useful diagnostic tool, you should learn how to use it and care for it. As it is expensive and easily damaged, instruct a dedicated nurse to look after it, and *don't leave it to anyone*. It is very frustrating to find that your machine does not work when you need it urgently. Store the endoscope hanging up in a locked cupboard where it can remain dry. *Don't keep it in its case*, which is easily stolen and where the flexible fibres can be damaged.

Keep the additional pieces carefully in a box, and the biopsy forceps from being tangled up or caught in doors. Store the light source and suction carefully from accidental damage.

You should try to find a room dedicated to endoscopy; this should have two trolleys for patients and one for the instruments. For endoscopy to be successful, you need:
(1) the endoscope,
(2) a light source, with an air/water supply with a special water bottle,
(3) a suction machine and tubing,
(4) lubricating jelly,
(5) local anaesthetic spray,
(6) biopsy/polypectomy accessories,
(7) cleaning brushes,
(8) disinfectant and washing dishes,
(9) IV sedation,
(10) a mouthguard,
(11) disposable gloves,
(12) biopsy containers with formalin.

Hydrogen peroxide is useful for unblocking channels.

A monitor and oxygen should be available. *Simple endoscopes don't require a TV screen.*

You also need an assistant, who ideally will be familiar with the instrument and has checked it before you start.

The endoscope has 2 controls which deflect the viewing tip up and down, or right and left; it also has 2 buttons for suction and blowing in gas, and lastly a channel for passing a biopsy forceps or guidewire. It connects into the light/air source in a specific way; connection to the water bottle must be airtight. Normally the endoscope is end-viewing, but some are side-viewing: these are rather more difficult to manipulate. The suction tubing connects to a specific spout. To be able to blow, which is essential, switch the light/air source on and switch it to 'blow'.

Test this by pressing the lower button with the endoscope tip in a bucket of water. Then test the suction by pressing the upper button. Make sure the biopsy channel is free by passing a lubricated wire or biopsy forceps through it.

N.B. Air is usually used, but if you have CO_2, it is more quickly absorbed and has no risk of air embolism!

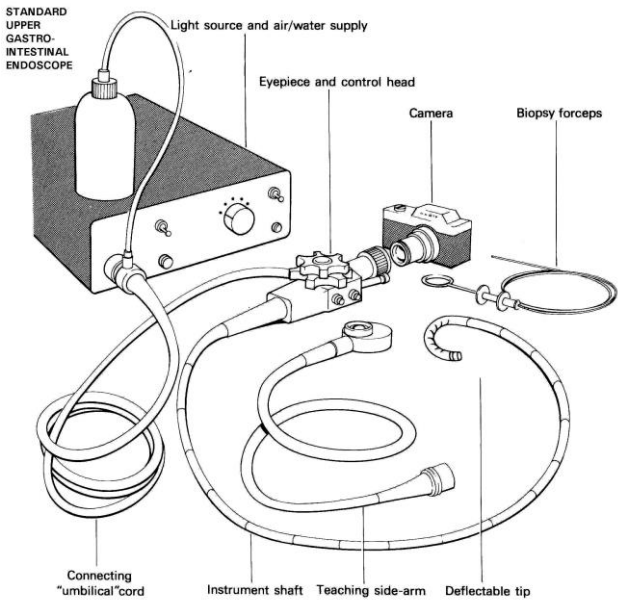


Fig. 13-2 STANDARD UPPER GASTROINTESTINAL ENDOSCOPE.
After Cotton PB, Williams CB, Practical Gastrointestinal Endoscopy, Blackwell 2nd ed 1982 p.2 Fig 2.1.

(a) Troubleshooting

If there is no light, make sure the mains fitting is working and the cable properly connected. Check the fuse box and the bulb: if necessary, replace them. If the view is dim through the endoscope and you see a mesh-like lattice pattern, the fibre-optic cables are worn and need replacing.

If it is not blowing, check the light/air source connecting 'umbilical' cord is properly pushed in, the switch is turned to blowing mode and the water bottle is connected airtight with fluid in the bottle. If all is well, remove the buttons, connect the cleaning device and flush with water. If this does not work, pass a brush down the blowing channel. If this also fails, flush the channel with compressed air. There may be some debris under the protective cap, at the end of the endoscope, through which the air bubbles out: clean this out with a fine needle. Take care when removing it not to drop it down the sink!

If it is not sucking, check the vacuum at the machine and that the tubing is properly connected and not collapsing. Remove the buttons as above, and flush and brush.

(b) Method

Explain the procedure to the patient: you are more likely to get cooperation if he understands what is to happen. Make sure he is starved and the consent signed. *Don't attempt endoscopy if you suspect he may have a perforation or pancreatic necrosis!*

Spray the throat with LA and ask him to gargle and swallow. Use a sedative unless he is severely debilitated or has respiratory problems: diazepam 5-10mg IV is usual; if you add pethidine 50mg IV you can reduce the amount of diazepam. (Midazolam 10-15mg IV gives a faster recovery.) Propofol in boluses of 30-50mg is ideal. *Beware:*

diazepam may make a young alcoholic more agitated! Ketamine is ideal in children.

If you are looking for a source of bleeding, and the patient is haemodynamically stable, either wash out the stomach with 500ml/hr tepid water till the nasogastric aspirate is clear, or administer 250mg erythromycin IV over 20mins and perform the OGD 30min later, or use both methods of preparation.

In an elderly or sick patient, attach a monitor (or have an assistant to check pulse and blood pressure) and add oxygen by nasal prongs.

Quickly introduce the mouthguard between the teeth, asking him to bite on it: do this before he becomes too drowsy with the sedative, especially if you use ketamine. Turn him onto the left lateral position, with the head and neck supported comfortably on a pillow. Ask the assistant to hold the mouthguard in place, and put her left hand behind the head and right arm over the patient's chest to restrain him gently (13-3).

NURSE POSITIONING PATIENT

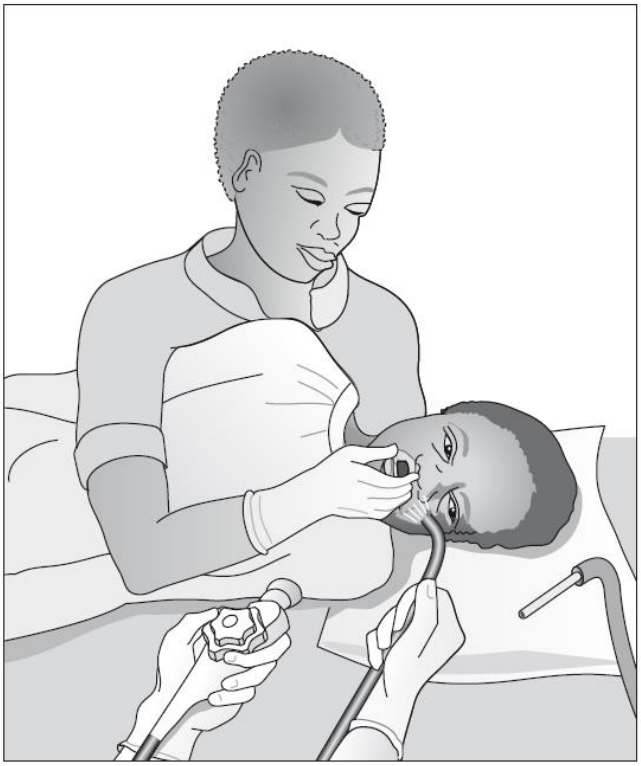


Fig. 13-3 NURSE POSITIONING. Gently restraining the patient and holding the mouthpiece.
After Cotton PB, Williams CB, Practical Gastrointestinal Endoscopy, Blackwell 1982 2nd ed p.25 Fig 4.8.

Holding the endoscope with the left hand, with the thumb free to manipulate the viewing controls, and the index and middle fingers on the suction and blowing buttons, pass the lubricated tip gently through the mouthguard.

Curve the endoscope over the tongue, which should be kept down inside the mouth, and into the pharynx keeping in the midline. Straightening the endoscope by deflecting the up/down control wheel, advance it behind the larynx, and with *slight* forward pressure, ask him to swallow. The endoscope then passes effortlessly down the oesophagus as resistance of the *cricopharyngeus* is lost: you can easily feel this.

This is the difficult part for the patient, so be encouraging and *don't get impatient*. If he coughs, suction secretions and straighten out the curve of the scope: *you are too far anterior*. If he fails to swallow the endoscope after 3-4 attempts, pull it out and try again, making sure your controls and orientation are correct.

(If you are passing the endoscope on an anaesthetized patient lying supine, you can use a laryngoscope to guide the tip down into the oesophagus.)

As you go down the oesophagus, suck out any secretions and look at the mucosa; note any irregularities, deposits of whitish *candida*, redness or stricturing. You will need to blow in a bit of air if you have used the suction. You will see the oesophago-gastric junction as the mucosa turns from pale pink to red; *where this is in relation to the diaphragm is not really relevant:* the degree of oesophagitis is.

Just as you pass the *cardia*, blow some air in and turn the scope slightly down and left (as the oesophago-gastric junction is at a slight angle), and blow air into the stomach so you can see its lining.

Don't advance if you can't see! If the view is red (unless the lumen is full of blood), the endoscope tip is against the mucosa, so withdraw it and blow air in.

GRIPPING THE ENDOSCOPE

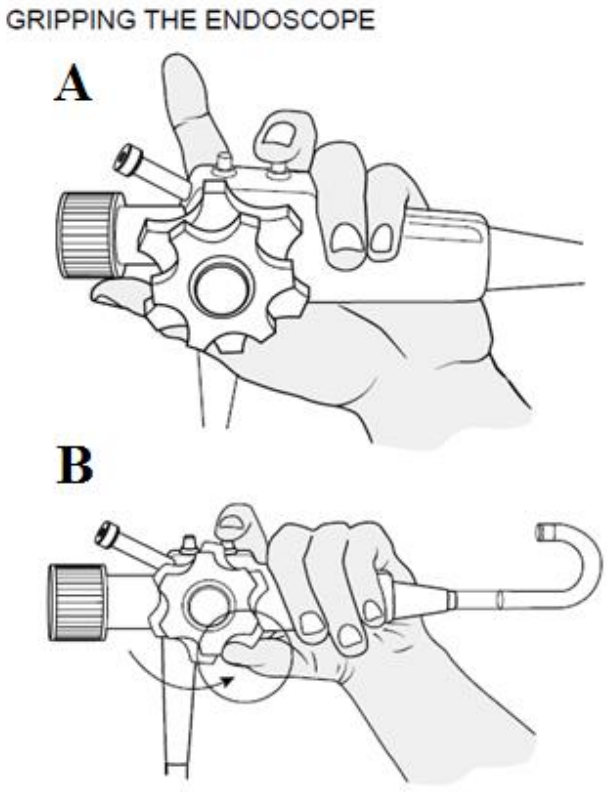


Fig. 13-4 GRIPPING THE ENDOSCOPE. A, use the middle finger for suction and blowing air. B, use the left thumb for the up/down and for the left/right controls.

ENDOSCOPIC VIEWS OF THE STOMACH

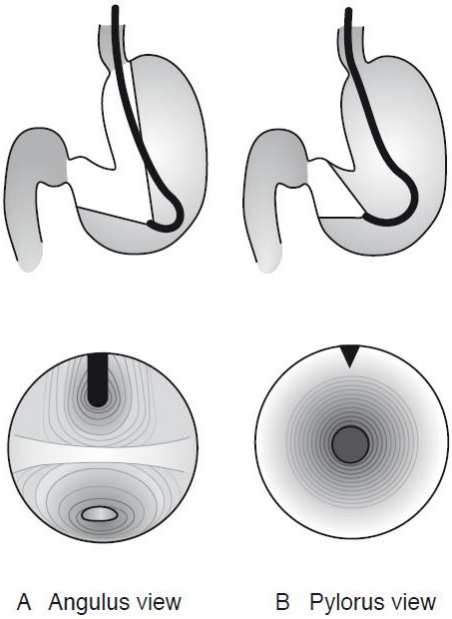


Fig. 13-5 ENDOSCOPIC VIEWS OF THE STOMACH.
A, *angulus* view. B, pylorus view. *After Cotton PB, Williams CB. Practical Gastrointestinal Endoscopy, Blackwell 2nd ed 1982 p.29 Fig 4.16*

You should see a small pool of gastric juice in the posterior part of the body of the stomach: suck this out and blow air in. You then will notice a ridge ahead (the *incisura*, or *angulus*) above which is a view of the lesser curvature.
Below this is the *antrum*, leading to the pylorus. You may be surprised by the very short distance there seems to be between the *incisura* and the pylorus such that the endoscope tip may 'jump' out of the pyloric opening back over the *incisura* into the body of the stomach. You need then to try to pass the endoscope tip through the pylorus which appears as a black hole; this may seem like driving a truck with your hands off the steering wheel into a moving tunnel entrance (13-5)!
Be patient: *don't suck or blow air in excessively* because this may irritate the patient. If he is very lively, hyoscine 20mg IV will reduce spasm at this stage.

N.B. Don't use hyoscine in patients with glaucoma!

Keep one hand on the endoscope controls and the other on the instrument tip and wait till the pylorus opens; then quickly pass the endoscope tip through. It will tend to slip past against the bulb of the duodenum, and so need withdrawing a little:

ENDOSCOPIC POSITIONS IN THE STOMACH

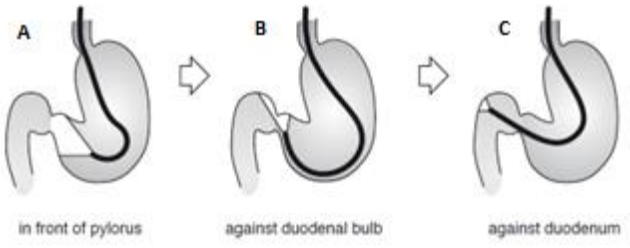


Fig. 13-6 ENDOSCOPIC POSITIONS IN THE STOMACH.
A, in front of pylorus. B, against duodenal bulb. C, against duodenum.
After Cotton PB, Williams CB, Practical Gastrointestinal Endoscopy, Blackwell 2nd ed 1982 p.30 Fig 4.23

Pass the endoscope round the duodenal angle, but *don't force it* because if the duodenal cap is distorted from old scarring, this is where you can perforate it! You rarely will need to go past the 2nd part of the duodenum, and anyway then you will need a side-viewing endoscope.

ENDOSCOPIC GASTRIC MANIPULATION

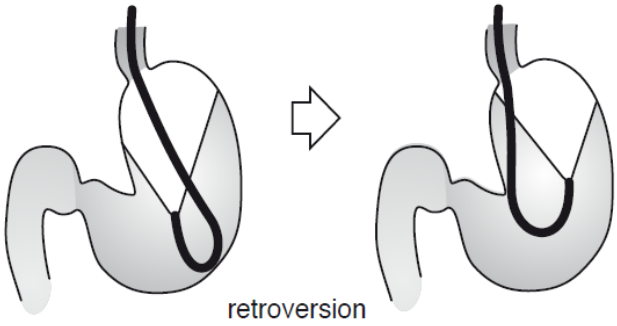


Fig. 13-7 ENDOSCOPIC MANIPULATION. Retroversion.
After Cotton PB, Williams CB, Practical Gastrointestinal Endoscopy, Blackwell Oxford 2nd ed 1982 p.29 Fig 4.19

Now scan the areas you have missed on the way in (13-7): gently withdraw the endoscope and look carefully at the first part of the duodenum, and then at the pylorus. An ulcer shows as a yellowish sloughy area, which may bleed slightly on touching with the endoscope tip. You can use this moment to take biopsies for *helicobacter* near the pylorus and examine the mucosa of the stomach. Make sure you look at the fundus by retroversion of the endoscope looking towards the *cardia* where you will see the black tube of the instrument coming through. As you pull the instrument out, you will be able to see the *cardia* close up; look again at the oesophagus and pharynx as you come out.

(c) Difficulties with OGD

If the patient becomes distressed, check the monitor and add oxygen by nasal prongs. If this fails, withdraw the instrument and try again later. If he is agitated because of alcohol misuse, diazepam may make him worse: use 50mg pethidine IV.

If you have done an inadvertent bronchoscopy, he will usually be coughing profusely. Withdraw the instrument, and suction at the same time, and when he has recovered, try again to introduce the endoscope into the oesophagus.

If severe abdominal or chest pain develops, abandon the procedure: There is either a perforation or a myocardial infarction. Resuscitate appropriately (13.3).

If you get lost, or only see red, *blow air in* so you can orientate yourself. If you find yourself seeing the instrument coming through the *cardia*, he will start belching. Withdraw the endoscope tip and turn it towards the left, and advance again provided *you can see where you are going!* You should find the *incisura* and then find the pylorus. Remember there may be gross pathology to confuse you: achalasia, large diverticulum, duodenal deformity, pyloric stenosis, previous surgery, *e.g.* a gastrojejunostomy, or congenital malrotation (with or without dextrocardia).

If there is excess food residue, the patient may not have starved or he may have an outlet obstruction; if you can ride the endoscope above the food, you may be able to see a cause. However, there is a risk of regurgitation and aspiration, so *don't persist* and try again after nasogastric suction. *Beware:* food particles and thick *candida* can block the endoscope channels and damage them.

If you can't withdraw the endoscope, check that the viewing control ratchet is free and manipulate them so the instrument is straight. Check that the patient is not biting on the endoscope!

(d) Appearances on OGD

(1) **Oesophagus.** Early OESOPHAGITIS has a fine vascular pattern of the mucosa disappearing with oedema; it then becomes red and friable, bleeding on contact. Patches of exudate and frank ulceration with a yellow slough then result, usually in the long axis of the oesophagus. A MALLORY-WEISS TEAR is a longitudinal 5-20mm split in the mucosa. A BENIGN STRICTURE is symmetrical and smooth, usually with normal mucosa proximally. A MALIGNANT STRICTURE is asymmetrical with exuberant abnormal mucosa and raised ulcer edges but a gastric carcinoma may infiltrate under the mucosa from below. *CANDIDIASIS* looks like white spots or plaques which may become diffuse: these don't wash off with a jet of water. VARICES are bluish mounds in the long axis of the oesophagus. DIVERTICULA and FOREIGN BODIES are obvious. ACHALASIA shows no abnormality except excessive food residue which may look like *candidiasis*.

(2) **Stomach.** The redness of GASTRITIS may not have clinical significance and biopsies may be more helpful. GASTRIC ATROPHY is seen as greyish white patches and associated with intestinal metaplasia. Erosions start as umbilicated polyps and then develop into smooth-margin GASTRIC ULCER. Irregular margin, base and surrounding mucosa suggest a GASTRIC CARCINOMA. However, a submucosal malignancy will not show any mucosal changes. *Biopsy all gastric lesions for a correct diagnosis*.

(3) **Duodenum.** Persistent deformity of the pyloric ring indicates current or past ulceration; a DUODENAL ULCER appears as a break in the mucosa with an oedematous smooth raised edge and yellowish slough in the centre. You can highlight lesions more easily by spraying the surface with a little methylene blue or ordinary ink, with an injection device passed through the biopsy channel. Minor changes of 'duodenitis' are of doubtful significance.

(4) **Bleeding ulcers.** During endoscopy you may see active bleeding or evidence of recent bleeding; the Forrest classification gives you an idea of who is likely to re-bleed and therefore who needs intervention:

Type		RISK OF RE-BLEED
IA	Pulsatile arterial haemorrhage	100%
IB	Oozing haemorrhage	>50%
IIA	Vessel seen	>40%
IIB	Clot over bleeding point	>20%
IIC	Haematin-covered flat spot	10%
III	No signs	5%

(e) Procedures

(1) **Biopsy** (GRADE 1.5). It is best to use a forceps with a central spike; check that the biopsy forceps is working, the correct width and long enough, and thread it through the biopsy channel. *Don't force it through at the tip*: it may not pass if the endoscope is very retroverted or, of course, if it is too big! Take specimens under direct vision by instructing an assistant how and when to open and close the forceps, and shake them directly into a container with formalin. You will need to take at least 3-6 specimens. Frustratingly, many biopsy forceps will work outside the instrument but not when passed through the biopsy channel when the cable is damaged: this happens easily if the endoscope case is closed on top of it, so *take care* to store these delicate accessories carefully.

(2) **Dilation** (GRADE 2.3). Balloon dilators can be used for oesophageal and pyloric strictures. If you don't have these, dilators which you can pass over a guide wire are an alternative. This is a great advantage over rigid oesophagoscopy under GA as patients are generally frail, wasted and dehydrated. However, if you are not experienced you may need longer than diazepam alone will allow; add ketamine or pethidine.

Pass the guide wire *under direct vision* via the biopsy channel through the stricture. You need good suction to clear the debris above the stricture to find the opening: *don't force the wire if it snags against tumour, a hiatus hernia or a pseudo-diverticulum*. With the tip of the guide wire nicely beyond the stricture, gently withdraw the endoscope, pushing the wire in as you pull the instrument out. When it becomes visible at the mouth, ask your assistant to hold it firmly, and remove the endoscope. Dilators are either of increasing size (Eder-Puestow type) or of stepped graduation (Celestin type); pass them over the guide wire past the stricture and then withdraw them. Warm the Celestin type in boiling water to make it flexible; use the Eder-Puestow type as bougies in increasing size from Ch21 to 40. When you have passed the dilator, introduce the endoscope again to check the stomach.

If the patient has severe pain, cannot swallow or has subcutaneous emphysema or peritonitis afterwards, you have probably perforated the oesophagus (30.7) or pylorus.

Such dilation will unfortunately not help in achalasia (30.6), which needs a special balloon distended to 300mm Hg.

(3) **Oesophageal stenting** (GRADE 2.3). Make sure you have measured the position of the malignant stricture. After successful dilation, you can pass a prosthetic tube, well lubricated, over the dilator of appropriate size and guide this together with an introducer (which can be home-made) into the correct position. The prosthetic tube must have a distal flange or rim to prevent upward displacement. Remove the dilator and guide wire whilst holding the tube in place with the introducer. Then pass the endoscope through the introducer to check the position of the tube; if all is well, disconnect the introducer with a twisting motion and withdraw it.

N.B. If you have self-dilating stents, these are a big improvement on the basic fixed tube described.

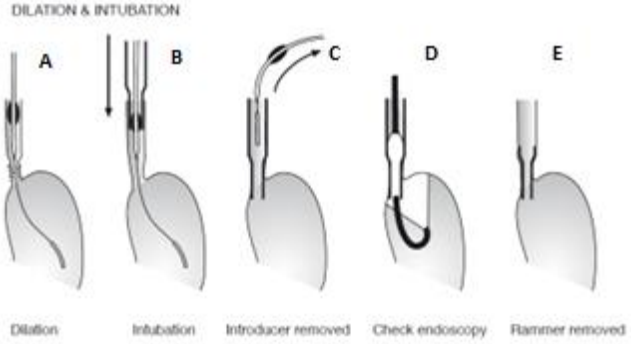


Fig. 13-8 DILATION AND STENTING. A, dilation. B, stenting. C, introducer removed. D, check endoscopy. E, rammer and endoscope removed.
After Cotton PB, Williams CB, Practical Gastrointestinal Endoscopy, Blackwell Oxford 2nd ed 1982 p.53 Fig 5.8.

(4) **Injection** (GRADE 2.3). Pass an injection device through the biopsy channel; when you see a large gastro-oesophageal varix, inject 0·5-2ml of absolute alcohol as sclerosant into the varix or just around it.
To prevent bleeding, it is best to have a plastic sleeve, specially made for the purpose from suitable tubing, with an oval window cut out at its tip, and pass this over the endoscope. Inject sclerosant through the window, and then rotate the plastic so that the tube presses against the varix and stops the bleeding. Put in up to 5 or 6 injections till you have satisfactorily dealt with all the varices; this may need several procedures. If bleeding persists, sedate the patient and leave in the overtube for 1h.
N.B. A more elegant method, if you have the tools, is gently to suck the varix into the tube, and shoot a small elastic band round it.

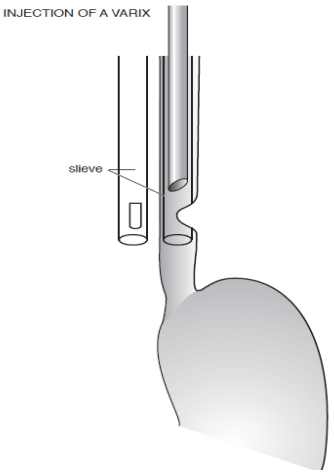


Fig. 13-9 VARIX INJECTION. Using an endoscope sleeve. Treat large grade 3 varices (coil-shaped, occupying $>^1/_3$ of the lumen) when you see 'cherry red' spots on them, or if they have bled before.
After Cotton PB, Williams CB, Practical Gastro-intestinal Endoscopy, Blackwell 2nd ed 1982 p.59 Fig 5.15.

(5) **Foreign bodies** (GRADE 2.3). Depending on what sorts of gadgets are available, you may be successful in removing foreign bodies in the stomach or oesophagus, but rigid oesophagoscopy under general anaesthesia gives more scope for manipulating objects, especially if they are sharp (30.2). Remember most foreign bodies in the stomach will pass normally. An overtube, as used for varix injection, is useful to protect the oesophagus and pharynx; pass it beyond the *cardia* and then grasp the foreign body and pull it into this protecting tube; then remove the whole lot together.

When you see an actively bleeding vessel in a duodenal ulcer, you can inject 1ml of absolute alcohol, adrenaline or hypertonic saline adjacent to the bleeding point. The problem is that you may not actually see the bleeding point if the stomach is full of blood, so make sure you have passed a nasogastric tube beforehand and sucked it out.

If you have the more sophisticated equipment, you may be able to clip a bleeding vessel.

(f) Cleaning and sterilizing
Physical cleaning of the instrument is essential: disinfectant may solidify mucus and actually make its removal more difficult if not impossible. *Don't leave this task to an untrained member of staff. Don't put off this job till hours after the endoscope has been used!*

After finishing each examination, leave the instrument tip in warm detergent with the light source still on, aspirate and blow air down the channel to loosen mucus, blood and debris. Do this till the channels seem clear. Clean the tip with a toothbrush. *Don't wet the control head of the instrument.*

Remove the rubber valve on the biopsy channel, and soak it in disinfectant. Pass the cleaning brush through the channel, and clean the bristles after they emerge from the instrument before pulling the brush back. You may need to repeat this several times. Connect the washing adaptor to the biopsy port and aspirate disinfect into the channel, leaving it there for 2mins. Soak biopsy forceps likewise in detergent.

Connect a bottle of disinfectant in place of the water bottle and flush this through the air/water channel, and then clean it with water and air. Rinse the insertion tube and biopsy channel with clean water. Remove the washing adaptor, suck hydrogen peroxide and then 30% alcohol through the biopsy channel, and then dry the instrument in air.

Wipe the tip and outside of the instrument with a gauze soaked in 30% alcohol and leave it to dry. It is then ready for the next patient.

For the first case, though, a full 10min disinfectant soak period is required. Remove the air/water and suction valves; clean these and lubricate them with silicone jelly before putting them back.

(g) Storage
Hang endoscopes vertically in a lockable cupboard with good ventilation; they should not be stored curled up in their transportation case. Biopsy forceps wires easily get tangled and caught in doors; make sure they hang on separate hooks.

(h) Documentation
Produce a regular form (13-10) with patient details, instructions, consent, indications for the procedure, and findings. Make sure you fill these correctly for each patient.

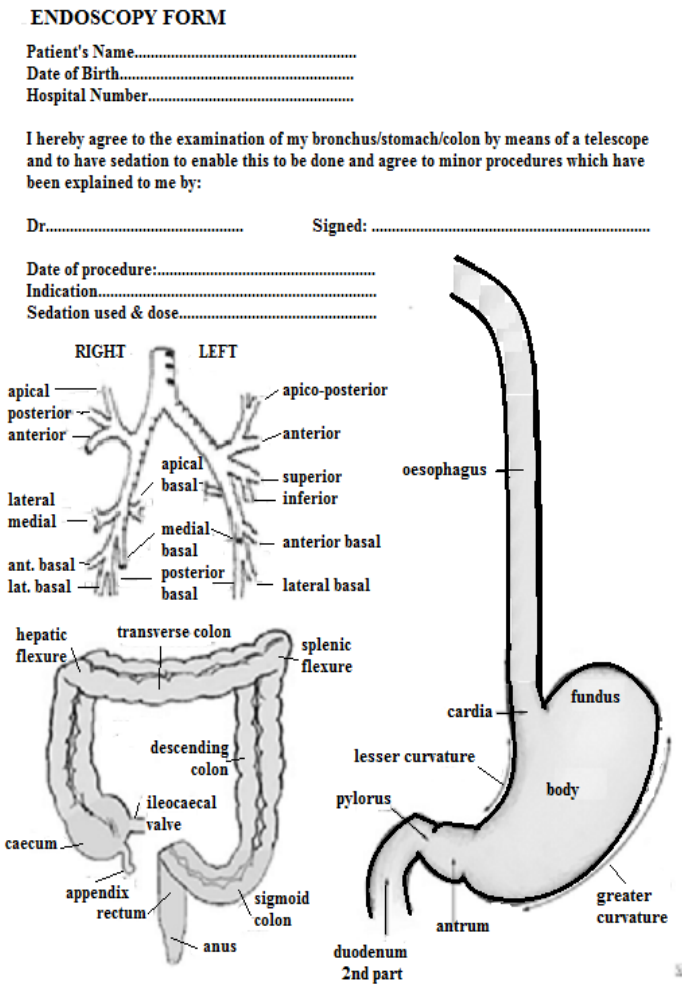


Fig. 13-10 ENDOSCOPY FORM.
Make sure you fill in all the relevant details: pictograms are best at demonstrating what you've seen.

13.3 Perforated gastric or duodenal ulcer

Classically, when a peptic ulcer perforates, it floods the peritoneum with the acid contents of the stomach, and results in sudden agonizing pain. The patient can often tell you the exact moment the pain began; it is constant, it spreads across the entire upper abdomen and later all over, and is made worse by deep breathing or movement. Usually, he lies still in excruciating pain, and breathes shallowly without moving the abdomen. He is pale, sweating, usually with a fast pulse and is hypotensive but has a normal temperature, and an abdomen which is not distended.

Typically, it has a board-like rigidity, unlike that in any other disease, which may be so complete that you cannot elicit tenderness, except when you examine him rectally.

After 3-6h the pain and rigidity lessen, he feels better and a 'silent interval' begins. Then, at about 6h, signs of diffuse peritonitis develop, accompanied by abdominal distension and absent bowel sounds.

There are difficulties:
(1) So many patients have dyspepsia, that a previous dyspeptic history is not much help.
(2) You may have difficulty in distinguishing severe gastritis or the exacerbation of a peptic ulcer from a subacute perforation (a small sealed leak).
(3) Fluid may track down the right paracolic gutter and cause pain and tenderness in the right iliac fossa, simulating appendicitis.
(4) In a patient taking corticosteroids, who is immunocompromised, or elderly, the dramatic onset may be absent. Instead, he may merely 'take a turn for the worse', a tachycardia with diminished respiratory effort and splinting of the right hemidiaphragm.

If there is a perforation, an urgent laparotomy is needed. If the patient is fit, and you operate within 6h, the result will be good. If you delay 12h, the chances of survival fall greatly. If he survives, there is a 50% chance of needing further peptic ulcer medical treatment, but <10% will require further major surgery.

Although the standard treatment is an urgent laparotomy to close the hole in the duodenum or stomach, and to wash out the peritoneal cavity, there are some indications for treating non-operatively, as described below. This is less demanding technically, but it needs careful clinical observation, and you will need good judgement to know: (1) when you have made a wrong diagnosis, and (2) when non-operative treatment is failing, so that you need to operate.

The rule in all emergency surgery is to do only what is necessary. Closing the perforation is not difficult, but be sure to wash out the peritoneum when it has been contaminated. For this you will need plenty of warm fluid.

(a) Differential diagnosis

The main diagnostic difficulty is pancreatitis or appendicitis, which is important because the first needs no operation and the second needs a different incision.

Suggesting perforation: referred shoulder pain, usually on the patient's right, the absence of fever (this develops late in a perforation), shock (when generalized rigidity is the result of appendicitis, shock is unusual), and >1L of stomach aspirate.

Suggesting pancreatitis (15.13): referred back pain, the absence of fever, shock, and a history of alcohol ingestion.

Suggesting appendicitis (14.1): central abdominal pain initially moving to the right iliac fossa, fever, a small stomach aspirate of mucoid or bile-stained fluid.

Suggesting oesophageal perforation (30.7): vomiting for any reason with sudden severe epigastric and lower retrosternal pain, or spreading between the shoulders.

(b) Radiographs
Take an erect *AP chest* radiograph. (*Abdominal views are no use, especially when the diaphragms are not fully seen*.) Make sure the patient is upright and the X-ray tube is horizontal. Look for a thin linear gas shadow between the diaphragm and the liver or stomach. If he cannot sit or stand, take a film semi-erect propped up in bed: this is better than a lateral decubitus film where you have to look for air under the anterior abdominal wall.

CAUTION!
(1) If the ulcer has perforated into the lesser sac, you may see a large irregular gas shadow in the centre of the upper abdomen, with an outline which is different from that of a loop of bowel.
(2) The absence of gas does not exclude the presence of a perforated ulcer.
(3) Gas can also come from ruptured small or large bowel, the appendix, or gas-forming organisms in severe PID.
(4) Free gas can be seen after a laparotomy or a penetrating stab wound, so *don't be misled if you see it on a chest radiograph taken post-operatively!*

(c) Non-operative treatment for a perforated peptic ulcer
INDICATIONS
(1) A perforation which appears to have sealed itself already as shown by diminished pain and improved abdominal signs.
(2) Severe heart or lung disease, which increase the surgical and anaesthetic risks.
(3) A late presentation almost moribund with diffuse peritonitis.

CONTRAINDICATIONS
(1) An uncertain diagnosis.
(2) The absence of really good nursing by day and night.
(3) The seriously ill patient, with a short history, whose only hope is vigorous resuscitation and an urgent laparotomy.

If you do decide that such a patient is 'not fit for surgery', wait until vigorous resuscitation has failed: *don't make the decision when he is first admitted*.

METHOD
Treat with morphine 5-10mg IV. As soon as this has had time to act, pass as wide a radio-opaque nasogastric tube as he will tolerate. Then get AP erect radiographs of the chest and abdomen. These should show that there are no fluid levels in the stomach, and that the tube is well placed. If not, adjust it and take more films. Look for subdiaphragmatic gas to confirm the diagnosis.
Start broad-spectrum antibiotics: gentamicin 240mg od and metronidazole 500mg tid IV.
Back in the ward, ask a nurse to aspirate the stomach every 30mins initially, making sure the tube is cleared by injection of 5ml of air before aspiration. Infuse IV saline or Ringer's lactate, and monitor the pulse and blood pressure hourly. *This is active management and needs careful observation!*
There is good progress if the pain eases, if there is no more need for analgesia after 8h, if another erect chest radiograph 12h later (optional) shows no fluid level, and if there is no increase in the amount of gas under the diaphragm. Continue to keep him nil orally on nasogastric drainage for 4-5 days, until the abdomen is no longer tender and rigid, and the bowel sounds return.

If pain persists, or the gas under the diaphragm increases, try to confirm that the perforation persists by passing some gastrografin *(never barium)* into the stomach and duodenum and taking radiographs to demonstrate the leak. If it is present, operate.

For a moribund patient unlikely to survive GA because:
(1) the presentation is >72h late,
(2) shock (BP <80mm Hg systolic) persists despite good resuscitation,
(3) there is severe cardiorespiratory disease,
(4) there is carcinomatosis, jaundice, or severe immunodeficiency,
(5) advanced age,
insert a wide bore drain through both flanks under LA into the peritoneal cavity. Irrigate the abdomen through the drains with warm water. This may improve the patient enough to avoid surgery altogether, or to allow you to operate definitively.

(d) Operative treatment for a perforated peptic ulcer
LAPAROTOMY FOR A PERFORATED PEPTIC ULCER (GRADE 3.3)

PREPARATION (10.1)
Pass a nasogastric tube and aspirate the stomach (4.9). Much fluid will be lost into the peritoneal cavity, so correct at least ½ of the fluid loss before you operate. Correct dehydration or hypotension by infusing 1-3L Ringer's lactate rapidly. If >12h have elapsed since the perforation, infuse even more. Operate soon, but not before proper resuscitation. *Unless there has been bleeding (rare), don't transfuse blood.* Pre-medicate with IV morphine.

INCISION
Make a midline incision (11.2). The escape of gas as you incise the peritoneum confirms the diagnosis of perforation (but *not necessarily a peptic ulcer*). You will probably see a pool of exudate under the liver, with food and fluid everywhere, and an inflamed peritoneum. The fluid may be odourless and colourless with yellowish flecks, or bile-stained especially if the perforation is in the 2nd or 3rd parts of the duodenum.

If you see patches of fat necrosis, this is due to acute pancreatitis (15.13).

If there is no fluid or little fluid, the perforation may have walled off: *don't disturb it!* Look in the right paracolic gutter and draw the stomach and transverse colon downwards: you may see flecks of fibrin, and perhaps pieces of food.

To expose the stomach and duodenum, place a self-retaining retractor in the wound. Place a moist abdominal pack on the greater curvature of the stomach.

Draw this downwards, and ask your assistant to hold it; at the same time ask him to hold the liver upwards with a deep retractor. Put an abdominal pack between the retractor and the liver to protect it. If necessary, get the help of a second assistant. If access is difficult, enlarge the incision. Suck away any fluid, looking carefully to see where it is coming from. Search for a small (1-10mm or more) circular hole on the anterior surface of the duodenum, looking as if it has just been drilled out. Feel it. The tissues around it will be oedematous, thickened, scarred, and friable.

If the duodenum is normal, look at the stomach, especially its lesser curve. If the hole is small, there may be more to feel than to see. Sometimes, a gastric ulcer is sealed off by adhesions to the liver. Remember that a gastric ulcer may be malignant: take a biopsy if this does not make closure difficult. You should not attempt to excise a malignant gastric ulcer as an emergency unless it is very small. Look quickly if there is a second perforation. Open the lesser sac through the lesser omentum. Feel the posterior surface of the stomach. An ulcer high up posteriorly may be difficult to find. Feel for it carefully, and if you still cannot find it, pass diluted methylene blue dye through the nasogastric tube and watch where it comes out.

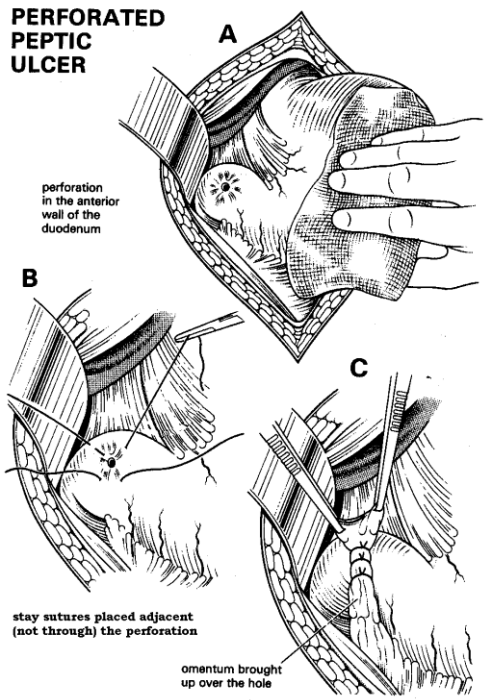

PERFORATED PEPTIC ULCER
A
perforation in the anterior wall of the duodenum
B
C
stay sutures placed adjacent (not through) the perforation
omentum brought up over the hole

Fig. 13-11 CLOSING A PERFORATED PEPTIC ULCER.
A, retract the stomach and expose a perforation on the anterior of the duodenum. B, place interrupted stay sutures of 0 or 2/0 silk or absorbable on an atraumatic needle adjacent to *(but not through)* the perforation, C, in order to pull a fold of omentum over the hole.
Kindly contributed by Gerald Hankins.

If the stomach and duodenum are normal, feel gently downwards towards the appendix, and pelvic organs in a female. Then look at the small bowel from end to end. If this is normal, examine the gallbladder, pancreas and finally the colon.

To close the perforation, place 0 or 2/0 long-acting absorbable sutures on an atraumatic needle superior and inferior to the hole (13-11B); then tie these sutures over an omental fold onto the stomach or duodenum thus covering the hole (13-11C). A hole so covered is unlikely to leak.

Don't try to bring the ulcer edges together: if the sutures cut out, the hole will be much larger than before. To improve closure, use the omentum actually to plug the hole. You can achieve this by palpating for the nasogastric tube, and pulling out its eye, threading a suture through it and attaching it to the omentum. When the nasogastric tube is pulled back, it will wedge the omentum into the hole. *This alone does not safely close perforations >2cm diameter* (see below). If you are in doubt, check if the hole is sealed by passing some dye (*e.g.* diluted methylene blue) down the nasogastric tube, and confirming no dye is leaking out.

Wash out the peritoneal cavity. This is absolutely critical, and may be more important than closing the hole. Tip a litre of warm fluid into the peritoneal cavity, splash it about well, and then suck it out again. Repeat this several times till the draining fluid is clear, and try to wash out every possible recess in the upper abdomen. Mop the upper surface of the liver.

Don't perform a vagotomy: this is an unnecessary procedure in a sick patient. *Don't leave a drain.*

(e) Post-operatively
Nurse the patient sitting up straight in bed. Breathing will then be easier, chest complications less likely, and any exudate will gravitate downwards. Continue with nasogastric suction and IV fluids (11.9). Replace the gastric aspirate with IV saline (4.9). Chest physiotherapy is vital in an asthmatic, a smoker, the immune-compromised, the elderly, or if there is widespread soiling in the abdomen. Treat with antibiotics for *helicobacter* as >80% of perforated ulcer patients have it. Start an H2-blocker or proton-pump inhibitor immediately (dilute crushed tablets with water and introduce this via the nasogastric tube, and then clamp it for 1h) and continue oral treatment for 6wks.

(f) Difficulties with a perforated peptic ulcer
I**f the ulcer is eroding into the pancreas or liver**, separate the stomach or the duodenum from the pancreas or liver by pinching between them with your finger and thumb. If this is difficult, or it is leaking into the peritoneal cavity, cut around it, and leave its base fixed. Then plug the hole with omentum.

CAUTION! Don't put your finger through the ulcer into the liver, it will bleed severely.

If the ulcer is huge, leaving only a small part of duodenum normal, closing it will be impossible or result in stenosis; prepare 2 appropriately sized, well-vascularized leaves of omentum, according to the anatomic pattern of their vessels. Plug the 1st leaf into the perforation and the 2nd as a patch over it: secure both to healthy stomach or duodenum with interrupted 2/0 long-acting absorbable.

Another method is to mobilize an upper right *rectus abdominis* flap (without the rectus sheath), maintaining its vascular pedicle superiorly, so that it reaches the perforation.
Get the anaesthetist to push the nasogastric tube further so that you can negotiate it out through the perforation. Then fix the *rectus* flap to it with long-acting absorbable suture, and get the anaesthetist to pull gently back on the tube so that the muscle flap plugs the defect.
Perform a feeding jejunostomy (11.7). Keep the patient nil orally till his nasogastric aspirate has settled, and then check closure of the perforation with gastrografin passed through the nasogastric tube.

If there is concurrent bleeding, there is probably a large circular or 'kissing' ulcer: try to undersew the bleeding vessel first. Make a large Y-shaped incision including the perforation and then try closing it making a V-type pyloroplasty (13.5, 3-12). If this is impossible, use an omental plug, with a retrograde tube duodenostomy and feeding jejunostomy as above.

If there is also gastric outlet obstruction, which is not relieved by the pyloroplasty you perform on closure, add a gastrojejunostomy (13.8).

If an 'hourglass stomach' perforates, it is from a stricture due to acid ingestion (13.10); perform a Polya gastrectomy (13.10).

If there is gross peritoneal soiling, **or suspicion of re-perforation**, consider a laparostomy (11.10) for further washout after 48h.

If there is a pergastric abscess in Morison's pouch or the lesser sac, drain it by a separate incision in the flank.

If pyrexia ensues in the 2nd week post-op, suspect there is a subphrenic abscess or other localized collection of pus (10.2).

If you continue to obtain much gastric aspirate, there is probably a pyloric stenosis aggravated by the duodenal closure. If it continues for >10 days, perform a gastrojejunostomy (13.8).

13.4 Bleeding from the upper gastrointestinal tract

In most of the world, a bleeding peptic ulcer is the commonest cause of bleeding from the upper intestinal tract, but in certain parts bleeding varices as the result of portal hypertension are more common.
Other causes of bleeding include stress ulcers, haemorrhagic gastritis, uraemia, gastric carcinoma, and a tear in the lower oesophagus following a forceful vomit (the Mallory-Weiss syndrome, 30.7).
In all these conditions the patient vomits bright or altered blood ('coffee grounds'), or he passes melaena stools, or occasionally bright blood, if the bleeding is brisk, from the rectum.

Your task is to:
(1) resuscitate the patient,
(2) make the diagnosis,
(3) assess the risk status, and
(4) control the bleeding.

Try to make the diagnosis epidemiologically and clinically, especially if you don't have a fibre-optic gastroscope, or you cannot do barium studies (38.1). The important distinction is whether or not the bleeding is from gastro-oesophageal varices, because you will not want to operate on these, whereas you may need to operate for most of the other causes. A large spleen is a most useful sign. Ultrasound will demonstrate portal venous distension (38.2c). Endoscopy is very helpful (13.2). However, even the best surgical centres cannot find a cause for the bleeding in about 10% of cases. You may need plenty of blood for transfusion.

(a) History. A history of peptic ulceration is suggestive only. Confirmation is often lacking. There is at least a 25% chance that the patient has a peptic ulcer and no symptoms. Has he been taking NSAIDs or steroids? These can cause haemorrhagic gastritis and ulcers.

(b) Examination. Look for signs of shock. A pulse of ≥120 is a reliable sign of recent blood loss *(but remember that β-blockers will mask a tachycardia).* Note sweating, restlessness, mental slowing and oliguria. Falling blood pressure is a sign that you may be losing the patient! Examine for epigastric tenderness, and rectally to make sure that a history of black tarry stools is correct. *If the blood is bright red, and the patient is not shocked, the bleeding does not come from the upper GI tract.* Look for signs of malignancy. Measure the blood urea, bleeding and clotting times. If there is vomiting blood and you have no reason to suspect severe oesophageal varices, pass a nasogastric tube and monitor the amount of bleeding into the stomach by flushing the tube with warm water from time to time.

(c) Diagnosis. The following three conditions account for 90% of cases. Other causes are rare.
(1) **Suggesting bleeding gastro-oesophageal varices:** a large spleen, a firm enlarged irregular liver, or a small hard one; abnormal vessels around the umbilicus, ankle oedema. Ascites is common in cirrhosis, less common and often not marked in periportal fibrosis, and very uncommon in extrahepatic obstruction. Spider naevi and palmar erythema are often not seen. The patient may be drowsy or in coma from hepatic encephalopathy (made worse by the digestion of the blood in the bowel). Liver function tests are abnormal in cirrhosis, but often normal in portal fibrosis.
(2) **Suggesting haemorrhagic gastritis (mucosal erosions):** the recent ingestion of alcohol or analgesic tablets. Use the history and physical signs to form some estimate of how much blood has been lost, and over how long. Decide if the blood loss has been mild, moderate, or severe. Anaemia on arrival suggests chronic blood loss.
(3) **Suggesting a duodenal or gastric ulcer:** a history of epigastric pain and dyspepsia, and especially NSAID use.

(d) Resuscitation. Cross-match 2-4 units of blood depending on the severity of the bleeding. Sedate the patient heavily with diazepam 5mg qds IV, or chlorpromazine 25mg, or use ketamine. Avoid morphine.

If the patient is in shock, set up 2 IV infusions of 0·9% saline or Ringer's lactate, with large-bore cannulae. If there is severe bleeding, infuse 1-4L of fluid rapidly, or more, until the blood pressure returns to 100mm Hg. You may need at least 3 units of blood and possibly many more. If you have a colloid plasma expander, infuse 1-2L while you wait for blood.

If you don't have blood, or enough blood, *don't hesitate to infuse large quantities of saline or Ringer's lactate:* the great need is for fluid to fill the vessels, but remember then that your threshold for operative intervention will be lower. Try to keep some blood in reserve in case bleeding resumes.

If you think gastro-oesophageal varices are unlikely, pass a large nasogastric tube. This will tell you if bleeding is continuing, and whether the blood is fresh or altered. If you aspirate clots, irrigate the stomach to wash them out. Then run into the stomach 200ml ice-cold saline containing 8mg noradrenaline and leave it for 30mins; aspirate and repeat this for 4h or until bleeding stops. *Beware that you don't make the patient hypothermic!*

(e) Monitoring. Measure and chart the pulse, the blood pressure, and the peripheral circulation ½hrly. A rising pulse or a sustained tachycardia are more important than isolated readings. Monitor the urine output, and, if possible, the central venous pressure if the patient is very ill. Measure the Hb and haematocrit as a baseline. Continued bleeding is suggested by: persistent nausea, tachycardia, pallor, restlessness, bright red fluid aspirated through the nasogastric tube, and a rapid fall in Hb or its failure to rise in spite of transfusion (a useful sign). Monitor glucose levels in liver disease, and liver function tests if possible. Correct coagulation problems if present.

(f) Endoscopy (13.2). This is most useful, if you can do it, but it will be almost impossible during heavy active bleeding unless you have very effective suction, and resuscitation facilities where you plan to perform the endoscopy. Once it has settled, it will allow you to inject gastro-oesophageal varices (13-9), or inject around a bleeding point in a duodenal ulcer.

(g) Outcome. Several things can happen. A gastric ulcer or gastro-oesophageal varices are more likely to continue to bleed than a duodenal ulcer. Melaena alone is not as serious as haematemesis, but beware of continuing melaena and unaltered blood in the stools, which indicate persistent bleeding.

(1) **Bleeding may stop** either before admission, or with the above treatment, and not occur again (75% chance).

(2) **Bleeding may continue moderately**, but responds to transfusion. Small melaena stools or small bloody vomits continue, so that the haematocrit drifts downwards. The resting pulse may be only 90/min, but the least exertion may send it up to ≥120/min. Non-operative treatment is dangerous if this continues for >72h or you have transfused >5 units of blood.

(3) **Bleeding may stop completely and start again** in a few hours, or a day or two later. This also is dangerous. (After 3 days, however, re-bleeding is unlikely.)

(4) **Bleeding may continue severely**, with vomiting of bowlful after bowlful of fresh or clotted blood, each bleed being accompanied by a wave of weakness and sweating. The passage of large tarry stools persists. A *rapid* fall in Hb 8h after an initial bleed indicates *continued* bleeding.

(h) Management depends on the risk status.

Remember Moshe Schein's dictum:

"When the blood is fresh and pink, and the patient is old, it is time to be active and bold;
When the patient is young and the blood is dark and old, you can relax and put your knife on hold."

(Schein M, Rogers PN, (eds). Common Sense Abdominal Emergency Surgery, Springer 2nd ed 2005 p.139)

For the low-risk patient, put him to bed, treat him with antacids, H2 antagonists, or PPI inhibitors whichever is available: the best are, alas, the most expensive. Later, if possible, perform endoscopy, or a barium meal.

For the high-risk patient (the indicators are: haematemesis as well as melaena, pallor, loss of consciousness, age >45yrs, BP <100 mm Hg, pulse >120/min), management depends on whether you suspect varices (13.7) or not (13.5). Ideally every patient with an upper gastrointestinal haemorrhage should have an endoscopy. Unless you have good suction, however, you will not be able to see much. Look for adherent blood clots in an ulcer, a visible vessel 'standing up' in the ulcer base, or active oozing or spurting from a vessel.

Endoscopy gives you the chance, if necessary, of sclerotherapy for varices and injection or clipping of vessels in bleeding ulcers (13.2), but this is quite specialized practice and needs the technology and some experience.

Remember *Helicobacter pylori* is almost always present where ulcers bleed, so use antibiotics (13.3).

13.5 Surgery for a bleeding peptic ulcer

There is about a 25% chance that conservative or endoscopic treatment is going to fail. At this point you will have to decide whether or not to operate in the hope of saving life. The patient needs surgery in the case of bleeding outcomes (2), (3) or (4) described previously (13.4); without surgery, there is about a 50% chance of death, especially if he is >45yrs. If you operate skilfully, the chances of death are only about 10%. In spite of the limitations of your services, about 90% of your patients with severe bleeding should live, most of them as the result of your efforts.

One of your main difficulties may be to get enough blood: in this case, operate earlier rather than later!

The purpose of emergency surgery is to save life, so decide when your patient is more likely to die if you don't operate than if you do. Try, especially, to judge the best time to operate, but *don't put off the decision!* When you do operate, try to find where the blood is coming from, and stop it. Obviously there is a big advantage if you can perform emergency endoscopy. Doing an operation which will prevent bleeding recurring is a lesser priority, because you may be able to arrange for a definitive operation later.

Remember though that surgery should be a controlled risk whereas further haemorrhage is an uncontrolled risk. If you have seen an adherent blood clot, or a vessel 'standing up' in an ulcer base on endoscopy, re-bleeding is very likely. If the patient is not suitable for surgery, or for some reason you decide not to operate, *don't give up*: continue ice-cold saline/noradrenaline lavage.

A gastric ulcer has stopped bleeding when the aspirate is no longer bloody. If this has not happened after 4h, abandon this method. If there is a duodenal ulcer, blood may not be returned in the nasogastric aspirate, so you will have to rely on the pulse and peripheral circulation to know when bleeding has stopped.

If you can effect endoscopic injection (13-9), try this first!

If you decide to operate, open the stomach and duodenum. If you find a bleeding duodenal or gastric ulcer, the simplest way to stop it bleeding is to undersew it.

Perform a pyloroplasty: *just remember not to close a pylorotomy longitudinally*, otherwise gastric outlet obstruction will result.

Surgery for gastrointestinal bleeding is difficult. The 2 common mistakes are:
(1) To choose the wrong type of patient to operate on.
(2) To operate at the wrong time: if you wait too long, you risk the patient's life, but if you operate too soon, the risk may be equally great, *especially if you operate before you have restored the blood volume*.

Be much more ready to operate on the patient who is bleeding repeatedly, moderately or severely, needing >4 units of blood.

The bleeding point may be difficult to find, and when you have found it, blood may obscure it, so that controlling it will be difficult. You will need a generous gastrotomy, a good assistant, a good light, and good suction.

Expect respiratory complications (11.11) and wound breakdown (11.14).

SURGERY FOR A BLEEDING PEPTIC ULCER (GRADE 3.5)

(a) Preparation. Make sure a large bore nasogastric tube is in place, blood and clots are suctioned out of the stomach, and blood is ready: you may need 4 units or more. Try to restore the blood pressure, but don't pour in fluids at one end only for him to bleed from the other end! Keep the systolic BP around 80mm Hg. Get 2 assistants to help you.

(b) Incision. Make a high midline incision extending up to the xiphisternum. Open the abdomen, and insert a self-retaining retractor in the abdominal wall. Insert a deep retractor under the liver, so that your assistant can retract it upwards. Gently draw the greater curve of the stomach downwards.

Suggesting peptic ulceration: a scarred, deformed first part of the duodenum or a puckered, thickened, hyperaemic area on the stomach, especially on the lesser curve. There may be nothing to feel if a posterior ulcer is eroding into the pancreas or the liver.

Suggesting bleeding gastro-oesophageal varices: a firm or hard, shrunken, irregular liver, and dilated veins on the stomach. If you find this, and there are no signs of an ulcer also, think about an oesophageal transection, and treat the varices (13.7). Treat for schistosomiasis if this is common in your area.

N.B. Sometimes a patient has varices *and* an ulcer.

If there is no obvious bleeding site, feel every part of the stomach between your thumb and forefinger, and go right up to the gastro-oesophageal junction. Open the lesser sac by dividing the greater omentum between the lower edge of the stomach and the colon. Feel the whole posterior surface of the stomach.

If you still cannot find the source of the bleeding, and there is melaena, check the small bowel first. Blood might be coming from anywhere from the duodeno-jejunal flexure to the caecum. If you are not sure if the contents of the bowel are blood or bile, insert a needle obliquely and aspirate. Look for a bleeding leiomyoma or gastrointestinal stromal tumour of the stomach or small intestine, Kaposi sarcoma or a bleeding Meckel's diverticulum. Then check the colon for ileocaecal tuberculosis, carcinoma, amoebic colitis, and intussusception.

If, even after you have done this, you cannot find the source of the bleeding after a haematemesis, the chances of the patient surviving are small unless you continue to try. If you have not been able to perform an endoscopy beforehand, do so now. You may not be able to see your way clearly because of a lot of blood clots in the stomach: in this case, unless there is continued massive bleeding and you simply don't have enough time to do this, evacuate the blood clots by water irrigation using a wide-bore tube through a small high gastrotomy.

Then pass the endoscope through this same opening, fastening it tight with a purse-string suture to make it air-tight in order to look for the bleeding site. (You can use a rigid cystoscope to do this, if you have no flexible endoscope.)
If this is unhelpful, or you are faced with catastrophic haemorrhage, open the stomach and duodenum. *There is no substitute for having a good look*.

(c) Opening the stomach and duodenum in gastro-intestinal bleeding
Insert moist packs to seal off the abdominal cavity. You have a choice of 2 incisions, depending on the degree of fibrosis of the duodenum:
If the scarring and fibrosis of the duodenum is mild or absent, make a linear incision (13-12A) with ⅔ of it in the stomach, and ⅓ in the duodenum.
If the scarring and fibrosis of the duodenum is severe, make a Y-shaped incision (13-12E).

Make your linear or Y-shaped incision through the serous and muscular coats of the anterior wall of the stomach, starting 4cm proximal to the pylorus, and extending over the front of the 1st and 2nd parts of the duodenum for 3cm beyond the pylorus. If there is an ulcer, centre the linear incision on this, and make it about 1cm above the lower border of the stomach and duodenum (13-12A).

Use tissue forceps and a scalpel to make a cut through the mucosa of the gastric end of the incision, so as to open the stomach. Enlarge the opening a little with scissors or diathermy. Slowly cut through the remaining mucosa with scissors. Pick up bleeding points as you reach them, or bleeding from the incision will obscure everything. If there are too many haemostats, run a continuous layer of absorbable suture along each side of the incision, and tie the bleeding points.

Inspect the inside of the stomach and duodenum. Mop out clots, and suck out fresh blood, trying to see where it is coming from. Evert the mucosal layer with Babcock forceps. Place a deep retractor in the upper end of the opening in the stomach and ask your assistant to expose as much of its interior as he can. If necessary, extend the incision 2-5cm proximally. Is there blood trickling down from anywhere? Feel the inside of the stomach. You may see or feel:
(1) An artery spurting from an ulcer on the posterior wall of the 1st part of the duodenum (the common site), or round the corner in its 2nd part.
(2) An ulcer anywhere in the stomach.
(3) Shallow erosions, high on the lesser curve.

If there was a haematemesis and you cannot find any abnormality, put the tip of the sucker, or a swab on a holder, into the second part of the duodenum, to make sure that there is no bleeding from a post-bulbar ulcer.

If you still cannot find any cause for the bleeding, try to pass the flexible endoscope through the duodenal opening distally.

If you find an acute ulcer, a solitary erosion, no longer bleeding, or multiple small bleeding erosions, close the duodenal opening with a pyloroplasty. Post-operatively, *advise against NSAID drugs*.

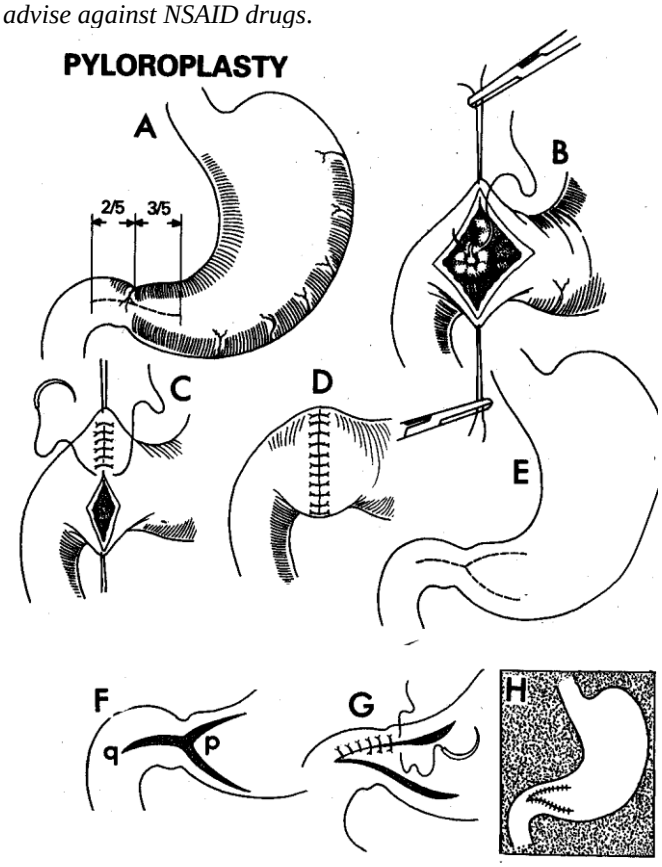


Fig. 13-12 PYLOROPLASTY (Heinicke-Mikulicz).
A, incision when there is only moderate fibrosis. The incision into the stomach is slightly longer than that into the duodenum. B, hold the incision open with stay sutures, held in haemostats, while you undersew a bleeding ulcer. C, pull on stay sutures, so as to elongate the incision transversely. Close it with close absorbable sutures of 2/0 through all coats. D, pyloroplasty completed. E, if there is severe pyloric stenosis, which makes suturing in the transverse direction impossible, make a Y-shaped incision. F, flap of the incision ('p') is going to be sutured into the duodenum ('q') so as to make a 'V'. G, suturing has begun. H, alternative pyloroplasty completed, effectively a Y-V plasty.

If you find a bleeding ulcer, control bleeding by undersewing the vessel. Retract the edges of the V-shaped pyloroplasty incision. Using non-absorbable suture on a curved needle, pass 2-3 sutures deep to the ulcer (13-12B). Tie the sutures so that you stop the bleeding. Ask your assistant to keep the area dry, and be sure to go deep enough to include the walls and base of the ulcer, but not so deep that you catch important structures, such as the common bile duct. Tie the sutures tight, but *not so tight that they cut out*.

If bleeding continues, put ligatures transversely across the pancreatico-duodenal artery above and below the centre of the ulcer, *taking care not to damage the pancreas or bile duct in the process*.

If the ulcer is in the distal duodenum, mobilize it, and make a small duodenotomy, and undersew the bleeding point as before.

(d) Pyloroplasty (GRADE 3.4)
METHOD
First make sure bleeding is controlled as described above. The kind of pyloroplasty you should make will depend on the kind of incision you made, which in turn depended on the severity of the fibrosis you found.

If you made a linear incision, because there was only mild fibrosis, hold it open with stay sutures. Pull on these so as to elongate it, and close it *transversely* with 2/0 absorbable sutures through the mucosa and serosa.

If you made a Y-shaped incision, because there was much fibrosis, sew it up as a 'V' (13-12G-H).

Finally, with both incisions, bring up a tag of omentum and fix this across the suture line with a few sutures which pick up only the seromuscular layer.

N.B. Vagotomy for peptic ulceration is really only rarely indicated for proven recurrent peptic ulceration that fails to respond to medical treatment, or, occasionally, where medical treatment is so expensive or just unavailable. This is however an operation for an expert. Consider first if, in your circumstances, a partial gastrectomy might not be a better option, even if you have to refer the patient for this.

(e) Difficulties with gastrointestinal bleeding
(1) **Stress ulceration and haemorrhagic gastritis** can occur after a burn, head or other injury, major surgical operation, or after alcohol or NSAID medication. These are usually superficial erosions in the stomach or typically in the second or third parts of the duodenum. They are usually multiple, shallow, and irregular. They usually give little pain, and severe bleeding is likely to have been the first sign. Minor harmless gastric bleeding is common after an alcoholic binge. Ulceration of this kind may ooze severely, so that there are melaena stools for several days. Treat with antacids ½hrly, and try a noradrenaline in saline lavage (13.4) and, if possible, IV cimetidine 400mg for 1hr repeated after an interval of 4-6h. (Alternatively use a continuous infusion at 100mg/hr over 24h, maximum 2·4g od.) Add tranexamic acid 1g IV and then 8hrly.

Don't operate unless the situation is critical. In this case, you need to devascularize the stomach by ligating both gastro-epiploic arteries as well as the left and right gastric arteries near the gastric wall. This effectively means an emergency gastrectomy! The chances of the patient dying are high, whatever you do.

If bleeding started after a severe episode of vomiting, from some other cause, such as a drinking bout, suspect that there is a tear in the oesophagus at or just above the gastro-oesophageal junction (the Mallory-Weiss syndrome), *which almost never requires surgery* (30.7).

If you tear the oesophagus (*which should never happen!*), repair the tear with a gastric patch bolstered by a fundoplasty (30-6). Leave the nasogastric tube in position, and feed the patient through this later.

If the bleeding point in the duodenum is obscured by blood, apply warm packs and pressure, and wait 10mins.

If bleeding re-starts after the operation, manage this non-operatively; *don't try to re-explore*.

If you find what looks like a malignant gastric ulcer, adapt what you do to the size of the lesion (13.10): if it is small, make a local excision with a 2cm margin, and repair the defect in two layers. If the lesion is advanced, and it is no longer bleeding, take a biopsy, and if it has not metastasized to lymph nodes or the liver, try to get more radical surgery done later if you can. If the lesion is still bleeding, try a figure-of-8 suture with haemostatic gauze, or as a desperate measure, devascularize the stomach as above. (Then arrange a salvage gastrectomy quickly.)

(f) Difficulties with pyloroplasty
If the duodenum is friable and cannot take sutures, close the gastric and duodenal stumps and so isolate the diseased part of the duodenum. Leave an adjacent drain and a wide-bore nasogastric tube *in situ*. If you can operate quickly, fashion a gastrojejunostomy (13-16); otherwise close the abdomen and return 48h later to do so.

If you cannot close the pyloroplasty without tension, mobilize the duodenum by dividing its attachment to the posterior peritoneum laterally, after drawing down the hepatic flexure of the colon. (This is the Kocher manoeuvre.)

If the spleen starts to bleed during the operation, you have probably pulled too hard on the crus. Pack around the spleen and wait to see if bleeding stops. Then finish the rest of the procedure, and if there is no more bleeding, carefully remove the pack. If further bleeding ensues, depending on your experience, either replace the pack and perform a 2nd look laparotomy, or proceed to splenectomy.

If peptic ulcer symptoms recur, try to do an endoscopy to confirm this. You will have to resort to medical treatment, especially with proton-pump inhibitors or misoprostol (13.1). Make sure you have eradicated *Helicobacter pylori*. Exclude hypercalcaemia and the Zollinger-Ellison syndrome (gastrinoma, usually of the pancreas). A 'maltoma', which is a precursor of B-cell lymphoma, may look just like gastritis on endoscopy: here you should try to eradicate *helicobacter* totally to prevent the development of a lymphoma.

13.6 Hypertrophic pyloric stenosis

In young children, hypertrophic pyloric stenosis is *not* due to duodenal ulceration. It presents as forceful bile-free vomiting, with constipation rather than diarrhoea, in a baby of about 3-6wks; the range can be 5 days to 5 months. It is more common in boys than in girls, and in the firstborn. To begin with, the child vomits 1-2 feeds each day, but as the obstruction gets worse, the vomiting becomes more constant and more projectile. Occasionally, he vomits brownish 'coffee grounds'. If he is not treated, he becomes dehydrated, alkalotic, hypochloraemic, hypokalaemic, and *constipated*; he loses weight, and becomes malnourished. Pyloric stenosis is not diagnosed as often as it should be, and is too often thought to be yet another case of 'gastroenteritis'. But *there is no diarrhoea!* Misdiagnosis is a tragedy, because surgery is not too difficult and is very effective.

You should be able to feel the hypertrophied pylorus *with warm hands* as a smooth olive-shaped swelling in the right epigastrium. If the baby cries, you certainly won't be able to feel it, so sit him on his mother's lap, and feel for it while she feeds him from the breast. If you have difficulty, return a few minutes later, while she is still feeding him. Sit opposite her, look for waves of gastric peristalsis passing from the baby's left upper quadrant towards the right. As they do so, the pyloric swelling will harden under your finger. Feel for the lump again. *If you are persistent*, you should be able to feel it in all cases: it establishes the diagnosis.

Ultrasound is a key diagnostic tool if you can interpret the images: muscle thickness should be >4mm and the pyloric channel length >16mm with failure of relaxation.

(a) Ramstedt's operation (GRADE 3.3)

(1) **Resuscitation.** You can correct minor degrees of dehydration with 60ml boluses of oral Ringer's lactate, but a child with severe dehydration and electrolyte imbalance needs IV fluids: infuse 20ml/hr 5% dextrose in half-strength saline, and reduce this to 10ml/hr when he is passing urine. *Don't administer >180ml/kg/24h*.

This is not a very urgent emergency and it is best to correct electrolyte loss and dehydration over a period of 24-48h before operating. The child will usually stop vomiting as soon as the stomach is empty. If not, aspirate it through a nasogastric tube. After you have corrected any severe dehydration, and the urine outflow returns, add 20-40mol K^+ to the IV fluid, depending on how ill he is. You should have a $[HCO^{3-}]$<28mM and $[Cl^-]$>100mM for safe recovery from anaesthesia.

If the child is not fit for surgery, or you cannot get this organized, try 0.18mg/kg/day oral atropine sulphate for up to 3wks: this may avoid the need for surgery altogether.

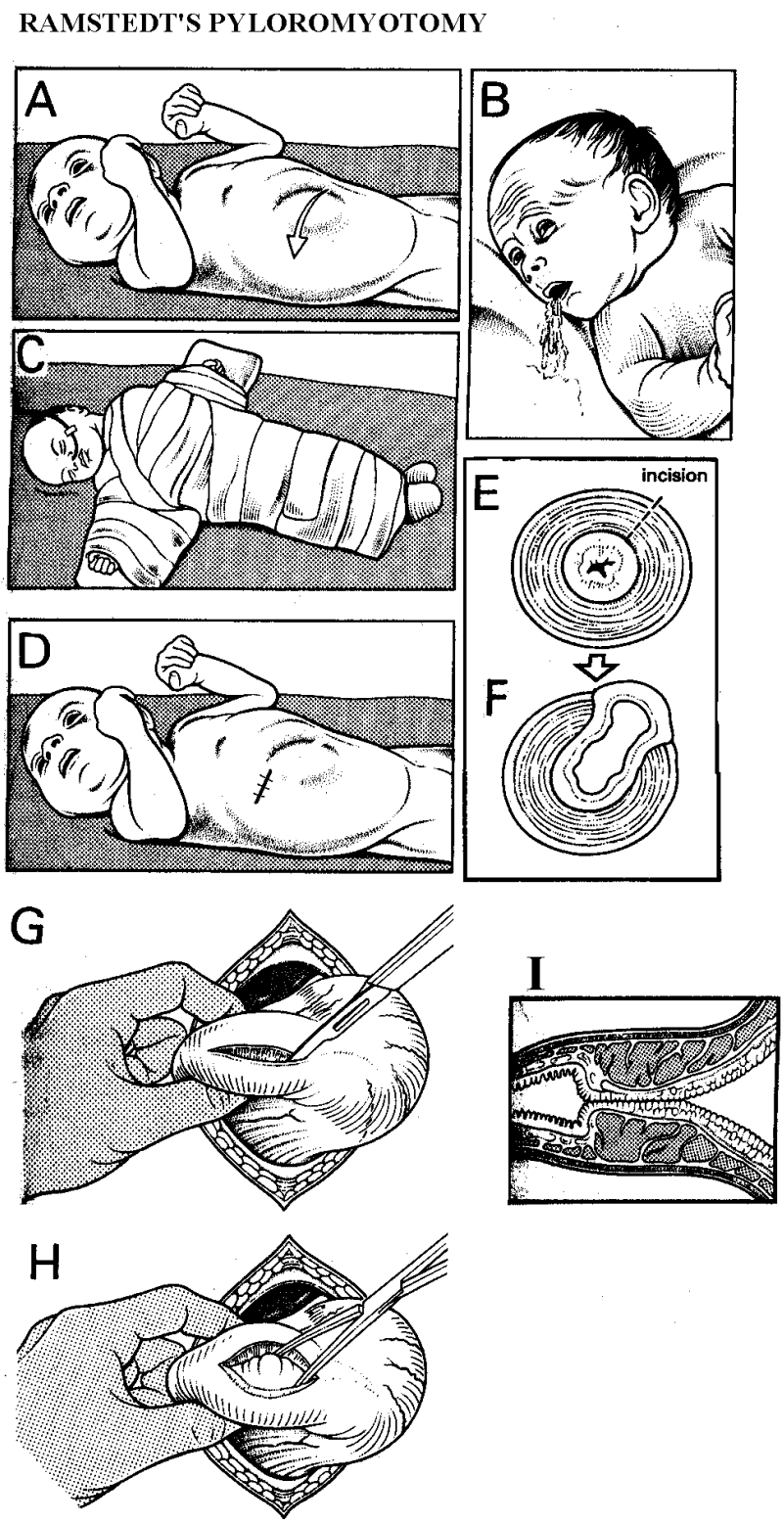


Fig. 13-13 RAMSTEDT'S PYLOROMYOTOMY for hypertrophic pyloric stenosis in young children.
A, waves of visible peristalsis passing across the abdomen. B, projectile vomiting. C, child anaesthetized on a Dennis Browne crucifix. Note the nasogastric tube. Cover the chest loosely and expose the abdomen. D, make a small right upper transverse incision. E, thickened muscle of the pylorus, showing the site of the incision. F, intact mucosa pouting out of the incision. G, incise the pylorus. H, open the incision in the muscle to reveal the mucosa. I, longitudinal section of the pylorus before surgery.
Partly after Harlow W. An Atlas of Surgery, Heinemann 1958 Figs 50-51, with kind permission.

(2) **Incision**
Open the abdomen through a transverse incision, centred over the swelling to the right of the midline (13-13D); it is usually halfway between the xiphisternum and the umbilicus. Divide all the tissues in the line of the incision.

Open the peritoneum. Make the incision long enough (3-4cm) to deliver the swelling into the wound. Retract the liver gently upwards and try to find the pyloric swelling. It may be quite difficult to find at first, because it may lie deep, partly covered by the transverse colon. Feel it with your finger. A small retractor may help to deliver it into the wound: it is always mobile.

You can gently pick up the stomach with Babcock forceps to help you find the pylorus, *but don't try to pick up the pyloric swelling with forceps as it will tear*. Hold the swelling between the thumb and index finger of your left hand. Keep your left middle finger against the distal extremity of the swollen pyloric muscle. Turn this so as to expose its antero-superior border.

Cut 1-2mm deep through the circular muscle along the length of the pylorus (13-13F). Start on the top of the swelling and continue just proximal to the white line (the junction of the pylorus and duodenum); at this point (the distal end of the swelling) the wall of the bowel suddenly becomes extremely thin. At this point, make your incision more oblique, or even V-shaped, and keep your cut very superficial. Extend the incision along the whole length of the thickened pylorus and onto the stomach (the proximal end is less clear, because the stomach wall is also thickened). Spread its circular muscle using a haemostat with its concave curve upwards, without harming the submucosa, which should bulge out of the incision. Still using the tip of the haemostat, separate the fibres distally on the duodenal side, under the white line, so as to divide all the circular fibres without perforating the duodenal mucosa (13-13G). Whilst spreading the muscle, continue to mark and protect the duodenum with the middle finger of your left hand.

CAUTION!
(i) *Don't cut the white line at the site of the pyloric vein*, or you may open the duodenum.
(ii) *Don't sew up the muscle incision*.
(iii) Make sure you have made an adequate myotomy.
(iv) Check for escape of bile or air by massaging air from the stomach distally, or inject air via the nasogastric tube, whilst holding the pylorus under water.

If you find you have opened the duodenal mucosa, close it with a 4/0 or 5/0 absorbable suture, *taking care not to occlude the lumen*: you may also suture omentum to cover the hole made by incising the muscle layer. If you have made a V-incision, you can use the distal part to cover the perforation as a transposition flap.

If a vessel bleeds, press with gauze for a few minutes; if this fails, transfix it with 4/0 multifilament.
Don't use diathermy as this may damage the mucosa.

Return the stomach to the abdomen, and place omentum over the operation site. Close the abdomen *en masse* with continuous long-acting absorbable sutures.

(3) **Post-operatively**, if you have not perforated the duodenal mucosa, remove the nasogastric tube 2h post-operatively. If you have made a perforation, leave the tube down for 24h before you remove it and start feeding. If the child is alert, provide sugar water orally by a spoon; if he tolerates this well, start breastfeeding at 6h. Proceed with feeding slowly: increase the volume of feed by 50% every 2h but leave off for 2h if he vomits: the stomach may simply not be ready.

If the child vomits frequently during the first 24h, wash out the stomach to remove the excess mucus.

If the child is not taking enough fluid by mouth to maintain an intake of 100ml/kg/day, infuse 5% dextrose in half-strength saline IV.

If the child continues to vomit after 48h, you may not have divided the hypertrophic pylorus adequately. If necessary, operate again. Wait however for 1wk to see if he is able to feed; but remember that it is better to operate earlier than allow him to become severely malnourished.

13.7 Bleeding gastro-oesophageal varices

Bleeding from gastro-oesophageal varices will be a formidable challenge; stopping the bleeding may prove impossible. If there is advanced cirrhosis, the prognosis may be so bad, and you will use up so much blood, that you may not feel it is justified using all your precious resources on this one patient.

Because oesophageal veins communicate with the portal and systemic venous system, they tend to dilate when there is elevated pressure in the portal venous system. The common causes are:
(1) cirrhosis of the liver,
(2) periportal fibrosis due to *Schistosoma mansoni* infection,
(3) non-cirrhotic portal fibrosis,
(4) portal vein thrombosis.

Death is from loss of blood and liver failure. The final cause of death may be hepatic encephalopathy, due to the failure of the liver to detoxify metabolites from blood absorbed in the bowel, either because its cells have failed, or because blood has been shunted from the liver. Liver failure commonly complicates cirrhosis, but *not* the other causes.

Your aim is to:
(1) stop the bleeding,
(2) restore the blood volume,
(3) prevent encephalopathy.

(a) Action

Get the patient to swallow 200ml ice-cold water and if this halts bleeding, repeat after 2h. Administer vitamin K 10mg IV od for 3 days, correct hypoglycaemia, and add 10mg propranolol IV over 10min *or* 20 units Vasopressin in 200ml saline or 5% dextrose over 20min. This may give rise to the side-effects of abdominal cramps, headache, and palpitations. It will also raise the blood pressure for a short time.

N.B. Vasopressin loses its activity in the heat, so, if there are absolutely no abdominal cramps, it may well be inactive. Arrange endoscopic sclerotherapy (13-9) if possible.

If bleeding continues, insert a Sengstaken tube for 24h, then deflate the balloon. If bleeding recurs, repeat the drugs and re-inflate the balloon.

(b) Inserting the Sengstaken tube

(GRADE 2.4)

Measure the capacity of the two balloons, and check that neither of them leak. The distal gastric balloon of a large tube holds about 120ml. Inflate the oesophageal one to 30mm Hg, checked against an ordinary sphygmomanometer. Add the contents of 2 ampoules of 45% 'hypaque' (or a similar contrast medium) to 250ml of saline. Make sure sedation is adequate: ketamine is useful. In an unstable patient, especially with encephalopathy, endotracheal intubation is safer.

Have a sucker available. LA in the nostril, mouth and pharynx is helpful. Lay the patient on his side, and pass the well-lubricated tube quickly through the mouth (or better, the nose); then get him to swallow the tube into the stomach. Advance the tube to the 50cm mark. Inflate the gastric balloon with the saline/'hypaque' mixture.

Withdraw it until it impacts against the cardia, and fix it by tape or suture under slight traction tension, *e.g.* to a baseball cap. Inflate the oesophageal balloon to 30mm Hg (c.50ml). Clamp and check this hourly. Tie a thread round the tube opposite the lips to mark the correct position of the balloons. Take a well-penetrated radiograph to check its position.

Aspirate intermittently from the gastric tube: this will show you if bleeding has stopped. Swallowing saliva will be impossible, so use the lateral position and aspirate continuously from the oesophagus; the Minnesota tube has an extra channel for this very purpose.

After 12-24h, deflate the oesophageal balloon, then the gastric one, and continue to aspirate the stomach.

If bleeding starts again (20% chance), you can apply the tube for a further 12h, but this is a sign that surgery is necessary, so try to refer if possible.

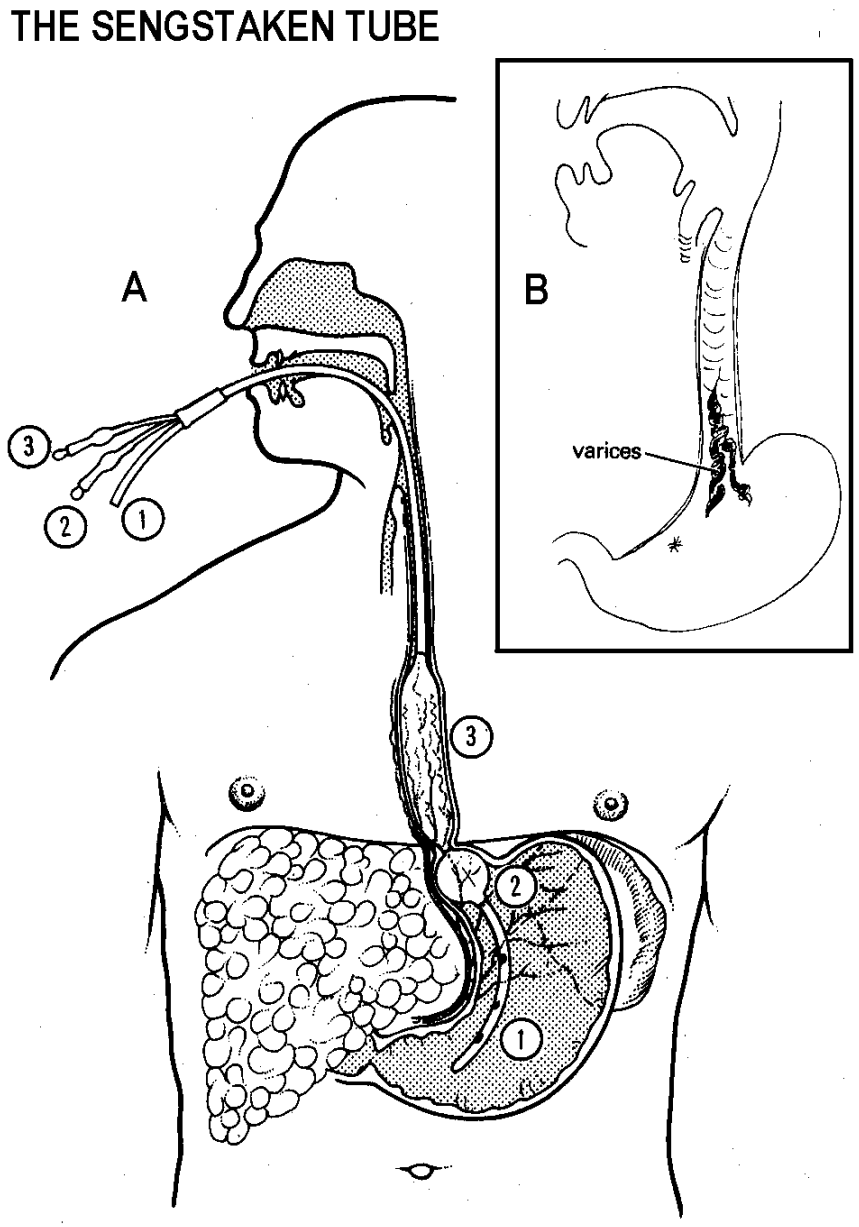


Fig. 13-14 THE SENGSTAKEN TUBE.
A, has 3 channels: (1) to aspirate blood from the stomach. (2) to inflate a balloon in the stomach to anchor the tube. (3) to inflate another balloon in the oesophagus to compress the varices. B, varices that the balloon tries to compress.
(The Minnesota tube has 4 channels, which may be more useful.)

CAUTION!

(1) If the tube displaces upwards, it may obstruct the glottis, causing respiratory obstruction. Warn the nurses about this, and tell them to remove it quickly if it does so.

(2) *Don't use it in children* because the balloon can compress the trachea.

(3) Deflate the tube after 48h. *Don't leave it in any longer*, because the mucosa will necrose.

(4) If you continue to aspirate fresh blood, reconsider your diagnosis: it may be coming from the stomach or duodenum after all.

(5) *Don't take a needle biopsy of the liver whilst in the acute bleeding stage*.

(c) Prevent encephalopathy. Use a saline purge, or magnesium sulphate 10g through the Sengstaken tube. Empty the large bowel with an enema. *Don't allow any protein orally*, but provide glucose through the gastric tube.

(d) Difficulties with bleeding varices
If you don't have a Sengstaken or Minnesota tube, use a Foley catheter although this is less satisfactory, except in children. Pass this through the nostril into the stomach, inflate the balloon with 30ml contrast, and draw it upwards so that it presses against the varices at the gastro-oesophageal junction. Either tape the catheter to the cheek or, better, tie it to a weight suspended from a pulley. Get a radiograph and aspirate as above.

If there is repeated bleeding after you have removed the tube, the prognosis is not good, but varies with the cause of the varices. If there is cirrhosis, prognosis is bad. Try endoscopic sclerotherapy (13.2): this is difficult unless bleeding has stopped, so try to pass the Sengstaken tube one more time.

If this fails, and you have a small size (25-27mm) anastomosis staple gun (4.10), and prothrombin and clotting times are satisfactory, as a rather desperate effort, try an:

(e) Oesophageal transection (GRADE 3.5)
METHOD
Make a left subcostal incision, and carefully expose the oesophago-gastric junction. You may need to clear the oesophagus of large vessels by ligating them individually: take your time! Mobilize enough of the oesophagus to be able to get a sling round it. Retract the posterior and anterior vagus nerves out of the way if possible.

Make an anterior gastrotomy, and pass the opened stapling device through the gastro-oesophageal junction; position the opened end where you intend to transect the oesophagus and tie a strong silk ligature around the stem of the device, screw it closed and fire the gun.

This transects the varices and re-anastomoses the oesophagus (13-15). Then close the gastrotomy, and if possible, tie off the left gastric vein.

This is heroic surgery and *may well not be what you should attempt in your setting without help!*

N.B. The varices will be likely to recur unless you can alleviate the problem of portal hypertension. Simple treatment of schistosomiasis, in an endemic area, may however achieve this.

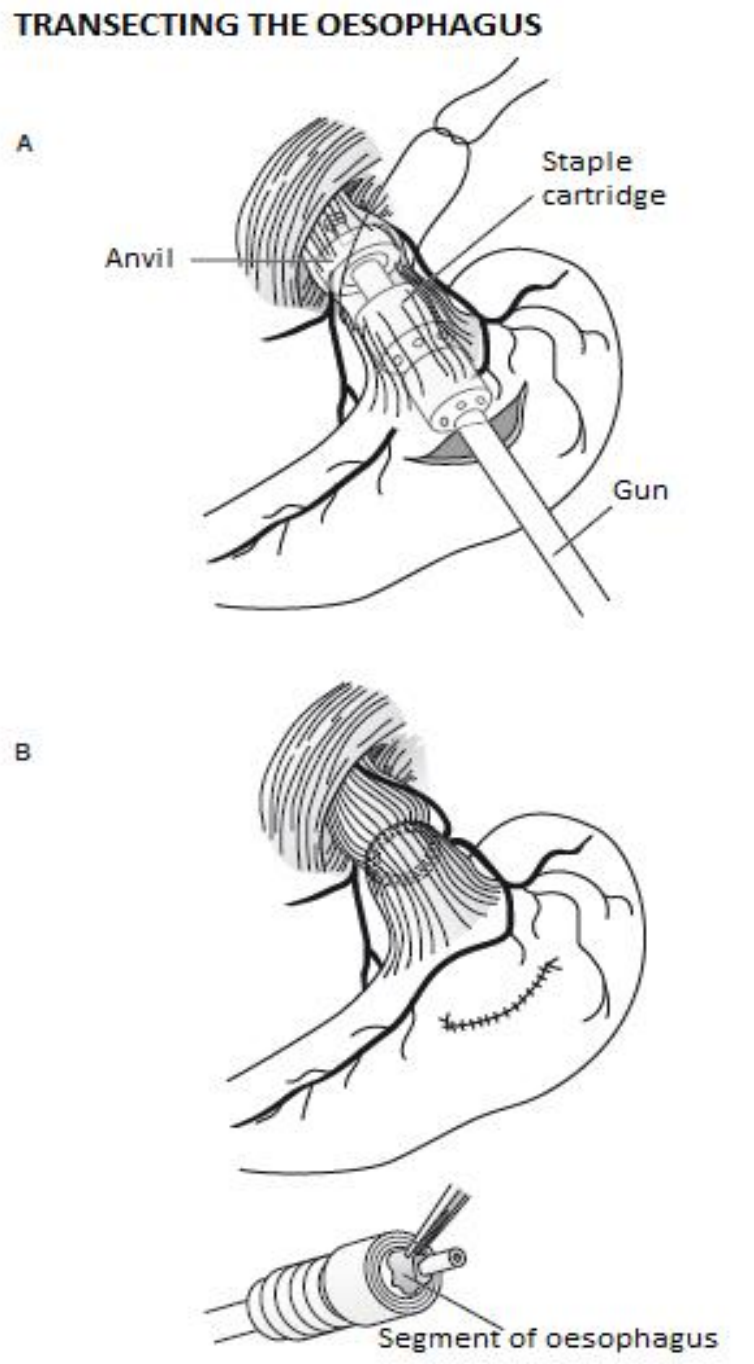


Fig. 13-15 OESOPHAGEAL TRANSECTION USING THE ANASTOMOSIS STAPLING GUN.
A, pass the instrument into the lower oesophagus via a gastrostomy, with the anvil separated and a strong ligature tied around the entire oesophagus. B, after firing the gun and closing the gastrostomy.
After Kirk RM, Williamson RCN. General Surgical Operations Churchill Livingstone 2nd ed. 1987 p.208 Fig 11.6a,b.

13.8 Gastric outlet obstruction

Scarring from a chronic duodenal ulcer, TB or ingestion of caustic sometimes causes pyloric obstruction (stenosis). Obstruction may also be caused by carcinoma of the distal stomach (13.10). The patient may come to you saying that he has been vomiting for days or weeks. He may only vomit once a day or he may say that he vomits 'everything he eats'. The vomit may contain food that he ate days before. Or, he may not actually vomit, but merely feel abnormally full and bloated after only small amounts of food. He may be burping, and he may have taught himself to vomit to relieve the symptoms. He loses weight. Continued vomiting depletes the extracellular fluid, and causes hypochloraemic alkalosis, and hypokalaemia; eventually he becomes dehydrated, wasted and oliguric.

Try to confirm the diagnosis by either endoscopy (13.2) or barium meal. A biopsy is necessary to differentiate between malignant and benign causes. TB may need deep 'well' biopsies. Rarely, there is a pyloric web. Very occasionally, the stomach is distended by a huge bezoar (13.12) or by chronic binge eating (bulimia).

To relieve the obstruction, if you cannot perform an endoscopic dilation (13.2), a gastrojejunostomy is the answer: this is anastomosing the jejunum side-by-side to the gastric antrum. As the pylorus is usually badly scarred, or infiltrated by tumour, *don't perform a pyloroplasty.*

In infants, hypertrophic pyloric stenosis is a different entity altogether (13.6). In Chagas disease, the myenteric plexus may be involved, giving rise to achalasia of the pylorus (30.6): this results in pyloric obstruction without any visible stenosis. You can perform a pyloromyotomy (as in the infant) to deal with this.

(a) Examination. Lay the patient down and look for visible gastric peristalsis, as the stomach struggles to empty itself through a narrowed pylorus. Look for slow waves moving from the left hypochondrium towards and beyond the umbilicus. Rock the patient from side to side. You may hear a succussion splash even without a stethoscope. You may also hear it if you depress the epigastrium sharply with your hand (*beware:* a splash may be normal after heavy drinking or a large meal).

(b) Preparation
Washouts will empty the stomach, remove debris, and provide some relief of colic. With luck, the inflamed and oedematous pylorus will open up. Washouts will also reduce the risk of post-operative infection.

Find a funnel, a *large* (Ch36, 10mm diameter) stomach tube or a catheter, and a longer piece of rubber connecting tube the same size. With the patient prone with the head supported over the end of the bed, pass the well-lubricated stomach tube through the mouth and encourage him to swallow it. Connect the stomach tube via the other tube to the funnel. Hold up the funnel and pour in 500ml of tepid water (250ml in a child).

Before the last drop has left the funnel, lower it over a bucket (to prevent air entering). The stomach contents will run out. Repeat the process, this time using 1L water. Go on doing this until the fluid returns clear. Finally, leave 500ml inside the stomach. Repeat this daily, for 3 days or until he is fit for surgery, whichever is later. *Don't perform the washout on the day of operation!*

CAUTION! Check the volume of water you have run in and out: a marked discrepancy indicates the stomach has perforated.

(c) Radiographs are useful if the diagnosis is in doubt. Take an erect abdominal film, and look for a large fluid level in the left upper quadrant. A drink of barium will produce a mottled shadow showing that the gastric outline is much enlarged. Little or no barium passes the pylorus. *Don't administer a large quantity*, because it may be difficult to wash out, and the patient may vomit and aspirate.

(d) Rehydration may be necessary over several days to restore the extracellular fluid volume. Treat with 0·9% saline or Ringer's lactate. If necessary, correct the potassium loss with up to 80mmol of potassium od, or use Darrow's solution ($[K^+]$=34mM). Be guided by the volume and specific gravity of the urine output.

(e) Gastrojejunostomy for pyloric stenosis (GRADE 3.4)
INDICATIONS
(1) Pyloric obstruction causing dehydration and weight loss, or other longstanding obstructive symptoms as described above.
(2) Duodenal ulceration with sufficient scarring to contraindicate pyloroplasty; combine it with a truncal vagotomy.
(3) As a palliative procedure for stenosis caused by an antral carcinoma or gastric outlet obstruction by pancreatic carcinoma.

EXPOSURE
Make an upper midline incision. If you find a large thick-walled stomach, the diagnosis of pyloric stenosis is confirmed. Ask your assistant to retract the liver upwards with a deep retractor, and to draw the stomach downwards at the same time. Make sure there is enough room, because traction may tear the spleen.

Is the cause malignant? If there are hard nodules, enlarged hard lymph nodes, and perhaps an ulcer crater, just proximal to the pylorus, suspect a gastric carcinoma.
If there is a mass in the head of the pancreas pressing on the duodenum from behind, suspect a pancreatic carcinoma. Biopsy a node, and perform an anterior gastrojejunostomy.
Is the cause benign? If there is:
(1) Puckered scarring on the front of the first part of the duodenum, perhaps with adhesions to surrounding structures.
(2) An indentation on the posterior wall of the stomach extending into the pancreas to which it is fixed, suspect a chronic peptic ulcer, or tuberculosis.
Carcinoma rarely affects the first part of the duodenum, so that lesions there are almost certainly benign.

METHOD

If you are not sure what is obstructing the outlet of the stomach, perform a gastrojejunostomy and biopsy a regional node. *Don't biopsy the stomach or pancreas itself* unless you intend to resect it.

GASTROJEJUNOSTOMY

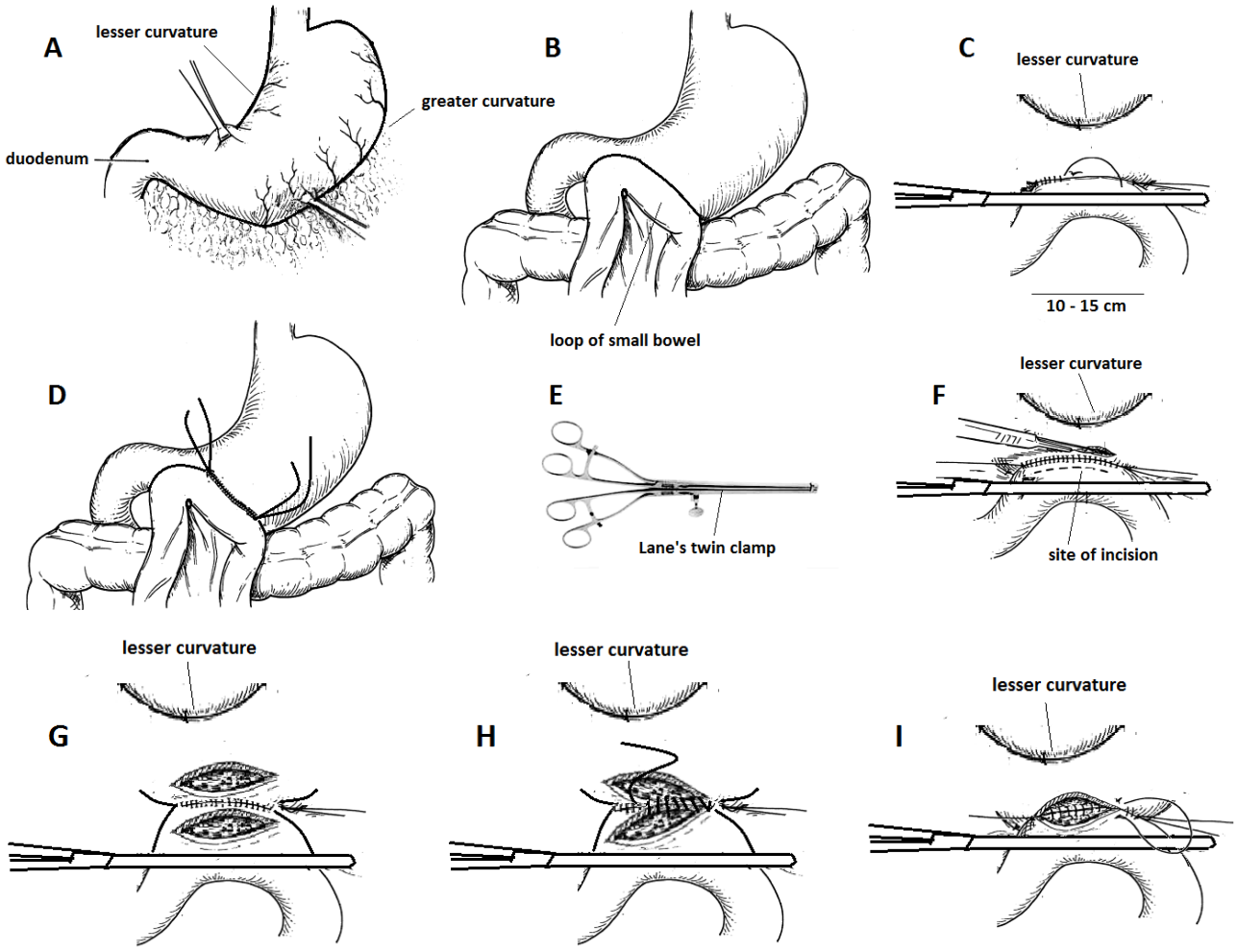


Fig. 13-16 GASTROJEJUNOSTOMY.
A, hold the stomach distally with Babcock forceps. B, pull up a proximal loop of jejunum with no tension. C, apply a non-crushing clamp to the jejunum. D, finish the posterior seromuscular (Lembert) layer. E, Lane's twin clamps are double non-crushing clamps which click together and simplify the anastomosis (especially without an assistant). F, open the stomach and jejunum (you can use cutting diathermy for this). G, stomach and jejunum opened. H, start the inner posterior all-coats layer. I, continue this as a Connell inverting suture on the anterior layer. Then remove the clamps and finish the outer anterior layer, and test the anastomosis digitally.

Grasp an 8cm segment of distal stomach, though far enough from any pathological lesion, and apply Babcock's forceps about 6cm apart (13-16A). Find the upper jejunum and apply Babcock forceps similarly (13-16B). The first should be about 8cm from the duodeno-jejunal flexure, and the second about 6cm distal.

Apply a non-crushing clamp (13-16C) to hold ⅔ of the width of the bowel, and another non-crushing clamp to hold the stomach. Insert stay sutures through the seromuscular coats of the stomach and jejunum at each end.

The stomach wall is likely to be thick, perhaps very thick, if the pyloric stenosis is longstanding. Complete the layer of continuous seromuscular sutures using 2/0 long-acting absorbable (13-16D).
(If you have Lane's twin clamps (13-16E), these will hold stomach and jejunum in position for you to operate without an assistant.)

Open the stomach by cutting parallel to the seromuscular suture line for 5cm (13-6F); then, open the jejunum for an equal length, halfway between the suture line and the clamp (13-16G). Use 2/0 atraumatic absorbable suture for the 'all coats' inner posterior layer (13-16H), starting at one end and continuing with an inverting Connell suture anteriorly (13-16I), in the same way as for a side-to-side anastomosis (11-10). Then complete the outer anterior layer. Remove the clamps. Feel the size of the stoma: it should admit 2 or 3 fingers. Cover the anastomosis with omentum.

CAUTION!
(1) Be sure to include all layers of the stomach wall in the anastomosis. If it is hypertrophied, the cut edges of its mucosa will curl away. If you fail to include them in your sutures, they may bleed, or the suture line may leak.
(2) *Take care not to rupture the spleen, or the gastrosplenic vessels* by pulling on the stomach too much: make sure you have adequate exposure.
Make sure a nasogastric tube is in place; if the patient is severely hypoproteinaemic, pass the tube into the jejunum through the gastrojejunostomy, and start enteral feeding as soon as bowel sounds resume.

N.B. There is no real advantage of performing a retrocolic gastrojejunostomy: *don't do this for malignant disease.*

(f) Difficulties with a gastrojejunostomy
If aspiration ≥1L fluid continues after the operation, the stoma is not functioning, or there is paralytic ileus. Bowel sounds and the absence of abdominal distension will exclude ileus.
The stoma will be less likely to obstruct if you make it big enough to take three fingers. It may remain obstructed for 2wks especially if the patient is hypoproteinaemic. Continue nasogastric suction, unless there is an indication to re-operate, and correct fluid losses. The stoma is almost certain to open eventually. You may be able to encourage it to function by passing an endoscope through it, or inserting a feeding tube into the jejunum.

If, some time after the operation, there is bilious vomiting, reassure the patient. Bile and pancreatic juice are accumulating in the afferent loop, and when they are suddenly released into the stomach, he vomits. The symptoms will probably improve with time. If they don't do so after 2yrs, consider a revision procedure.

If there is persistent very loose diarrhoea and vitamin deficiencies develop, you may have made a gastro-ileostomy in error: perform a barium meal to check. If you have, re-open the abdomen, take down the anastomosis, resect the portion of ileum you inadvertently used, and perform a gastrojejunostomy!

If a recurrent ulcer develops at the anastomosis (which you will probably only find by endoscopy), treat it medically in the first instance; re-do surgery is complicated.

If there is malignant gastric outlet obstruction, perform a gastrojejunostomy, proximal enough to avoid the tumour (13.10).

DIFFICULTIES WITH CHRONIC DUODENAL ULCERATION

If medical treatment fails, or is too expensive, you may be able to help a poor patient by operating. If there is uncontrollable pain and dyspepsia, or if the quality of life has been spoilt over the years by nagging pain, heartburn, and indigestion, there may be still a place for a truncal vagotomy and gastrojejunostomy or pyloroplasty.

Don't wait until there is severe haemorrhage, or the overwhelming vomiting of pyloric obstruction. Try by all means, however, to confirm the diagnosis before laparotomy, because the real diagnosis may be a chronic pancreatitis, liver disease, cholecystitis, or other abdominal pathology, or actually be psychosomatic!

13.9 Gastrostomy

If the oesophagus is obstructed, swallowing food is impossible and so starvation results. Saliva cannot descend, so it drips from the mouth. You can feed such a patient through an opening in the stomach, but this will not help him to swallow saliva. This is such a disabling symptom that there is little to be gained by prolonging life merely to endure it. There is thus seldom an indication for doing a gastrostomy for inoperable carcinoma of the oesophagus or pharynx. The possible indications for it are given below. For many of them, a jejunostomy (11.7) is a better alternative. Otherwise, you may be able to introduce a feeding gastrostomy percutaneously with the aid of a gastroscope: this is difficult without the right gadgets, and may well give you big complications. So attempt it only if you have mastered the use of the endoscope and you have all the necessary equipment available.

(a) Indications
(1) Temporary feeding during recovery from bulbar palsy or curable pharyngeal disease (*e.g.* retropharyngeal abscess).
(2) Temporary post-operative drainage of the stomach, when a nasogastric tube is impractical, *e.g.* where there is severe respiratory embarrassment.
(3) Treatment of a duodenal fistula: one tube is used for gastric aspiration, and another passed into the jejunum for feeding.

N.B. A feeding jejunostomy (11.7) *is preferable to a gastrostomy prior to oesophageal reconstruction*.

(b) Gastrostomy (GRADE 3.2)
METHOD. Under LA or GA make a small upper midline incision. Pick up the cut edges of the peritoneum and draw them apart. You will probably find that the stomach is small and tubular, so that the first thing you see is the greater omentum or transverse colon. Pull this downwards and deliver the upper part of the stomach into the wound.

CAUTION! Check that you really have found the stomach, and not the transverse colon by mistake! **If you have opened the colon**, close the perforation in two layers (14.3), and continue the operation *unless there was massive soiling*.

Make a small stab incision lateral to the midline and use a haemostat to pull a Ch20 or Ch24 Malecot or Foley catheter through it. Make the gastrostomy high on the anterior wall of the stomach, midway between its greater and lesser curves, and as far from the pylorus as you can. Hold the stomach with two pairs of Babcock's forceps, and draw it upwards and forwards into a cone.

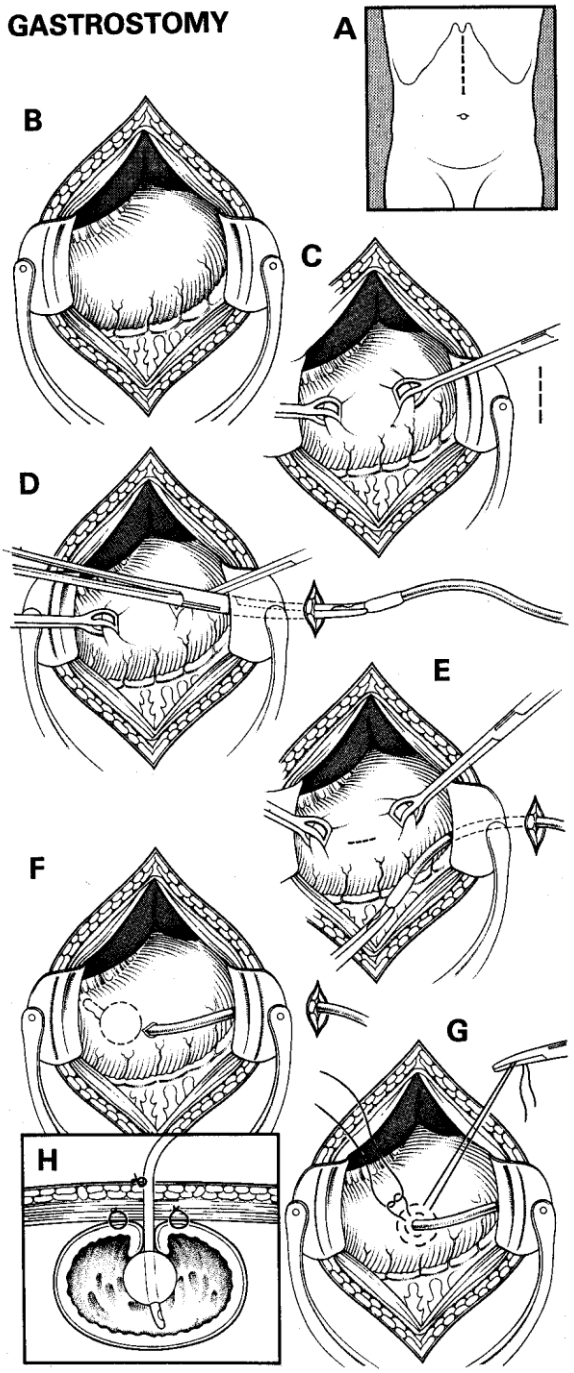


Fig. 13-17 GASTROSTOMY.
A, incision. B, expose the stomach. C, pick up the stomach with Babcock forceps. D, introduce the catheter through the abdominal wall. E, incise the stomach. F, introduce the catheter. G, purse-string sutures. H, invaginate the stomach wall.

Make a small incision between the forceps, aspirate the gastric contents and push the catheter through this. Encircle it with 2 purse-string sutures, and invaginate the stomach wall as you tie them.

CAUTION!
(1) Take the bites of the inner purse-string suture through the full thickness of the stomach wall, so as to control bleeding: the main dangers are haemorrhage and leaking.
(2) The gastrostomy must be leak-proof, so that gastric juice does not enter the peritoneal cavity; test it by flushing water through the tube.

If there is no leak, anchor the stomach above and below the tube to the posterior rectus sheath.

Close the tube with a spigot, and fix it to the skin with an encircling suture. Before the patient leaves the theatre, instil some fluid through the tube, to make sure it is patent.

(c) Difficulties with a gastrostomy
If stomach content leaks early around the tube, inflate the balloon more, or insert a larger-sized catheter.

If stomach content leaks later around the tube, and there is no abdominal pain, this may be due to some pressure necrosis of the gastric wall from the balloon, or infection of the adjacent abdominal wall. Try a course of gentamicin; if the leak persists, remove the tube and allow the gastrostomy to drain naturally. It will start to close, and before the stoma is completely shut, re-insert a catheter if the gastrostomy is still needed.

If the gastrostomy tube falls out or is blocked, re-insert a new one through the same track, if necessary with a guide wire. If you can, check by endoscopy that it is in the stomach. If you use a paediatric gastroscope or uroscope, you can pass this through the stoma to view the stomach directly.

If there is bleeding from the gastric tube, it is probably due to irritation from small vessels around the stoma; insert and inflate a larger catheter balloon to tamponade these vessels. If this fails, perform an endoscopy to rule out gastric ulceration, and treat this with cimetidine or omeprazole.

If there is persistent vomiting after gastric tube feeds, or the upper abdomen swells, or undigested food comes out *via* the tube, the tube and its balloon have probably migrated and got stuck in the pylorus. Deflate it, and re-inflate it just after its entrance into the anterior wall of the stomach.

If there is excess granulation around the stoma, apply silver nitrate.

If faecal matter comes out *via* the gastric tube, this is probably because the tube was inadvertently inserted into the stomach through the colon! This requires a laparotomy to disconnect the stomach from the colon, which will not be easy.

If peritonitis develops, there may be a leak into the abdomen from the open stomach, or a perforation of a gastric ulcer, or another cause. Perform a laparotomy.

If necrotizing fasciitis (6.23) **develops around the stoma**, start broad-spectrum antibiotics, resuscitate with IV fluids and perform a wide debridement immediately.

If you find a pneumoperitoneum on an erect chest radiograph (12-2), and there are no signs of peritonitis, there is no indication for surgical intervention.

13.10 Gastric carcinoma

Carcinoma of the stomach presents usually in a male >40yrs with:
(1) Dyspeptic symptoms which may last for months, before he presents with anorexia, nausea, and increasingly severe dyspepsia. The pain lacks the periodicity of peptic ulcer pain, and is not relieved by food.
(2) Vague ill-health, anaemia, and weight loss.
(3) Vomiting 'coffee grounds' (altered blood), or passing melaena stools.
(4) Vomiting after food; a distal gastric carcinoma causes protracted vomiting, like that of pyloric stenosis due to a duodenal ulcer (13.8).
(5) An upper abdominal mass, due either to the carcinoma itself, or to metastases in the liver.
(6) Jaundice, usually due to malignant nodes in the porta hepatis.
(7) Ascites as the result of peritoneal deposits.
(8) Other symptoms of secondary spread.

Gastric carcinoma may take the form of:
(1) a cauliflower type of growth;
(2) a malignant ulcer with raised, irregular everted edges, especially in the distal third of the lesser curve;
(3) diffuse infiltration, either in its antrum, causing pyloric stenosis, or more diffusely ('leather bottle stomach'). Lymphatic involvement and spread to the liver occur early. Late presentation is the norm.
Radiotherapy and chemotherapy are not very useful.

You may not be able to perform a partial or total gastrectomy, so try to:
(1) Make the diagnosis as best you can.
(2) Select out any resectable and potentially curable cases. These are mostly those with a small lesion seen on endoscopy or with a barium meal.
(3) Perform a palliative gastrojejunostomy (13.8), if the pylorus is obstructed. This will make the patient's last days a little more bearable, stop him vomiting, and improve nutrition temporarily.
(4) As always, palliate and comfort him as he dies (37.1).

(a) Examination
Look and feel for:
(1) An enlarged hard supraclavicular (Virchow's) node.
(2) A firm, or hard, slightly mobile, irregular epigastric mass, separate from the patient's liver.
(3) An enlarged and often irregular firm to hard liver.
(4) Signs that the stomach is not emptying normally: visible peristalsis, a tympanitic epigastric swelling, and a succussion splash.
(5) Signs of advanced disease: cachexia, jaundice, and ascites.
(6) Deposits in the rectovesical pouch: feel for a firm, fixed 'rectal shelf'.

(b) Special tests. If there is a firm enlarged accessible node, especially in the supraclavicular fossa, biopsy it.

(c) Radiographs. If possible, get a barium meal. There will probably be a filling defect, or an ulcer, which you can see quite easily on screening. Inhibited peristalsis suggests a tumour.

(d) Ultrasound will often show a mass in the region of the stomach, separate from the liver.

(e) Differential diagnosis is mainly that of 'dyspepsia'. Endoscopy is most helpful.
Suggesting peptic ulceration: a long history (>2yrs); periodic rather than constant pain.
Suggesting non-ulcer dyspepsia: diffuse tenderness, no mass, less weight loss, and a variable appetite.

(f) Management
If you think the tumour might be operable, try to evaluate it endoscopically. If the tumour is on the lesser curve, metastatic spread to lymph nodes occurs early. If the tumour is within 5cm of the gastro-oesophageal junction, excision may still be possible by an abdominal approach.

Before deciding on major surgery, remember that although a partial gastrectomy might be feasible, if you don't manage to remove all the tumour and metastatic nodes, you can only palliate the condition. Therefore *don't attempt gastrectomy unless your goal is clear*: *i.e.* relief of intractable symptoms, stopping haemorrhage, or cure of the cancer.

If there are signs of progressive pyloric obstruction, causing daily vomiting, with no signs of advanced disease (except perhaps metastatic cervical nodes), perform a gastrojejunostomy (13.8). Choose a part of the stomach wall near the greater curvature, ≥5cm proximal to the mass. Make the stoma well away from the tumour, and make it big (≥5cm), in the hope that it will stay open until he dies. Make it on the anterior or posterior aspect of the stomach in front of the colon. Try to refer the patient afterwards for definitive surgery.

If there is dysphagia because of obstruction at the cardia, *don't try to insert a Celestin tube*, as for carcinoma of the oesophagus (30.5), because you may well perforate the oesophagus doing so, and it will be difficult to keep in place. The only feasible option is an oesophago-gastrectomy, which is very major surgery.

(g) Partial (Polya/Bilroth II) gastrectomy
(GRADE 3.5)

PREPARATION. Organize chest physiotherapy. Cross-match 2 units of blood. Empty the stomach with a nasogastric tube.

EXPOSURE. Make a midline incision that extends below the umbilicus; divide the *ligamentum teres* and falciform ligament. Explore the whole abdomen looking for metastases. Assess the mobility of the tumour.

METHOD. Make an opening in the gastrocolic omentum and lift the stomach gently off the pancreas and mesocolon. Clamp and divide the gastrocolic omentum in sections including the left gastro-epiploic vessels and first 2 short gastric arteries on the left side, and the right gastro-epiploic vessels on the right; do likewise with the right gastric vessels in the lesser omentum close to the lesser curve. Avoiding the biliary tree, free the first 1-2cm of duodenum, apply crushing clamps across it, and divide between them. Close the duodenal stump in 2 layers with long-acting absorbable suture.

Lift up the mobilized stomach and apply non-crushing clamps (preferably Lane's) proximally across it, and crushing clamps just distal to these; divide between them. Bring up a loop of proximal jejunum 10-12cm from the duodeno-jejunal flexure so that the afferent loop lies against the lesser curve, and apply non-crushing clamps. Approximate the gastric stump and jejunum and make an end-to-side anastomosis (11-9). Lavage the abdomen, examine the spleen for lacerations and close.

POST-OPERATIVELY. Treat the patient sitting upright in bed, and make sure he gets vigorous chest physiotherapy. There is no evidence that a nasogastric tube is helpful post-operatively. *Don't put one in after the operation,* because you may perforate the anastomosis with it!

(h) Difficulties with gastrectomy
If there is significant bleeding after gastrectomy, place a pack, press and wait 5mins. Look at the spleen: if it is badly damaged, remove it (15.17).

If you cannot close the duodenal stump, insert a Ch20 Foley catheter to produce a controlled fistula.

13.11 Gastric stricture

Swallowing a corrosive causes damage to the oesophagus (30.3) but ingestion of concentrated acid causes intense spasm of the pylorus, allowing the corrosive acid to pool in the body of the stomach. This produces an intense inflammatory reaction and subsequent scarring, resulting in a stricture of the terminal portion of the body of the stomach or antrum. If you use antacids in the acute situation, the acid is neutralized in a highly exothermic reaction which burns the gastric mucosa further. The resulting stricture may take months to develop; the vomiting of gastric outlet obstruction is frequently preceded by heartburn, epigastric pain and anorexia. Later carcinoma develops in the scar.

(a) Examination
Look for a gastric splash in a grossly cachectic patient who has a history of acid ingestion.

(b) Special tests
Barium meal shows a typically distended proximal stomach with a long narrowed stricture extending to the pylorus with complete loss of rugosity and lack of motility (hourglass stomach). Endoscopy will show a stricture not admitting the endoscope.

(c) Management
Because of gross malnourishment, perform a simple proximal gastrojejunostomy (13.8) unless you have done a feeding jejunostomy (11.7) beforehand. *Don't perform a pyloroplasty* because the thickened scarred pylorus does not hold sutures well. *Don't try a gastrectomy unless nutrition is satisfactory;* if gastric carcinoma has developed, it is best to feed first by a jejunostomy and then arrange a partial gastrectomy (13.10) when body weight has been regained.

13.12 Gastric foreign bodies

Most ingested foreign bodies will pass through the pylorus and exit *via* the anal canal, although their passage is often missed unless stools are examined assiduously. However, large, long, sharp or multiple objects may impact at the pylorus. Most of the time these don't result in gastric outlet obstruction, but may cause obstruction in the small bowel. They may not pass if there is pyloric stenosis from another cause. Occasionally they may cause bleeding or even perforation, especially alkaline disc batteries, or potassium tablets.

(a) Bezoars are concretions of ingested material: *trichobezoars*, principally hair (chewed by long-haired girls), or *phytobezoars* (unripe persimmons or citrus fruits). These form a glutinous mass in the stomach which eventually can occupy the whole organ and cause obstruction, as well as bleeding, anaemia and weight loss, mimicking malignancy.

(b) Indications for extraction of gastric foreign bodies
(1) Impacted foreign bodies at the pylorus,
(2) Haematemesis and/or melaena,
(3) Multiple foreign bodies accumulating in the stomach,
(4) Danger of or actual gastric perforation,
(5) Danger of toxic absorption of chemicals (*e.g.* heroin).

(c) Special tests
Plain abdominal radiographs will show metallic objects; remember to take these films just prior to any attempt at removal, because foreign bodies are notorious in moving on! A bezoar may show up as a mottled density, but is often not seen on barium studies because the contrast infiltrates into the bezoar.
Endoscopy confirms the diagnosis.

(d) Management
Allow smooth foreign bodies to pass naturally: be patient and wait 4wks if necessary. *Don't be tempted to perform an unnecessary gastrotomy*.

Endoscopy may not be so easy because you may have difficulty grasping the foreign body, and pulling it out may damage the oesophagus on the way through. It is best if you can pass a protective plastic sheath over the foreign body before pulling it out together with the endoscope *en bloc*,

especially with ingested heroin packets which may rupture on removal, causing sudden absorption of opioid.

Dissolving a phytobezoar is usually possible with oral cellulose, coca-cola, acetylcysteine, or papain; the latter can be given as papaya (paw-paw) fruit, followed by gastric lavage. Metoclopramide 10mg qds or erythromycin 500mg qds help in emptying the stomach afterwards.

If these measures fail, you may need to extract these objects *via* a gastrotomy.

(e) Gastrotomy for extraction of foreign bodies
(GRADE 3.2)
INDICATIONS
(1) Where endoscopy has failed, is impossible or is unavailable.
(2) Gastric perforation.
(3) Trichobezoar.

EXPOSURE. Make sure the stomach is emptied with a nasogastric tube. Make a midline upper abdominal incision. Palpate for the foreign body in the stomach and examine the duodenum and small bowel for further foreign bodies, especially broken-up bezoars; if you find these, try to break them up and push them into the colon. Check if there is any evidence of perforation.

METHOD. Clamp the proximal stomach with a non-crushing clamp. Open the stomach longitudinally proximal to the pylorus, and extract the foreign body, taking care not to cause further damage if the object is sharp. Beware of injury to yourself also!

Close the gastrotomy transversely with long-acting absorbable suture. Continue nasogastric drainage post-operatively till the aspirate is no longer blood-stained. Treat with antacids when eating begins.

13.13 Gastric volvulus

The stomach can twist around its long (organo-axial) or, rarely, its transverse (mesentero-axial) axis if ligaments are lax or absent (13-18). This twisting may be complete or partial. There may be other pathology associated: peptic ulceration, gastritis and hiatus hernia, especially a diaphragmatic defect.

If it is acute, the result is non-productive retching after recent foods had been swallowed. Frothy retching of saliva is typical. Upper abdominal pain is intense, and circulatory collapse occurs early. A nasogastric tube does not usually pass into the stomach.

If it is chronic, there are episodic bouts of crampy upper abdominal pain and retching. There is usually dysphagia and an inability to burp. Gastric peristalsis is noisy after meals, but less so on lying down; the patient may need to adopt strange postures to get his food down. However, usually you will only make the diagnosis after contrast studies or endoscopy.

(a) Special tests
Abdominal radiographs show a grossly distended stomach with a double fluid level on an erect film. (The stomach may have herniated into the chest if there is a diaphragmatic defect, which is common in infants.) An ECG is useful to differentiate from myocardial infarction.

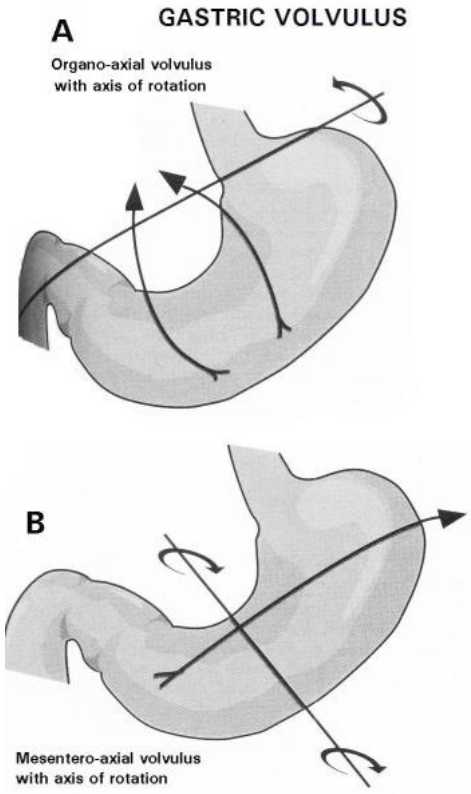


Fig. 13-18 GASTRIC VOLVULUS.
A, organo-axial volvulus. B, mesentero-axial volvulus.
After Morris PJ, Malt RA. Oxford Textbook of Surgery, OUP 1994 p.953 Figs 1,2.

N.B. Barium studies are unhelpful in the acute case. Endoscopy however may be helpful and may allow spontaneous untwisting, but not in the chronic case because the abnormal orientation of the stomach is very difficult to interpret. Barium studies show the greater curve facing superiorly and the body of the stomach assuming a globular shape, if the volvulus is incomplete and some contrast passes into the stomach.

There may be associated motoneurone disease or similar myopathy in the chronic type.

(b) Management
Try passing a nasogastric tube to deflate the stomach: this will buy you time in a chronic or incomplete case.

At laparotomy you may have difficulty seeing the stomach as it is tucked away in the left hypochondrium; you will need to decompress the distended twisted stomach by a needle or small-bore suction tube before you can untwist it.

Simple gastrostomy (13.9) fixes the stomach, but this is not usually a permanent solution.

For organo-axial volvulus, fixing the greater curve to the duodeno-jejunal flexure seems to be successful.

In Tanner's gastropexy, you detach the transverse colon from the stomach, and place the colon under the left hemidiaphragm. Then fix the stomach to the edge of the liver and falciform ligament. A feeding gastrostomy helps to avoid gastric stasis post-operatively.

14 Bowel inflammation & perforation

14.1 Appendicitis

Appendicitis is becoming the commonest abdominal surgical emergency in most of the world, and one with widely variable symptoms. It becomes more common as people discard a high-fibre diet. It can occur at any age, but is rare in children <5yrs.

The disease starts as localized ischaemia of the appendix, probably due to an impacted faecolith; this then either resolves, or complicates with super-added infection, leading to gangrene or perforation. Occasionally the inflammation resolves leading to fibrosis. Sepsis may occasionally spread to the liver, giving rise to portal pyaemia with jaundice and rigors. If peritonitis does develop, the infection can either remain localized or become generalized. If it remains localized, it does so by forming an 'appendix mass' of adherent coils of bowel and omentum. This may then resolve, or suppurate. The distinction between a 'mass' which is not tender or is only minimally tender, and over which there is no guarding or rigidity, and an obviously tender 'abscess' is important, because an abscess needs draining, but a mass can be treated non-operatively.

An abscess may enlarge until it drains spontaneously to the surface, or into the bowel, or into the peritoneal cavity, where it causes generalized peritonitis. It may occasionally become very large but firmly walled off from the peritoneal cavity, giving an appearance like tuberculous ascites.

Appendicitis takes some time to develop, although this may be <6h: you can generally follow its course. Therefore try to work out if the symptoms have been present long enough or too long to fit the clinical picture because the development of an appendix mass or 'abscess' takes several days. Conversely, if symptoms have been present long, but the signs are not impressive, appendicitis is unlikely.

Central abdominal pain is usually the first symptom, and it may be severe enough to disturb sleep. Some hours later the pain moves to the right iliac fossa (or where the appendix is situated: this may be under the liver) and the patient may be able to localize it with one finger. The pain then increases gradually, and is constant. It is now *not* colicky, and is worse on moving, coughing, straining, walking or taking deep breaths (so irritating the parietal peritoneum). The patient moves with caution, and may find it easier to stoop forwards. Lying in bed, he is more comfortable with the right leg flexed. *He does not writhe around in bed.* In a more advanced case, he is almost always anorexic and nauseated. He usually vomits once or twice only, soon after the pain starts. At this stage vomiting is never as severe as in cholecystitis, pancreatitis, or bowel obstruction.

Peritoneal inflammation is responsible for the most important sign of appendicitis: tenderness in the right iliac fossa. Significant rigidity is a sign that peritonitis is spreading.

(a) Presentation occurs at various stages:

(1) **When the infection is localizing as an appendix mass.** The history is likely to be that the symptoms began as above, then the patient began to feel better, the pain improved, and the appetite began to return. He now looks fairly well and has only a mild fever (37·5ºC). The mass in the right iliac fossa is only mildly tender, with no guarding or rigidity.

(2) **When infection is still localized but has become an 'abscess'.** In this stage he is very unwell, anorexic and toxic; there is pain in the right iliac fossa, and a swinging temperature. The abscess may:

(i) be only just palpable,
(ii) be bulging, tender, and fluctuant,
(iii) be in the pelvis, so that you cannot feel it abdominally,
(iv) bulge into the rectum, or the vagina (unusual),
(v) be palpable above the pubis,
(vi) track along the right paracolic gutter to present in the right flank,
(vii) stretch and obstruct loops of bowel.

(3) **Just after perforation,** when the pain of the distended appendix is suddenly relieved, with apparent relief of symptoms, before peritonitis has had time to spread.

(4) **When infection is spreading to cause generalized peritonitis.** There is now generalized abdominal pain, tenderness, guarding, and rigidity. If presentation is very late, there may also be dehydration, cachexia, oliguria, and hypotension with a silent, distended abdomen. All you will know is that there is peritonitis: appendicitis is merely one of its possible causes. If nothing is done at this stage, the patient may become moribund, when the signs of peritonitis may be less obvious.

Try to recognize appendicitis early, before it is allowed to reach the stage of peritonitis or an abscess. The delay of even a few hours can be especially critical in a small child. Experience will teach you when to operate. Some of the alternative diagnoses require operation anyway, and some of those that might be harmed by operation, such as basal pneumonia, *ascaris* infestation, or gastroenteritis, should be easy to exclude. There are always some definite signs in a real case of appendicitis.

In spite of the long list of differential diagnoses that follows, the diagnosis is usually easy. But, remember that:

(1) There may have been no central abdominal pain, so that pain first appears in the right iliac fossa.

(2) There may be no tenderness in the right iliac fossa if the appendix is deep in the pelvis; *you may only find tenderness rectally*: so always do a rectal examination.

(3) The diagnosis is particularly difficult, but no less important, if the patient is very young, very old, fat or pregnant.
Occasionally you may see a patient with appendicitis with an appendix scar, or even with a history of having had an appendicectomy! He may have been misled, or the scar made for a different reason. A significant-sized stump may have been left behind, to become re-inflamed, or very rarely one of a duplicated appendix may have been missed. If the signs indicate surgery is required, *don't hesitate!*

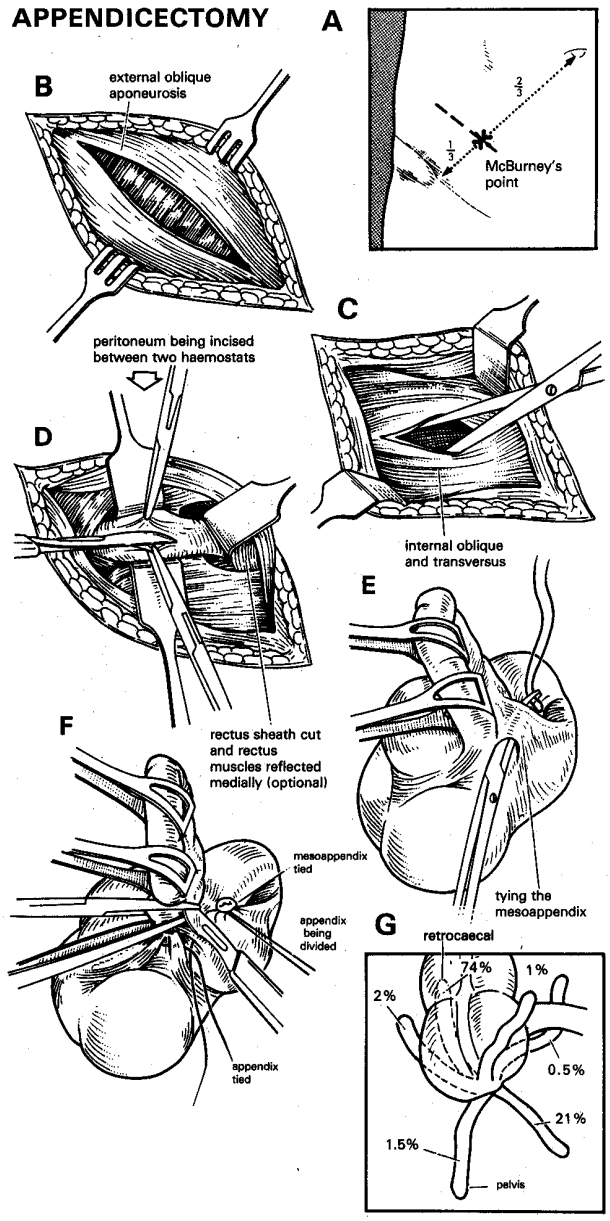


Fig. 14-1 REMOVING AN APPENDIX through McBurney's incision (if the diagnosis is in doubt, a low midline incision is better). A, McBurney's point and the position of the incision. B, split the *external oblique* aponeurosis. C, split the *internal oblique* and *transversus* muscles. D, open the peritoneum between 2 haemostats. This drawing also shows how you can, if necessary, extend the incision medially by incising the anterior rectus sheath, and retracting the rectus muscle. E, pass a ligature through the mesoappendix. F, transfix the appendix base, and remove it. G, various positions where you may find the appendix: 74% behind the caecum, 22·5% in the pelvis. It is seldom anywhere else.
Partly after Maingot R, Abdominal Operations, HK Lewis 4th ed 1961 p.811 Figs. 2,3. with kind permission.

Removing an appendix is usually easy, but is sometimes very difficult:
(1) The appendix may be difficult to find.
(2) It may be difficult to deliver, if it is stuck deep in the wound and is obscured by bleeding.
(3) The caecum may be fragile.
(4) You may find totally different pathology.

Finally: if signs are equivocal, it is reasonable to administer gentamicin and metronidazole and review the situation periodically.

(b) Early history. Ask carefully how and where the symptoms began. How do the other symptoms fit into the story? Most importantly, at which point in the natural history of the disease does the patient find himself now? Remember that a retrocaecal or pelvic appendix may cause diarrhoea or frequency of micturition.

N.B. If vomiting or nausea preceded the onset of the pain, appendicitis is unlikely.

(c) Examination
(1) PULSE AND TEMPERATURE. In the early stages the pulse is normal, and the temperature nearly so. If the pulse is raised, the appendix is probably phlegmonous. A steadily rising pulse is always serious. If there is rigor or high fever within 24h of the onset of symptoms, appendicitis is most unlikely.

(2) INSPECTION. Typically, the lower abdomen does not move with respiration.

(3) TENDERNESS on deep palpation in the right lower quadrant over McBurney's point (14-1A) *is the single most useful sign.* Ask the patient to inflate his lungs: if this causes pain in the right lower abdomen, it is a good sign of peritoneal irritation.

You must, however, examine the whole abdomen systematically with the flat of your hand. Examine the left hypochondrium first. Compare both sides, and the upper and lower quadrants on the right. *Don't dig your fingers into the right lower quadrant.*

If the appendix is behind the caecum, there may be tenderness in the flank. If it is in the pelvis, there may only be tenderness in the rectum, or above the pubis.

If there is spreading peritonitis, there will be tenderness over much of the abdomen.

If you press gently in the right iliac fossa, and then quickly release your hand, this may produce a sudden pain. *This is not so reliable if the patient is fat or pregnant.* This is rebound tenderness, and is a sign of early peritoneal irritation. *It is impossible to have guarding but no rebound tenderness!* A kinder way of eliciting this sign is to test for tenderness to light percussion. This is not so painful and is a better sign.

Try to feel a child's abdomen when he is asleep, or resting on the mother's lap. If he resents any attempt to examine the abdomen, there is probably something seriously wrong inside it. Examine him repeatedly at intervals of 1h, until you have enough evidence to justify a laparotomy.

(4) GUARDING is a sign of local peritonitis. Lay your hand flat on the abdomen, and gently flex your knuckle joints. If there is tightening over the right fossa, the sign is +ve, especially if the patient winces with pain. *Remember to look at his face, not your hand!* Tenderness is relieved by flexing the hip, but worsened by flexing the hip against resistance (10-2).

(5) RIGIDITY is a comparatively late sign, and shows that infection has reached the anterior abdominal wall. Generalized rigidity is a sign of generalized peritonitis (10.1). It is less marked in the obese, emaciated, very old, very young, or those with HIV disease. Advanced peritonitis becomes less tender as the volume of ascitic fluid increases.

(6) AN APPENDIX MASS may be palpable if the symptoms have lasted >2-3 days. With obesity, or a very low pain threshold, it will be difficult to feel. Distract the patient's attention while you palpate.
The mass is ill-defined and is probably an 'abscess' if:
(i) it is tender,
(ii) there is a high fever,
(iii) there are features of intestinal obstruction.
Confirm the presence of pus by aspirating with a wide-bore needle.

Don't assume a mass in the right iliac fossa is an appendix mass: it may be an adnexal mass, an intussusception, a mass of *ascaris* worms, an amoeboma, a caecal carcinoma, a lymphoma, an ileocaecal tuberculoma, an ileal phytobezoar, a bilharzioma, or due to *actinomycosis* or *angiostrongyliasis*. *Check the history!*

(d) Rectal (or vaginal) examination: *never forget this to feel for tenderness or a mass:* the inflamed appendix may be dangling in the pelvis. A rectal examination will often distinguish salpingitis, and a right-sided ectopic gestation. Slowly pass your half-flexed, well-lubricated index finger into the rectum (use your little finger in a child <10yrs). When it is completely inside, keep it still for a moment. Wait for the patient to relax, then gently press anteriorly, posteriorly, and on each side on the pelvic peritoneum with the tip of your finger.

CAUTION! Don't let the patient confuse the discomfort of you putting your finger into the anus, with the pain of you pressing on the pelvic appendix. Wait with your finger in the rectum until the initial discomfort has settled, then rotate and flex the tip of your finger and note the response.

(e) Special tests
A leucocytosis is a useful sign if present, but a normal white cell count may be present in advanced appendicitis with HIV disease. *Alone, without other signs, it is not enough to warrant exploration.*

(f) Ultrasound is helpful in detecting free fluid (38.2l), a mass or lymphadenopathy (38.2h), or the appendix swollen and non-compressible with a diameter >6mm, *but a diagnosis should not rely on an ultrasound report alone.*

(g) Differential diagnosis of appendicitis
This is a long list, but the most important possibilities are the first two, because surgery will make the patient worse:

Suggesting an upper respiratory infection, a viral infection *(mesenteric adenitis)***, or tonsillitis:** upper respiratory symptoms, tachypnoea and alar flaring, generalized muscle aches. All these can cause central abdominal pain in a child. Watch and examine repeatedly, especially the pulse, and if this does not settle, get a chest radiograph.

Suggesting gastroenteritis: diarrhoea, perhaps with vomiting. The pain will be colicky, the tenderness poorly localized, and there may be pus cells in the stool. Be sure to do a pelvic examination, if necessary several times, because a pelvic appendix abscess may be developing. Try to get an ultrasound scan (38.2).

Suggesting amoebiasis: a history of diarrhoea with blood and especially mucus: look for *amoebae* in the stools (14.5).

Suggesting typhoid: a history of fever, diarrhoea, and diffuse abdominal pain for c.3wks, suddenly becoming acute (14.3).

Suggesting ileocaecal tuberculosis: chronic pain which is sometimes colicky, with a general deterioration in health, especially with HIV disease (16.1).

Suggesting a perforated peptic ulcer (13.3): the pain, which is now in the right iliac fossa, *started suddenly* in the upper abdomen; there may be a history of chronic dyspepsia. Enquire for shoulder tip pain. Get an erect chest radiograph and look for gas under the diaphragm.

Suggesting iliac adenitis (6.16): a tender fluctuant mass in the lower quadrant, and a marked flexion contracture of the hip. Look for the primary source of the infection in the legs or perineum.

Suggesting septic arthritis of the hip (7.18): intense pain on any movement of the hip, which is kept flexed. Aspirate from the joint to detect pus.

Suggesting a urinary infection: frequency and pain on micturition, with central lower abdominal tenderness and little guarding. *These symptoms can also be caused by appendicitis, if the appendix lies against the bladder*. Examine the urine.

Suggesting ureteric colic: severe intermittent *colicky* pain radiating into the groin without fever. Test the urine for red cells: these are most often present if there is a stone in the ureter.

Suggesting 'caecal distension syndrome': a result of constipation where the ileocaecal valve is competent, allowing the caecum to swell uncomfortably. Look for constipated stool in the rectum. Although tenderness may be marked, there is no fever and no constitutional upset.

Suggesting caecal carcinoma: an elderly patient with chronic constipation and/or iron-deficiency anaemia. Look for a firm mass in the right iliac fossa.

Suggesting diverticulitis: an elderly patient with a Westernized diet, an irregular bowel habit and episodes of constipation.

In women there are several more possibilities:
Suggesting PID (23.1): pain on both sides of the lower abdomen for ≥72h (rather than 12-36h, as is usual with appendicitis), a history of infertility, and previous pelvic infection. A tender fixed, or occasionally fluctuant, adnexial mass on the right side. A short history with advanced signs of pelvic peritonitis suggests that the mass (a tubo-ovarian abscess) may have ruptured. Examine the cervix for a purulent discharge (23.1). *PID may be impossible to distinguish from pelvic appendicitis.*

Suggesting torsion of an ovarian cyst (23.9): a brief history of acute pain localized to the suprapubic area. A mass palpable vaginally or bimanually. The temperature will not be high.

Suggesting a right-sided ectopic gestation (20.6): a history of a missed menstrual period, signs of hypovolaemia, signs on pelvic examination, and the aspiration of blood on paracentesis. If the ruptured ectopic gestation bleeds more slowly, diagnosis may be more difficult (20.7). Do a pregnancy test and get an ultrasound examination (38.3).

Suggesting ovulatory bleeding: the pain started in the middle of a menstrual cycle *(mittelschmerz)*; mild abdominal tenderness without fever. It will settle in a few hours.

(h) Management of appendicitis
Treatment is usually straightforward.

(1) In the early case, with appendicitis or localized peritonitis, remove the appendix.
(2) Later, with a satisfactorily localizing condition (an appendix mass), and nothing suggesting peritonitis, an abscess or obstruction, treat non-operatively and observe (see below).
(3) If the history has lasted >3 days, with signs of an abscess which is enlarging, drain it.
(4) If presentation is with general peritonitis, resuscitate and treat (10.1) with vigorous resuscitation and damage-limitation laparotomy if the condition is very poor.
(5) If there is no toxaemia, it might be best not to operate in the middle of the night (especially if the numbers of nursing staff are low then) but treat with antibiotics and operate first thing in the morning.

CAUTION!
(1) Infection is less likely to localize at the extremes of life, so *don't be too conservative in the very young or very old*.
(2) In pregnancy, appendicitis does not localize: the danger to the foetus from untreated appendicitis is far greater than surgery, even in the first trimester. The appendix is pushed upwards by the gravid uterus, so tenderness may be high up. Hyperemesis may be confusing.
(3) *Don't try to remove an appendix if it is very adherent and you are afraid of damaging bowel*. Leave it, and insert a drain: it will probably resolve.

(i) Laparoscopy. If you cannot make a diagnosis, you might be tempted to use a laparoscope (19.5) to help. *Don't rely on the findings unless you are very experienced with this technique.* You may easily miss some relevant pathology. However, it may give you a definite indication to proceed surgically, but it already commits the patient to an operation!

(j) Non-operative treatment for appendix mass
(1) **Indications**
An appendix mass with no signs of infective spread. *This is not advisable in children <10yrs, because the omentum is too short to wall off the appendix, nor in the elderly.*

(2) **Method**
Monitor with the greatest care. Treat with metronidazole and gentamicin, and restrict oral intake. Rely on the patient's own assessment of himself, especially with such questions as "Is your pain still subsiding?", "Can you move about more freely?", and "Has your appetite improved?". Monitor the temperature, the pulse, and the white blood count. Palpate the mass gently, and mark its outline on the abdominal wall daily with a felt pen. Allow fluids only by mouth when he starts to improve, then after a day or two, a light diet. Stop the gentamicin, and use metronidazole orally for 3 more days. Check that the mass continues to shrink and that improvement continues. Review in 1wk *but only remove the appendix if symptoms recur.*

(3) **Abandon non-operative treatment if**:
(i) the pain gets worse, or the patient begins to feel generally worse
(ii) the mass enlarges, or just does not shrink
(iii) the abdominal tenderness increases and peritonitis develops
(iv) signs of intestinal obstruction develop
(v) the pulse rate increases and an abscess develops.
This is a very important sign. A slightly raised temperature is of less importance in the early stages, provided that the pulse is steady or falling. A persistently high or swinging temperature implies the presence of an abscess that needs drainage. Any or all of these things show that infection is spreading, so operate for an enlarging abscess, peritonitis, or obstruction.
Remember the danger signs as 4Ps: pain, pulse, pyrexia, and palpable mass.

CAUTION! Non-operative treatment is only applicable with surveillance in hospital.

(k) Operative treatment for appendicitis
(1) APPENDICECTOMY (GRADE 3.1)

This describes the operation where appendicitis is early, and inflammation is located to the right iliac fossa. Where this is *not* the case, and especially when you find a mass when the abdomen is relaxed under GA, or are not certain of the diagnosis, particularly in a woman, perform a formal laparotomy through a midline incision (10.1) because access is better and you can wash out the abdomen more satisfactorily: *this is crucial.*

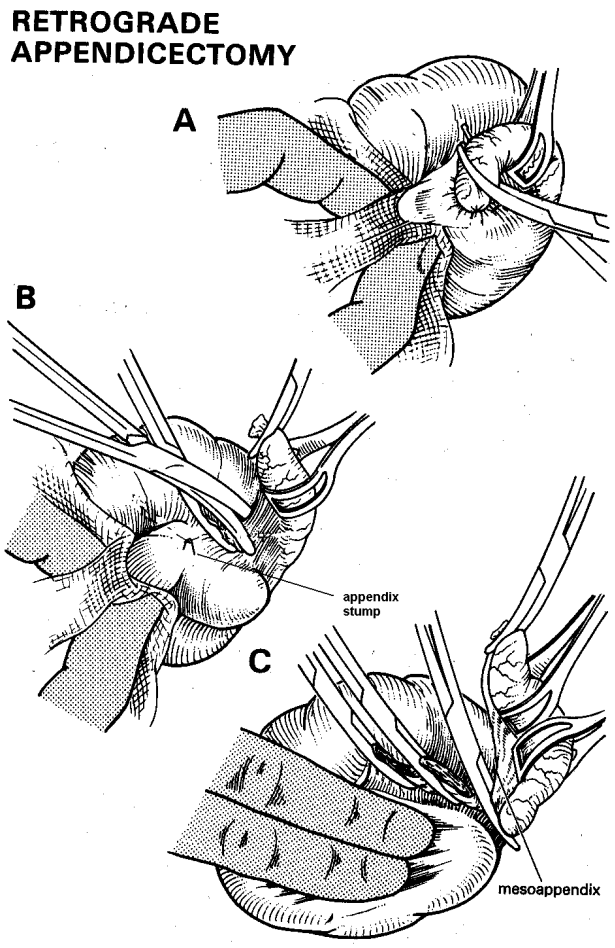


Fig. 14-2 RETROGRADE APPENDICECTOMY.
A, free the proximal end of the appendix from the caecum and transfix it. B, divide it proximally and release it distally from the caecum. C, it is very nearly free. *N.B. This requires adequate exposure.*
Partly after Maingot R. Abdominal Operations, HK Lewis 4th ed 1961 p.816 Fig 7 with kind permission.

PREPARATION. Infuse Ringer's lactate or saline IV. Treat with oral or rectal metronidazole. Have suction ready. Make sure you have an assistant.

INCISION
Centre a 4-5cm slightly oblique skin incision at the point of maximum tenderness and resistance. This may be at McBurney's point (⅔ the way from the umbilicus to the anterior superior iliac spine), or it may not. *(For example, it is in the left iliac fossa in a case of situs inversus!)*
If you are not experienced, *don't try to operate through a keyhole incision,* and *don't site your incision too low* (to be hidden by a bikini!).
Remember, in a pregnant woman, the appendix is pushed much higher up, depending on gestational age.
Split the external oblique muscle aponeurosis in the line of its fibres, which is the same as that of the skin incision, over its whole length. Resist the temptation to extend the incision too far medially. Get an assistant to retract.
Hold the edges of the oblique aponeurosis aside with haemostats, and you will see the fleshy fibres of the internal oblique running transversely, and a little upwards. Insert a closed pair of blunt scissors between them and use the 'push and spread technique' (4-9) to separate them. Then extend the incision with your fingers. Replace these by retractors, to expose the *transversalis* fascia and peritoneum. Pick these up as a single layer and separate them in the same way. Open the peritoneum between haemostats (11-2), making sure you have not inadvertently picked up bowel.

EXPLORING THE ABDOMEN FOR ACUTE APPENDICITIS
Raise the edges of the peritoneum with retractors and look inside. Some exudate may escape. It does not indicate peritonitis, unless it is obviously purulent and foul-smelling. Suck it away using a Poole's sucker. If you cannot see the caecum, it is probably lateral to your incision, or is covered by small bowel. Search for it by sliding a finger into the paracolic gutter. If there is then much fluid coming out, suck it away. Try to feel for the appendix and lift it gently out if it is mobile; if not, retrace the appendix to its base and so locate the caecum. If you pull out small bowel, or sigmoid colon, replace it and try again: *don't keep pulling on small bowel hoping to get to the caecum!*

If you have difficulty finding the appendix:
(i) Look for the pink to grey-blue caecum first. It is often higher than you expect, and always lies laterally; it may unusually lie under the liver. The 3 *taeniae coli* of the caecum converge on the appendix, which lies normally on its posteromedial side. Follow the anterior *taenia* to its base. The tip of the appendix may lie under the caecum, or in the pelvis. With your index finger, feel for something worm-like, tense and rigid.
(ii) Retract the wound edges a bit more.
(iii) Extend the incision.
CAUTION!
(i) If there is localized peritonitis, take particular care not to spread the infection.
(ii) *Don't mistake the sigmoid or transverse colon for the caecum:* the transverse colon has greater omentum attached along its anterior surface.
(iii) Break down as few fibrinous adhesions as you can.
Put your finger under the anterior *taenia* and test the mobility of the caecum. If the tip of the caecum is free, it and the appendix should come to the surface easily. Hold the caecum with Babcock forceps and grasp it with a moist pack, and gently drag its lower end into the wound.

The appendix should follow. *Don't rupture it*, and use the minimum of force: you may be able to lift it out gently by finger dissection. Try to keep the appendix away from the wound edges.

If omentum is folded round the appendix, try not to separate it. Instead, tie it, and remove the adherent part with the appendix.

If you need to extend a McBurney incision:

(i) extend the muscle splits; or

(ii) cut across the muscles supero-laterally; or

(iii) cut into the rectus sheath medially (14-1D), *taking great care not to cut the inferior epigastric artery*, which runs vertically on the deep surface of the rectus muscle.

CAUTION!

(i) *Don't try to work through too small a hole*.

(ii) If you cannot proceed satisfactorily, make a midline incision *(and learn from your mistake next time!)*.

If you have been able to deliver the caecum and appendix into the wound, hold the appendix with a Babcock forceps round it away from the skin edges. Clip, ligate and divide the vessels in the mesoappendix. Pass an absorbable suture through the base of the appendix to transfix it (14-1F), and ligate it firmly. *(This avoids a ligature falling off.)*

If the appendix has stuck in the pelvis, or behind the ileum, and is surrounded by a small abscess, improve exposure by retraction, and by extending the wound downwards. Pack the area off with swabs, and cautiously free it by sharp or blunt dissection.

CAUTION!

(i) If the mesoappendix is very inflamed, *don't apply artery forceps* but pass a ligature round it.

(ii) Be patient and gentle when you try to remove a tense, unruptured, gangrenous appendix: if it is on the point of bursting, try to deliver it intact. (If it bursts, you will greatly increase the chances of infection.)

If the appendix is stuck down behind the caecum or colon, it may be held by fibrous tissue, making it impossible to free with your finger. Extend the incision upwards and laterally by an oblique cut through all layers of the abdominal wall to get better access. Now expose the caecum and, using scissors, carefully divide the peritoneal reflection on the lateral side of the caecum, using the 'push and spread' technique. Using a swab on a sponge-holding forceps, mobilize the caecum medially. Grasp it with a swab, and gently draw it up and out of the wound. Then, work your finger in the plane posteriorly. Tie off the base of the appendix first (14-2) and then remove the rest.

If the appendix has perforated, there is a 90% chance that there is a faecolith somewhere, either in the abdomen or the appendix. Faecoliths are calcified, and may show on a plain radiograph. Try to find and remove it: if you fail, insert a drain.

LAVAGE. Make sure you wash out the abdomen as thoroughly as you can with warm fluid; continue till the fluid aspirated is clear.

CLOSURE. Close the peritoneum with a running suture. It helps to keep straight forceps on the peritoneal edges and ask your assistant to hold them up: be careful you don't put a suture through bowel. If bowel keeps coming out through the wound, ask the anaesthetist for more relaxation. Bring the edges of the muscles together with a few stitches, if you have divided them. Close the external oblique with continuous absorbable, and the skin with subcuticular suture, or if you have removed a really dirty contaminated appendix, leave the wound open, and close it secondarily.

DRAINS are not usually indicated. They are much less important than sucking out and washing out the infected area at the time of surgery. It was thought that it would provide a tract for a fistula if one did form, but it actually may *be its cause.* If you had to leave the appendix (or part of it) behind or there was serious infection and abscess formation, and the patient is not improving, it is better to re-open the abdomen rather than rely on drains.

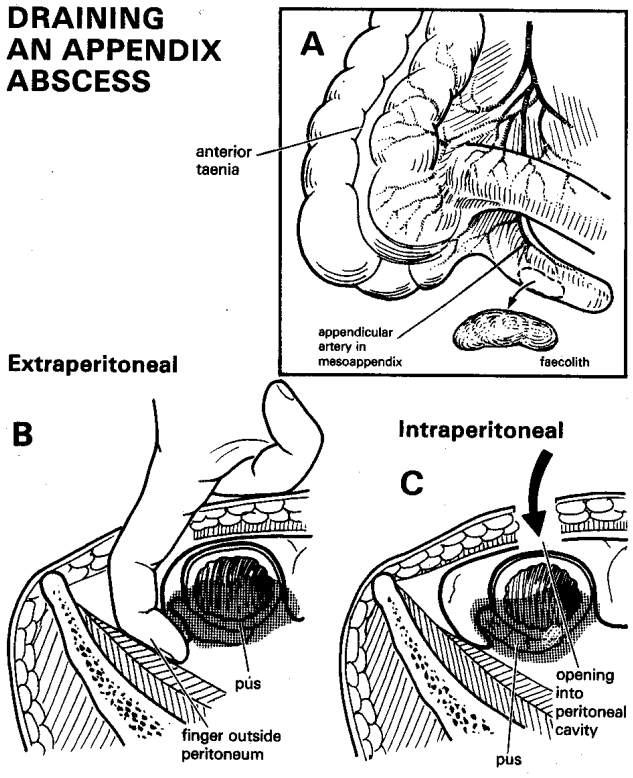


Fig. 14-3 DRAINING AN APPENDIX ABSCESS.
A, anatomy of the appendix. If you cannot find the appendix, follow the anterior *taenia* of the caecum down to it. Sometimes, a faecolith forms in the appendix. If this escapes into the peritoneal cavity, it may be the cause of a persistent abscess. B, approach an abscess extraperitoneally. C, avoid the intraperitoneal approach, unless you happen to come across an appendix abscess unexpectedly during laparotomy.

(2) DRAINING AN APPENDIX ABSCESS (GRADE 2.3)

INDICATIONS. A tender mass which is increasing in size with a history of appendicitis, or more rarely following appendicectomy 3-4 days previously, especially if there is increasing pain, pyrexia, and toxaemia.

THE EXTRAPERITONEAL APPROACH is best. If the abscess is dull to percussion, there is no bowel between it and the abdominal wall. It has probably stuck to the abdominal wall, so that you can easily drain it under LA. You may be able to do this satisfactorily by aspiration under ultrasound guidance; this is preferable to surgery but may mean more than one aspiration to empty the abscess.

Try to enter the abscess, *but not the peritoneal cavity*. Mark the point of maximum tenderness and fluctuation with a felt pen. Anaesthetize and incise the skin and muscles at this point. Try to enter the abscess as far laterally as you can. The muscles will be soggy and oedematous, but you can split them in the usual way, by pushing in a haemostat and opening it. Push a finger in laterally and backwards to make sure that the drainage track is big enough. Suck out pus, break down any loculi, and feel for and remove any faecoliths. Then push a large corrugated rubber drain well in. Suture this to the skin and shorten it gradually after the 3rd day. Remove it completely when it is no longer draining anything.

CAUTION! Don't try to remove an appendix from the bottom of a large abscess cavity with much friable tissue that bleeds easily. Drain the abscess and leave the appendix in place.

If you cannot adequately drain the pus (or there is none present), perform a formal laparotomy through a midline incision.

(3) INTERVAL APPENDICECTOMY

If you have treated an appendix mass conservatively or an abscess by drainage, and left the appendix in, there is only occasionally a need for an interval appendicectomy. However, >80% of appendices in this situation fibrose or disappear by autolysis. Finding the appendix may actually be very difficult. It is probably not indicated in HIV disease. If symptoms of right lower abdominal pain recur, however, appendicectomy should be carried out. If initial histology showed tuberculosis or schistosomiasis in the appendix mass, and proper treatment was provided, interval appendicectomy is unnecessary.

DON'T REMOVE THE APPENDIX AS AN INCIDENTAL STEP DURING ANOTHER OPERATION.

DON'T REMOVE A NORMAL APPENDIX.

(4) DIFFICULTIES AT APPENDICECTOMY

If you find greenish fluid in the peritoneal cavity, it has probably escaped through a perforated duodenal ulcer, and tracked down the right paracolic gutter. Make an upper midline incision and close the perforation (13.3).

If the caecum is much thickened, suspect amoebiasis, tuberculosis, or actinomycosis. Insert a drain and treat with metronidazole. Take a biopsy of adjacent tissue or lymph node.

If you find an appendix mass or the appendix is inflamed, but is so tied down by adhesions that it is difficult to remove safely, insert a drain and close the wound.

If the base of the appendix is necrotic, amputate it because you cannot tie it. This will leave a defect in the base of the caecum: if the margins are healthy, insert a purse-string suture around the edge of the defect and invert it. If the edges are unhealthy, and will not take a suture, make sure you have good enough exposure (if necessary, extend the incision) and remove more of the caecal base till you reach healthy tissue and close the bowel formally. Secure some omentum over the repair, insert a drain adjacent to it, close the muscles of the abdominal wall, but leave the skin open. The alternative, a formal laparotomy and an ileocaecal resection (12.7), is a formidable procedure and, unless you are experienced, may give you more problems.

If the appendix is buried in a mass of adhesions and pockets of pus, avoid spreading the infection. Enlarge the incision, lift its medial side forwards, isolate the mass with warm packs, suck out the pus, and remove the appendix if this is not too difficult. Otherwise, leave it, lavage the abdomen thoroughly and put in a drain.

If you lose the appendix stump or appendicular artery, enlarge the incision and withdraw the caecum into the wound once more to find the culprit. If this fails, open the abdomen formally by a midline incision.

If you find a tumour in the caecum, make a midline incision and perform a right hemicolectomy (12.11).

If you find a tumour in the appendix tip, it is likely to be a carcinoid. No further treatment is necessary apart from the appendicectomy, unless there are metastases in the liver.

If you find a mucocoele of the appendix, make a midline incision and perform an ileocaecal resection, together with a right oophorectomy (in a woman) and excision of any involved peritoneal surface.

If the appendix looks normal, look for other pathology:

(i) If there are enlarged mesenteric nodes, and a clear yellowish serous exudate, suspect mesenteric adenitis, and close. If the nodes are very large or numerous, take a biopsy from one for tuberculosis.

(ii) If you find a purulent exudate, suspect PID in a woman and other causes of peritonitis: make a midline incision.

(iii) If you can feel a tensely distended gallbladder when you pass your finger up through the incision, this may be due to cholecystitis or an obstructed gallbladder. *Don't be tempted to perform a cholecystectomy via a small incision in the right iliac fossa!*

(iv) If there is a tensely distended caecum, there is large bowel obstruction. Make a midline incision and feel for its cause (12.2).
(v) If there is blood in the abdominal cavity, the possibilities include ectopic pregnancy, a leaking ovarian follicle, pancreatitis, trauma, or necrotic bowel: make a midline incision.
(vi) If there is an inflamed Meckel's diverticulum about 1m from the ileocaecal junction, do a bowel resection to remove it. This will probably mean a midline incision.

If you make a formal laparotomy incision, don't remove a normal appendix, but tell the patient the appendix remains in situ*! Don't excise an uninflamed Meckel's diverticulum.*

(5) DIFFICULTIES FOLLOWING APPENDICECTOMY

If shock develops some hours after the operation, suspect that there is bleeding from the appendicular artery, or that you missed an ectopic gestation. Start resuscitation with IV Ringer's lactate or saline, cross-match blood and re-open the abdomen by a midline incision. Suck out the blood. Locate the caecum, find the artery and tie it, or deal with the ectopic gestation (20.6).
If the abdomen distends, with vomiting and ileus, suspect:
(i) Intestinal obstruction (12.15) due to an abscess or to kinking of the bowel. If necessary, drain the abscess, insert a nasogastric tube and manage conservatively (12.16).
(ii) Intussusception (12.7).
(iii) Gram-ve septicaemia and septic shock, with or without generalized peritonitis. If there is tenderness and guarding, re-open the abdomen by a long midline incision and drain the pus. If there is a faecal leak, exteriorize it as a caecostomy (11.6).
If a faecal fistula develops, it will probably heal spontaneously in 2-3wks, provided there is no distal obstruction (11.15). If it persists, suspect tuberculosis, amoebic colitis, actinomycosis, or a retained foreign body (a faecolith or retained swab). Check the HIV status. Exclude distal obstruction by doing a barium enema (38.1f).
If the temperature rises in the 2nd week, accompanied by malaise and local symptoms, there is probably pus somewhere.
(i) There may be a wound abscess.
(ii) There may be a subphrenic abscess (10.2), localized intra-abdominal collection, pelvic abscess (10.3) or a metastatic abscess in the liver (15.10). If there is a mucous rectal discharge or diarrhoea, suspect that there is pus in the recto-vesical pouch. Feel for a boggy inflammatory mass above the prostate, or in a woman's recto-vaginal pouch. Feel also for an inflammatory mass in the abdomen. Get an ultrasound scan (38.2l).
If the wound continues to discharge, there may be a non-absorbable suture left behind. Explore the wound and remove any foreign body. There may be a faecolith left behind. Explore the track, irrigate it and flush it out. There may be: (i) amoebiasis, (ii) tuberculosis, (iii) actinomycosis, or (iv) HIV disease.

If the appendix has a tumour found on histology, ileocaecal resection is only necessary if it was not completely removed or within 2cm of the resection margin.
If the appendix shows tuberculosis macroscopically or on histology, start anti-TB therapy and check the HIV status.
If the appendix shows *schistosoma* eggs, treat with Praziquantel 60mg/kg stat.
If miscarriage follows surgery during pregnancy, remember to ensure that the placenta is also delivered. Evacuation is rarely required.
If a hernia develops in the right lower abdomen, there is probably a small defect in the closure and you should repair this to avoid bowel strangulation (18.13).

14.2 Inflammatory bowel disease

Much inflammatory bowel disease you probably will have difficulty diagnosing; **Crohn's disease** affects the small and large bowel, causing strictures and fistulae, whilst **ulcerative colitis** affects the large bowel and may result in a toxic megacolon like in Chagas disease (12.13). Both may produce loose, blood-stained, mucus-filled stools. They are difficult to treat, needing steroids, sulfasalazine or azathioprine, and IV antibiotics, but fortunately are uncommon in the low- and middle-income countries. Differentiate such diseases from the treatable infective causes of dysentery; a biopsy may be all that you can manage, though you can treat the multiple complex perianal fistulae of Crohn's disease with setons (26.3). Avoid smoking, NSAIDs, opiates and anti-diarrhoeals.

Remember that **schistosomiasis**, **paracoccidiomycosis** and **radiation** >30Gray can also cause inflammatory bowel disease. Generally try to reserve these cases for an expert. Schistosomiasis of all types can cause, in the acute phase, a florid ulcerative enterocolitis with perforation, and with chronic infestation, granulomata, fibrotic strictures and polyps anywhere in the gastrointestinal tract, but especially in the colon. These may result in chronic stricture, intussusception, fistulae and rectal prolapse from persistent straining.

Infective causes are many; typhoid (14.3) is common in endemic areas. ***Campylobacter jejuni*** affects the caecum and causes lymphadenitis in the terminal ileal mesentery; ***Yersinia enterocoli*** affects the terminal ileum with similar lymphadenitis. You may be able to diagnose and differentiate these by ultrasound.
People eating Western diets for long periods often develop diverticular disease, where there are weak spots in the wall of the descending and sigmoid colon due to increased intraluminal pressure, usually on account of chronic constipation. These 'blow-outs' or *diverticula* may become inflamed, or stenosed, fistulate, perforate or bleed just like other causes of inflammatory bowel disease.
Ultrasound may help in the diagnosis: the bowel wall will be >4mm thick and if there is an abscess, you will see a poorly defined hypo-echogenic area adjacent.

Diverticulitis usually settles with eating restriction, and IV metronidazole and gentamicin or a broad-spectrum cefalosporin. If you are confident that an abscess has formed, you may be able to drain it under ultrasound guidance; but if pain gets worse with tenderness and fever, indicating localized peritonitis, perform a laparotomy.

If the affected large bowel is inflamed but not perforated, wash out the abdomen and leave a drain adjacent to the bowel.

If there is severe large bowel inflammation or perforation with often an adjacent abscess cavity, resect the affected segment, make a proximal end-colostomy and close the distal end (Hartmann's operation: 12.9), or fashion a mucous fistula if the distal segment is long enough. This may be difficult and bloody surgery, so have blood cross-matched, and proceed carefully. If you are unable to resect the affected bowel, make the proximal defunctioning colostomy as before, and leave in a drain. The inflammation will usually settle but may form an abscess which needs local drainage.

Diverticular disease occasionally affects the ascending colon, and is then more prone to complications. This is found commonly in Southeast Asia. If localized sepsis develops, wash out the abdomen as above; if this is severe, it is reasonable to perform a primary ileocolic resection.

There is no indication to perform elective surgery for uncomplicated diverticular disease, nor probably for patients who have had several episodes of diverticulitis successfully treated conservatively. However, follow-up with colonoscopy or barium enema to exclude malignancy is wise.

14.3 Typhoid and small bowel perforation

Although typhoid is common in many low- and middle-income countries, perforation seems to occur more commonly in some regions than in others. Where perforation is common (Madagascar, West Africa, particularly Sierra Leone and Ghana), it may be one of the commonest causes of peritonitis. Less often, typhoid can also cause serious intestinal bleeding, cholecystitis, pancreatitis or osteomyelitis.

Typhoid is more common in *Schistosoma* carriers, those with sickle cell disease, and achlorhydria. The disease is seasonal, and is most prevalent in the wet season. It is characterized by high fever (38·5-39·5ºC), headache, confusion, bloody diarrhoea and abdominal tenderness. Bradycardia is only present in ⅓, splenomegaly in ⅓ and hepatomegaly in ¼ of all patients.

A typhoid perforation is seldom dramatic, because loops of diseased bowel stick together, so that the bowel leak remains contained. It is usually difficult to tell exactly when it occurs, unlike the perforation of a peptic ulcer. Often, there may be no specific complaint. The signs will depend on:

(1) how long ago the bowel perforated, and
(2) how localized the peritonitis is.

Exactly the same scenario exists with spontaneous bowel perforation due to HIV or lymphoma (17.6), Crohn's disease, Behçet's disease or lupus (the latter common in Southeast Asia): the symptoms and signs are virtually identical, and the same advice holds true.

You will seldom miss a perforation if:
(1) you examine the abdomen of any patient with typhoid fever and HIV-related abdominal pain twice a day: *perforations which occur in hospital are easily missed.*
(2) you think of it in any case of acute abdominal pain, with signs of peritonitis, during a febrile illness especially with signs of depressed immunity.

If there has been toxaemia and fever, with chronic abdominal pain for 2-3wks, and then sudden worsening of the pain, a typhoid ulcer in the ileum has probably perforated. This usually happens in the 3rd week, but can occur in the 1st week, or during convalescence. The bowel is oedematous and friable, so surgery may be difficult.

If a perforation presents insidiously, and appears to be localized, you might like to opt for a conservative approach, but if the patient deteriorates, surgery will be that much more difficult. If you resuscitate aggressively and operate early, you can reduce the mortality to 3-10%.

N.B. Salmonella typhi is now resistant to both chloramphenicol and ampicillin in many areas. So adjust antibiotic treatment accordingly: quinolones are best. Ceftriaxone and ciprofloxacin are alternatives. When a typhoid ulcer perforates, many different bacteria are released into the abdominal cavity, including anaerobes. *S. typhi* is only one of them, and *not the most aggressive.*
Be sure that the staff of your outpatient department watch for typhoid perforations. There must be little delay between diagnosing a perforation and closing it. The prognosis will depend on timely intervention.

(a) Special tests
Blood culture is useful but the result will arrive after you have had to start treatment. The Widal test is only helpful if there is a fourfold rise in titre. The Diazo test (visible pinkish froth giving a +ve result) with urine is cheap and most reliable.

(b) Diagnosing perforation
Fever and headache at the onset of the illness are followed by vomiting, abdominal pain, and distension. Following perforation, tenderness usually starts in the right lower quadrant, spreads quickly, and eventually becomes generalized. There is usually guarding present, but seldom the board-like rigidity characteristic of a perforated peptic ulcer.
Percuss the lower ribs anteriorly; if there is gas between them and the liver, the percussion note will be resonant (due to the absence of the normal liver dullness).
The bowel sounds may be absent. Hypotension, oliguria, and bradycardia are terminal signs. If possible, culture the stools, if necessary more than once.

CAUTION! The bradycardia and leucopenia of typhoid may occasionally mask the tachycardia and leucocytosis of peritonitis.

If presentation is several days after perforation, *the diagnosis will be difficult*, because abdominal distension will overshadow other signs.

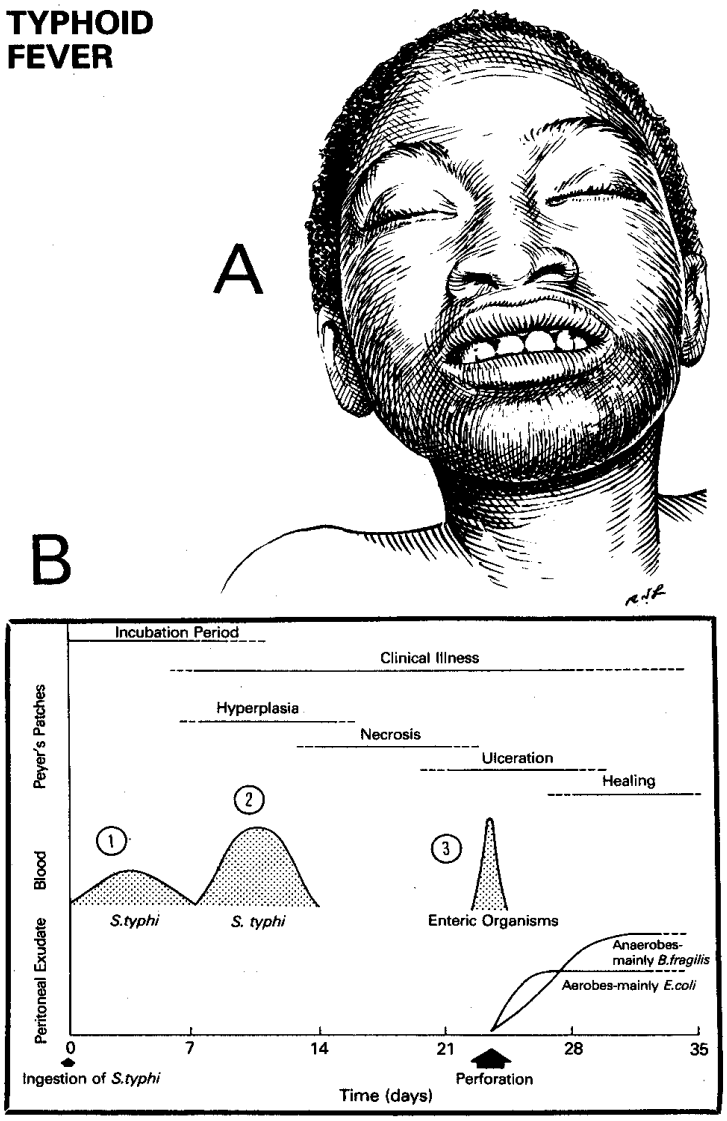


Fig. 14-4 TYPHOID FEVER.
A, characteristic appearance ('typhoid facies') of a patient with typhoid fever. B, bacteriology of intestinal perforation in typhoid fever. (1,2) Note the two phases of *S. typhi* bacteraemia, and (3), the third phase of bacteraemia, with organisms from the bowel, that follows perforation.
After Bitar R, Tarpley J. Intestinal Perforation in typhoid fever. Rev Inf Diseases 1985:7.257-271.

RADIOGRAPHS. Take an erect chest film, and look for gas under the diaphragm (50% +ve). If the patient is too weak to sit up, take a lateral decubitus film, and look for gas under the abdominal wall. *This is a very useful sign*. You may also see loops of the small bowel dilated with gas, usually without fluid levels.

(c) Ultrasound. Look for free fluid!

(d) Differential diagnosis of bowel perforation includes appendicitis (14.1), perforations from other causes, such as HIV disease (5.6), tuberculosis (16.6), ascariasis (12.5), schistosomiasis, paracoccidiomycosis, and necrotizing amoebic colitis (14.5).
Tumours of the bowel may also perforate: in the small bowel these are likely to be lymphoma or Kaposi sarcoma; in the large bowel, adenocarcinoma (12.11). Crohn's disease, ulcerative colitis, colonic diverticulitis, and radiation bowel disease are uncommon causes of inflammation and perforation outside the Western world. A perforated peptic ulcer (13.3) and other causes of a septic abdomen (10.1) such as small bowel obstruction from adhesions also of course give rise to peritonitis.

N.B. Don't forget that unreported trauma may be a cause of bowel perforation, particularly in a child!

Suggesting appendicitis: pain starting over the umbilicus and moving to the right iliac fossa; pain precedes fever.

Suggesting HIV disease: cachexia, lymphadenopathy, absent leucocytosis, and other signs of HIV disease (5.5).

Suggesting tuberculosis: a cough, cachexia, chest radiographic changes and lymphadenopathy.

Suggesting ascariasis: worms seen in the stool or on a radiograph.

Suggesting schistosomiasis: large numbers of ova in the stool in an endemic area.

Suggesting paracoccidiomycosis: lymphadenopathy, mucocutaneous lesions and chest radiographic changes in Central and South American agricultural workers.

Suggesting necrotizing amoebic colitis: a history of diarrhoea (especially with the passage of blood and mucus), followed by acute pain in the right lower quadrant, with guarding and a silent abdomen. Look for *trophozoites* in the stools.

Suggesting a perforated peptic ulcer: a sudden onset, and a history of ulcer symptoms.

(e) Management. Here are some guidelines:
If there are signs of localized or generalized peritonitis, perform a laparotomy.
If the patient is moribund 36-48h after a perforation, with a distended or board-like abdomen, a thready pulse, and very low blood pressure (septic shock), pour in large volumes of warmed Ringer's lactate or saline IV by 2 wide-bore cannulae. When the condition improves, perform a laparostomy (10.1) with copious lavage of the abdominal cavity in the first instance.
If large volumes of melaena stools are passed (***or occasionally frank blood***), transfuse blood to replace the loss. Bleeding will probably stop spontaneously. *Only operate if there is persistent or alarming bleeding.*
Tranexamic acid 1g IV stat and then 10mg/kg/hr is effective in arresting massive bleeding, and worth starting

before rushing to a difficult laparotomy in an unstable patient. Dexamethasone 3mg/kg/day IV may reduce bleeding from inflammatory ulcers, usually in the distal ileum, but which may be difficult to find. If you do find one, perform an ileocaecal resection (12.7), or clamp the segment of bowel that is bleeding, and perform a 2nd look laparotomy after 24h. This is a critical situation.

(f) Resuscitation is critical. Be prepared to rehydrate vigorously. You may need to infuse >4L IV fluid. *Don't forget to add potassium lost (1-2mmol/kg/day)*. If the Hb is <70g/L, transfuse blood. Administer oxygen by mask. Monitor the urine output, and maintain the fluid balance. If possible, monitor the CVP. Add vitamin K 10mg od IM for 3-5 days.

(g) Antibiotics. Treat with IV chloramphenicol (up to 4g qds in very ill patients) with gentamicin 3mg/kg od; or ceftriaxone 1g od; or ciprofloxacin 500mg bd. Decrease the gentamicin dose if there is renal insufficiency. Add metronidazole 1g as loading dose, followed by 500mg tds.

(h) Confusion. Treat with phenobarbitone for a restless patient; avoid sedatives which depress respiration.

(i) Nasogastric suction will empty the gas from the stomach, and, hopefully, diminish the distension of the small bowel. Respiratory complications, particularly the aspiration of stomach contents, before, during, or after anaesthesia are an important cause of death.

(j) Decompress a pneumoperitoneum by needle aspiration in the right upper quadrant (below the liver) if breathing is severely compromised.

(k) Operative treatment

LAPAROTOMY FOR PERFORATED SMALL BOWEL (GRADE 3.4)

PREPARATION. Make sure there is vigorous resuscitation already in process, there is a good urine output, there is a nasogastric tube inserted, and you have administered antibiotics.

INCISION

Make a midline incision, most of it below the umbilicus. As you incise the peritoneum, there will probably be a puff of gas, confirming that some hollow viscus has perforated. *Take care not to cause more perforations*, especially if the bowel is adherent. Aspirate the free fluid. Gently divide the adhesions. Expect to find:

(1) Greenish ileal contents in the abdominal cavity.

(2) In typhoid, the last 60cm of ileum inflamed and oedematous, and the adjacent structures somewhat less so. Also, often an inflamed gallbladder (15.3).

(3) In late cases particularly, dilated loops of jejunum and proximal ileum.

(4) Soft, soggy mesenteric lymph nodes.

(5) Lots of purulent fluid.

Start at the ileocaecal junction, hold the bowel very gently with moist laparotomy pads, and work your way proximally until you reach healthy bowel, or the duodeno-jejunal junction. Look for one or more tiny perforations in the ileum. The jejunum does not perforate in typhoid, but may with HIV. Typhoid perforations are usually on the antimesenteric border of the ileum, not far from the caecum.

Note each perforation you find, until you have found them all. There is usually only 1, and rarely >5. Divide any adhesions very gently by sharp or, if they are soft and thin, by blunt dissection.

CAUTION! Handle the bowel with the greatest possible care: it may come apart in your hands at any moment. If you drop the bowel, start the examination again at the caecum.

Send any fluid for culture in a blood culture bottle. When you get a result, alter the antibiotics accordingly.

If a perforation is small (<1cm), freshen its edges if it is sloughy, by excising 1mm of mucosa all round its circumference, and close it transversely with 'all coats' sutures of continuous or interrupted 3/0 absorbable. *If you put them through only part of the bowel wall, they will cut out.* Invert these 'all coats' sutures with a continuous layer of non-absorbable Lembert seromuscular sutures (11-5E), and wrap some omentum around the closure.

If there is a considerable amount of soiling, and the edges of the perforation are sloughy, *trying to close perforated inflamed bowel will lead to disaster: it will leak!* You should resect the affected segment of bowel, and either make an end-to-end anastomosis (11-7), or bring both ends of bowel out as cutaneous stomas. (If you open the bowel, and it is very distended, make sure you empty it through the cut you have made, outside the abdominal cavity, into a kidney dish, after protecting the abdomen from contamination.) *Wait till the inflammatory process has settled before attempting to re-anastomose the bowel.*

If there is a perforated Meckel's diverticulum, resect the affected segment of bowel: because the inflammation is usually localized (in the area of ectopic gastric mucosa which is present in 50%), you can usually perform an anastomosis.

N.B. Don't be tempted to remove a normal appendix.

If the perforation is jejunal, pass a nasojejunal tube past the perforation for post-operative enteral feeding: if you use a fine tube, this is much more comfortable for the patient, and you can insert a nasogastric tube as well adjacent to it to empty the stomach in the immediate post-operative period. *(This can only be done for proximal small bowel perforations.)*

If there is matted bowel with one or more perforations, which you may not be able to see, and you fear that manipulating the friable bowel will cause worse problems, you can simply drain the area in the hope that a controlled fistula will result.

If there are multiple perforations, or a large perforation, or a severely diseased discrete segment of bowel, or if there is alarming bleeding, resect the diseased segment, and perform an end-to-end anastomosis (11-7). Exteriorize this, because it may well fall apart. An alternative is a mandatory second-look laparotomy (10.1).

If there is reasonable length of normal bowel proximal to the perforation, insert an ileostomy tube (or a Foley

catheter) through it. Fix it in place with a purse-string suture, correct fluid losses post-operatively, and then when the fistula track has matured (2-3wks), remove the tube and allow the fistula to close. *(You can only do this for distal small bowel perforations.)* Alternatively, make a formal proximal defunctioning ileostomy (11.6) to divert the intestinal contents. Do this where *presentation is late, with a localized collection of pus.*

If there are nodes or peritoneal nodules present, take a biopsy of a node or piece of mesentery for TB examination.

Remember there will be much fluid loss, and excoriation of the skin will be difficult to prevent.

If peritonitis is localized, perform a local toilet only, and avoid spreading infection to the rest of the abdominal cavity.

If peritonitis is generalized, wash out the entire abdominal cavity several times with several litres of warm fluid.

CLOSE THE ABDOMEN completely without drains. Leave the skin open, for secondary closure later (11.8). If sepsis is severe, leave the abdomen open as a laparostomy (11.10).

POST-OPERATIVELY. Chronic pre-existing illness and pre-operative metabolic abnormalities will still be imperfectly corrected. Manage as for other kinds of peritonitis. Monitor daily for the early detection of collections of intra-abdominal pus. Continue chloramphenicol (or better, quinolones) at ordinary, rather than high, doses for 2wks. This will help to combat typhoid, but not necessarily peritonitis. Add metronidazole. Fever usually subsides in 4-5 days.

(l) Difficulties with small bowel perforation

Be prepared for:
(1) wound sepsis (11.13),
(2) a burst abdomen (11.14),
(3) intestinal obstruction (12.2),
(4) intra-abdominal sepsis (10.1),
(5) fistulae (very serious, 11.15),
(6) anaemia and difficult nutrition and hospitalization,
(7) respiratory complications (11.12) especially, and
(8) an incisional hernia (18.13).

If you don't find a perforation, there may not be one, and the peritonitis may be primary (haematogenous), or from some other cause. It is doubtful if typhoid ever causes peritonitis without perforation, but primary peritonitis is common especially with HIV.

If there is severe diarrhoea about the 4th day, it will be very difficult to treat, and may be fatal. Replace the fluid loss energetically, and *don't forget to add potassium*.

If there is renewed pain, with post-operative deterioration, suspect that there is another perforation. Perform a re-laparotomy, leaving the abdomen open (11.10).

If there is a sudden spike of fever after about 5 days, when there should have been recovery from the typhoid, suspect wound infection (11.13), a subphrenic abscess (10.2), pelvic abscess (10.3), pneumonia, or an intestinal leak (11.15).

14.4 Necrotizing enterocolitis
(Pigbel, *Darmbrand*)

Like typhoid fever, necrotizing enterocolitis (pigbel or *darmbrand*) is much more common in the tropics, but is occasionally seen elsewhere. It is probably due to the ß-toxins of *Clostridium perfringens* type C which multiply in the bowel following a large meal, classically a feast of pork (hence its New Guinea name 'pigbel'), especially where there is trypsin and chymotrypsin deficiency. This occurs with ingestion of sweet potato especially and threadworm infestation which inhibit trypsin secretion, and protein malnutrition where chymotrypsin levels may be undetectable. The result is a patchy necrosis of the small and/or large bowel.

This was at one time, before vaccination against ß-toxin, the commonest condition requiring laparotomy in New Guinea. It also occurred in Germany in chronically starved people who were given a large meal, hence the term *darmbrand*, meaning 'burning bowel'. A virtually identical condition is seen in HIV patients, and occasionally after feeding through a jejunostomy (11.7). There may be associated mesenteric venous thrombosis (12.14).

If bowel needs resecting, the chances of death are c.50%. This condition in premature neonates is discussed in 33.2.

Presentation is usually in a child, or young adult, with:
(1) Acute toxaemia.
(2) Severe colicky abdominal pain and vomiting.
(3) Constipation with foul flatus, followed by bloody diarrhoea.
(4) Continued vomiting often with blood, and abdominal distension.
(5) An obscure abdominal illness, ending in a pelvic abscess that is the result of a perforation.
(6) Rectal bleeding (26.4).

Typically, the abdomen distends with generalized tenderness, sometimes with a soft mass above the umbilicus. The patient is ill and may have a high fever. There may be erythema of the abdominal wall.

(a) Radiographs. An early sign is intramural gas seen in the bowel; later, a single dilated loop of bowel is highly suspicious; multiple fluid levels on an erect film, with gas in the large bowel down to the rectum, indicate peritonitis. Free gas under the diaphragm indicates a perforation of the bowel.

(b) Special tests. Leucocytosis (unlike the leucopenia of typhoid) is usual, unless there is HIV disease. An abdominal tap may reveal bloody peritoneal fluid.

(c) Ultrasound. Look for free fluid and gas in the portal vein!

(d) Differential diagnosis
Suggesting intussusception (12.7): the presence of a tender abdominal mass and bloody mucoid stools.

Suggesting ischaemic colitis: tests confirming sickle cell disease, or an elderly patient with aortic vascular disease.

Suggesting ulcerative or Crohn's colitis (14.2): a more chronic illness, especially in a wealthier patient.

Suggesting amoebic colitis (14.5): the presence of *Entamoeba histolytica* in the stools.

(e) Non-operative treatment may succeed.

Resuscitate aggressively. Pass a nasogastric tube and treat with large doses of penicillin (6MU IV stat, then 2MU IV qds) and metronidazole IV 1g tds. If the patient is too ill to undergo laparotomy, perform a percutaneous peritoneal lavage. Blood transfusion may be necessary.

N.B. The disease in infants is somewhat different (33.2).

(f) Operative treatment
INDICATIONS
(1) Failure to improve, or deterioration on non-operative treatment.
(2) Signs of peritonitis, and persistently large volumes of gastric aspirate.

LAPAROTOMY FOR NECROTIZING ENTERO-COLITIS (GRADE 3.4)

You may see the disease at any stage in its development. It usually only involves the small bowel, but it may involve the distal stomach, or the large bowel. Classically, several loops of the small bowel, from near the duodenal flexure onwards, are acutely inflamed, oedematous, and congested, often with localized necrotic areas mostly on the antimesenteric border, with a sharp line of demarcation between normal and diseased areas. There may be perforations, localized abscesses, and multiple adhesions causing partial obstruction. The necrotic areas are usually separate, but may occasionally extend from the distal stomach to the sigmoid colon. The mesenteric artery is patent, and you can feel pulsation down to the terminal arterioles at the margin of the affected bowel. The regional nodes are enlarged, and may be necrotic.

Only if any bowel is non-viable (11.3), resect it with an adequate margin of healthy bowel, so that the blood supply to the area of the anastomosis is adequate.

If there is no sharp demarcation of healthy and affected bowel, make a double-barrelled enterostomy (11.5). Wash out any infection liberally. If you have removed a considerable length of the small bowel, follow up carefully, and treat any small-bowel deficiency that may develop with multivitamins, codeine phosphate, and cimetidine.

If you decide to leave inflamed but not obviously necrotic bowel, plan a routine second-look laparotomy in 48h, and resect any gangrenous bowel.

(g) Prevention. In endemic areas, war zones and famine regions, prevent necrotizing enterocolitis by vaccination with attenuated β-toxoid; this gives good immunity.

14.5 Amoebiasis: surgical aspects

Amoebiasis has some surgical complications, ranging from the very acute to the very chronic. They usually involve the bowel, but the liver (15.10), or occasionally the lungs, or even the skin can be involved. Amoebiasis is common, and no age is immune; amoebae may invade the bowel of babies. It is less often seen in women, but in pregnancy it can be fulminating.

Entamoeba histolytica normally lives harmlessly in the colon, but *trophozoites* occasionally invade its mucosa to cause shallow discrete circular or oval ulcers, with yellow sloughs in their bases, and sometimes red edges. These ulcers are most common in the caecum and ascending colon, the sigmoid colon, and the rectum. They cause diarrhoea, with or without blood, pus, and mucus.

The lesions in the bowel are usually quite superficial, but if immunity is low, amoebae may invade more deeply, especially in the HIV+ve, the pregnant, the diabetic, the alcoholic or recently severely injured patient. Invasive intestinal amoebiasis may cause massive mucosal necrosis of the colon, so that large pieces of it separate as casts, and are passed rectally. Alternatively, sloughing, gangrene and perforation result in an acute necrotizing amoebic colitis.

Bacterial infection may then spread as generalized peritonitis, or it may remain localized as a pericolic abscess which you can feel as a tender mass. Peritonitis may develop without actual perforation, or the bowel can perforate extra-peritoneally. As a result of this suppuration, bowel may obstruct, or develop an ileus. Occasionally, the colon bleeds severely, or distends massively as a toxic megacolon.

If amoebiasis is more chronic, there may be:
(1) An amoeboma; this is a diffuse, oedematous, hyperplastic granulomatous swelling anywhere in the colon or rectum, which is often multiple, and may be palpable, and may obstruct the bowel (usually temporarily). Although an amoeboma may form anywhere, a mass in the caecum is more easily palpable. If you do feel a mass in a patient with amoebiasis, it is more likely to be a paracolic abscess than an amoeboma.
(2) A fibrous post-amoebic stricture, which is one of the end results of an amoeboma. An amoeboma and a stricture are two stages in the same process, and there may be a lesion with some of the features of both. Both are common

in some areas (*e.g.* Durban, South Africa), and are the late, chronic complications of amoebic colitis; they occur years after the initial bloody diarrhoea, and are less serious than acute invasive amoebiasis.
The stricture usually involves the rectum (where you can feel it), the sigmoid, and the descending colon, in that order. All can cause diarrhoea and other abdominal symptoms, and obstruct the large bowel, usually incompletely.

(a) Treatment
Treat with metronidazole 800mg tds for 5 days and diloxanide furoate 500mg tds for 10 days. In severe disease, add doxycycline 100mg bd for 5 days.

(b) Invasive intestinal amoebiasis
If amoebae are invading the wall of the bowel, the danger is that it may perforate. If you can make the diagnosis before it has done so, metronidazole will probably be effective. Necrosis of the bowel wall in fulminant amoebic colitis is commoner in pregnancy, HIV disease, diabetes mellitus and the severely injured.

When the bowel has perforated, treatment is much more difficult. There are 3 forms of perforation:
(1) An extraperitoneal ('sealed') perforation.
(2) The perforation of an amoeboma, or an amoebic ulcer, into the abdominal cavity, in the absence of acute dysentery.
(3) A similar perforation in the presence of acute dysentery (this is rare in patients on metronidazole).

Presentation is usually with an 'acute abdomen', but diagnosing that invasive amoebiasis is causing it is difficult pre-operatively.

Typically, there is abdominal pain, fever, diarrhoea, and pain in the right iliac fossa. Often, there is a history of diabetes, alcoholism, pregnancy, or trauma. On examination, you find a mass in the right iliac fossa, or rigidity masking its presence, and often a distended abdomen.

If amoebiasis is endemic from where your patients come, think of this as a likely diagnosis. It is better to start treatment on suspicion, than to miss a treatable disease. If possible, treat non-operatively. Avoid surgery if you can, because the colon will be friable and difficult to suture. Fortunately, surgery is usually unnecessary, because the perforation will probably have been localized by the diseased colon sticking to the surrounding small bowel and omentum.

If there is generalized peritonitis because the perforation has not sealed off, operation is mandatory even though the risks will be great.
At laparotomy you may find:
(1) A large inflammatory mass in the region of the caecum. This is more likely to be a paracolic abscess than an amoeboma.
(2) Greyish patches in the caecum.
(3) Multiple and often adjacent perforations, mostly in the caecum and sigmoid colon.
(4) Inflammatory lesions elsewhere in the large bowel.
(5) A hugely dilated megacolon.
(6) A single stricture usually in the anorectum or rectosigmoid.

SAROJ (45yrs) was admitted with a history of fever, bloody diarrhoea, abdominal pain, and a tender right suprapubic mass. Scrapings from typical amoebic ulcers in her rectum showed *trophozoites*. After only 2 days' treatment with metronidazole, she felt better, her diarrhoea improved, and her abdominal mass started to resolve.
MIRANDA (46yrs) had fever, diarrhoea, and vague abdominal pain for several weeks, worse during the last few days. She had a tender indurated mass in her right lower quadrant. At laparotomy, she had acute necrotizing colitis of her caecum, with multiple perforations, and much sloughing tissue. After a rigorous washout of the abdomen, an ileostomy, and tying off the distant colon, she recovered slowly.
LESSONS (1) Patients often respond to metronidazole rapidly. (2) When surgery is indicated, it is difficult.

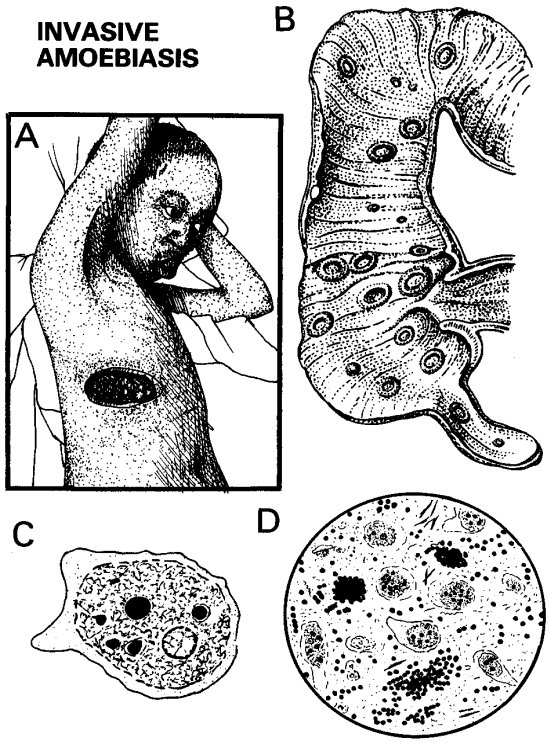


Fig. 14-5 INVASIVE AMOEBIASIS.
A, an amoeboma of the skin secondary to a liver abscess. B, the caecum and ascending colon with amoebic ulcers (seen in cross-section). C, *Entamoeba histolytica.* D, an amoebic stool with *trophozoites*, red cells and Charcot-Leyden crystals.
A, after Bowesman C, Surgery & Clinical Pathology in the Tropics, Livingstone 1960 with kind permission.

N.B. Avoid these mistakes:
(1) *Don't attempt a right hemicolectomy and ileocolic anastomosis*, which is more difficult, and more dangerous.
(2) *Don't try to oversew a perforation*: a necrotic colon will not hold sutures.
(3) *Don't attempt a primary anastomosis*.

If there is severe sepsis, perform damage-limiting surgery. Repairing a perforation is impossible, because the whole colon is usually affected, very friable and adherent to other organs. But, if the bowel has perforated, you must

divert the faecal stream somehow and remove the source of sepsis. Resection and exteriorization is a bloody procedure which is heroic surgery, but it does relieve obstruction, and remove the focus of infection. Ileostomy is the better option initially, combined with drainage of the caecum by a tube; you may need, however, later to remove the affected bowel once there is some improvement in the general condition. Whatever you do, *the danger is that the caecum will burst and flood the peritoneum with faeces, so try to avoid this.*

(c) Special tests
Examine warm stools for *trophozoites*, and look for amoebic ulcers with a sigmoidoscope. Take a scraping and examine it for amoebae. Take a biopsy of the adjacent mucosa and send it for histology. Look for the cysts of *E. histolytica* in the stools. Only some strains are invasive, but unfortunately it takes a sophisticated laboratory to tell which ones.

CAUTION! You will not always find amoebae, so *don't be misled by a negative finding*. If the patient is engaging in anal intercourse, check the sexual partner.

(d) Ultrasound may reveal dilated colon, or a mass in the right iliac fossa (an amoeboma).

(e) Differential diagnosis includes:
Suggesting a typhoid perforation: a 2wk history of fever and vague abdominal pain, which becomes acute when the bowel perforates; intestinal bleeding is uncommon.

Suggesting ileocaecal tuberculosis: often HIV+ve, usually (but not always) less sick than with amoebiasis. The mass in the right lower quadrant is not so large, or tender (unless it has perforated). The course of the disease is usually more chronic.

Suggesting an appendix abscess: pain which starts centrally and then moves to the right lower quadrant; no history of diarrhoea, especially no bloody diarrhoea; less toxic, and not so sick as with amoebiasis. The distinction is not important, because both need a laparotomy.

Suggesting intestinal paracoccidiomycosis: a male agricultural worker in Central/South America with lymphadenopathy and skin lesions, complaining of weight loss, anorexia, headache and fever. Sigmoidoscopic biopsies show granulomas which include *P. braziliensis.*

Suggesting carcinoma of the caecum: not so ill, or so toxic, often with rectal bleeding, which may be occult, presenting with anaemia. The mass is firm to hard, but not particularly tender. Subacute obstruction is more common.

Suggesting ulcerative colitis: the history is usually longer, in a wealthier and better-nourished patient with no *E. histolytica* found despite an extensive search. Previous response to steroids and sulfasalazine is typical.

(**f) Non-operative treatment for invasive amoebiasis**
INDICATIONS. Manage non-operatively if you can, especially if there is:
(1) An amoebic perforation of the large bowel, producing *localized* peritonitis, as indicated by a mass.
(2) A critical illness with prolonged fever, diarrhoea, toxaemia, and peritonitis unfit for surgery.
(3) Simple amoebic appendicitis.

METHOD
Correct the dehydration, hypovolaemia, and oliguria, and especially the hypokalaemia. If there is much blood lost, replace it. Insert a nasogastric suction tube. Monitor the vital signs.

Chronic diarrhoea can cause severe potassium deficiency (resulting in confusion, weakness, hypotension, and ileus) which you can correct simply, and dramatically improve the 'toxic' state. Treat with IV metronidazole 800mg tds, (or 7·5mg/kg tds for a child) and chloramphenicol or gentamicin. Alternatively use metronidazole 1g rectally tds. Mark the outline of the mass on the skin, before you start treatment. Note how tender and indurated it is. Thereafter, examine it 8hrly. If it increases in size, becomes more tender, especially with guarding, perform a laparotomy.

(g) Operative treatment for invasive amoebiasis
LAPAROTOMY FOR INVASIVE AMOEBIASIS (GRADE 3.5)

INDICATIONS
(1) Frank peritonitis.
(2) Failure of non-operative treatment.

PREPARATION
Resuscitate thoroughly, and follow all the steps described above for non-operative treatment: this is critical. Under GA when the muscles are relaxed, examine the abdomen again, and perform a careful sigmoidoscopy. This may confirm the diagnosis, and determine if the sigmoid colon is disease-free. *Take great care yourself not to cause a perforation!*

EXPLORATION
Make a midline incision. Open the abdominal cavity, and examine it as for peritonitis (10.1). Gently feel for a mass. If there are greenish-grey, gangrenous patches on the soggy, soft caecum, your diagnosis of invasive amoebiasis was correct. It may fall apart and leak as you touch it. If the whole colon looks oedematous and inflamed, this may also be invasive amoebiasis, but in an earlier stage.

If there is extensive caecal amoebiasis, perform an ileostomy (11.6). This is easier and less dangerous than trying to manipulate the caecum to exteriorize it. Insert a drain to the right iliac fossa. If the caecum has perforated, aspirate the spilt faecal contents, pack off the rest of the abdominal contents, and insert a large Foley catheter through the perforation into the distal bowel.

If the large bowel has ruptured extraperitoneally (unusual), drain it *via* large tube drains through stab incisions in the flanks.

If you find generalized peritonitis, with no obvious local lesion, lavage the peritoneum thoroughly with warm water. Close the wound, and rely on metronidazole to effect a cure. *Don't try to break off adherent fibrin, which may have sealed off a colonic perforation*.

If the whole colon shows necrotic patches, which look as if they are about to perforate, perform an ileostomy. Plan a second-look laparotomy unless the condition dramatically improves.

If distension is excessive, a toxic megacolon has developed. The bowel is extremely friable and will come apart in your hands, with surprisingly little bleeding. Gently pack away the rest of the abdominal contents, and lift out the diseased colon. You will not be able to use clamps, so be prepared for some faecal spillage and minimize its effects.

Perform a proximal colostomy (or ileostomy) and bring out any distal disease-free portion as a mucous fistula, or close it off. It is probably best to leave the abdomen open (11.8), and close it later.

CAUTION! Remember to lavage the abdomen with plenty of warm water.
Restore bowel continuity after recovery from the acute disease.

(h) Difficulties with intestinal amoebiasis
If there is a mass in the large bowel, *don't forget the possibility of an amoeboma*. This usually responds rapidly to metronidazole, sometimes in only a few days.

If there is a stricture, remember the possibility of post-amoebic fibrosis. You may need to dilate it with your finger, or through a sigmoidoscope. If you cannot do this, you may have to perform a proximal colostomy, or resect the stricture. Because amoebomas and post-amoebic strictures are so rare in some areas, the danger is that you may think that this is a carcinoma. If you are in any doubt, try metronidazole, and take a biopsy.

If you find a regular, firm, sausage-shaped mass in the large bowel, remember the possibility of intussusception (12.7). You may find that it has ulcerated, but the ulcers are unlikely to be amoebic.

If there is severe bleeding from the colon, this may be fatal, because at laparotomy it may look normal externally, so that you will not know where the blood is coming from. Perform a Peep test (26.4). Arrange a colonoscopy if you can. If the bleeding does not stop with IV tranexamic acid and/or installation of cold water, perform a colectomy of the affected segment.

15 Gallbladder, pancreas, liver & spleen

15.1 Introduction

The gallbladder may be diseased due to stones, *ascaris* (15.6), infection *per se*, tumour or volvulus. The frequency of these diseases varies from region to region, but stones are more common in women than in men, and especially in obese, parous women >40yrs (*fat, fertile, female over forty*). Gallstones are found in c.80% of adults with sickle cell disease. Many patients are found at postmortem to have gallstones which have caused no symptoms. *Just because someone has gallstones, they may not be the cause of dyspepsia!* Stones or thick biliary sludge may however pass into the common bile duct and cause biliary colic, or obstructive (cholestatic) jaundice. Stones can promote infection of the gallbladder and cause acute or chronic cholecystitis, although this can arise *de novo* especially with HIV disease. Stones, especially in the form of gravel (sludge), can also promote infection in the biliary tree, especially in association with obstruction (cholangitis, 15.7), though this may also arise *de novo*; they can also obstruct drainage of the pancreas and cause pancreatitis. Rarely stones may fistulate into the small bowel and cause obstruction therein.

You can usually treat acute cholecystitis non-operatively (15.3), but if this fails, you can drain the gallbladder by doing a cholecystostomy. If you are experienced enough, you can treat chronic cholecystitis by removing the gallbladder (15.8). If obstructive jaundice is due to carcinoma of the head of the pancreas, you may be able to relieve the symptoms by bypassing the obstruction and performing a cholecystojejunostomy (15-5), but the situation is more complicated if the obstruction is due to gallstones, *ascaris*, *clonorchis sinensis* (Chinese liver fluke) or tumour obstructing the bile duct.

You can usually treat acute pancreatitis non-operatively (15.13), but a pancreatic abscess (15.15) and a large pancreatic pseudocyst (15.14) need drainage.

You will not be able to remove a pancreatic carcinoma, which may be very difficult to differentiate from chronic pancreatitis. Likewise you will not be able to remove tumours of the liver whether primary (hepatoma) or secondary except with sophisticated equipment, and only when presenting early. However, you will be able to treat hepatic tuberculosis. You may have to drain liver cysts, and may need to remove large hydatid cysts carefully (15.12). You may need to drain liver abscesses especially if they are large (15.10), and likewise splenic abscesses (15.18), which you can usually best deal with by splenectomy. This is also indicated for a number of diseases, other than for trauma (15.17). Splenic tuberculosis is usually only diagnosed after you have removed the spleen!

15.2 Biliary colic

Biliary colic is due to a stone or sludge impacting in the cystic duct. Very rarely it may be due to volvulus of the gallbladder. There is severe colicky epigastric pain which radiates to the right subcostal region and right scapula. The patient wants to bend herself double, she rolls around, and rarely keeps still. Intense pain comes in waves against a background of a dull ache, typically in attacks lasting about ½h, 1-3h after a fatty meal. Pain makes breathing difficult and may be accompanied by nausea and vomiting. Attacks occasionally last as long as 6h. If unrelieved >24h, cholecystitis develops. There may be tenderness in the hypochondrium or the right epigastrium, giving a +ve Murphy's sign (15.3).

ULTRASOUND is a simple, cheap and accurate way of finding stones in the gallbladder, whether or not there is jaundice: they cast an 'acoustic shadow' behind them (38-5). Occasionally you might find *ascaris* in a bile duct (15.6).

N.B. RADIOGRAPHS. *Most gallstones don't show up, so a plain film is unlikely to help.*

DIFFERENTIAL DIAGNOSIS OF BILIARY COLIC

Suggesting ureteric colic: pain radiating towards the groin and genitalia. Blood in the urine on microscopy. Radio-opaque calcifications on abdominal radiographs along the line of the ureter.

Suggesting right basal pneumonia: cough, fever, and lung signs at the right base.

Suggesting upper small bowel obstruction: central colicky pain with profuse unrelieved vomiting.

NON-OPERATIVE TREATMENT OF BILIARY COLIC

Treat with pethidine 50-100mg 3-hrly IV or IM, for 24-48h. Hyoscine 20mg may help.

Restrict to clear fluids only by mouth. If vomiting ensues, replace fluids IV.

Normally, pain will stop in 24-48h, and you can start feeding cautiously, avoiding oily or fatty foods.

If symptoms persist >24h with tenderness in the right hypochondrium, acute cholecystitis has developed.

15.3 Acute cholecystitis

Symptoms are often initially those of biliary colic (15.2), but they last >24h and pain becomes constant. There is a very good chance of recovery in 10 days, even without treatment. There is a 5% chance that

(1) the infection will build up in the gallbladder to produce an empyema,

(2) peritonitis will develop,

(3) a fistula into bowel will occur from a perforation of the gallbladder.

Recurrent episodes of cholecystitis are likely in >50%.

In HIV disease, the gallbladder can be markedly inflamed *without the presence of stones* (acalculous cholecystitis). This is due to *cryptosporidia* or *cytomegalovirus* in 20%, and produces an ischaemia of the gallbladder wall. Infection may also be present with *salmonella*; in typhoid, organisms infect the gallbladder but cholecystitis is often masked by generalized peritonitis. Cholecystitis without stones may also be caused by brucellosis, dengue, leptospirosis and *campylobacter*.

Stones may be in the gallbladder but also in the bile duct and cause partial or complete obstruction with jaundice or cholangitis. Biliary debris at the sphincter of Oddi may result in pancreatitis.

Operate if:
(1) there is cholangitis which is life-threatening,
(2) the gallbladder forms a gradually enlarging acute inflammatory mass,
(3) there are repeated attacks leading to chronic cholecystitis.

The acutely inflamed gallbladder is oedematous, and perhaps gangrenous, and often adherent to surrounding structures, *so don't try to remove it unless you are experienced*. Instead, drain it (cholecystostomy). This may be life-saving and is simple and safe, but it may not cure the disease permanently, so you may have to think of a cholecystectomy later.

N.B. Never try to repair a perforation in an inflamed gallbladder.

Repeated attacks of acute inflammation are usually less severe than a typical acute attack. Symptoms may subside without infection and leave the gallbladder shrunken and fibrosed. However, if a stone remains impacted in Hartmann's pouch (15.4, 15-3M), the gallbladder will distend with fluid (mucocoele of the gallbladder) and if this becomes infected, the subsequent distension with pus (empyema of the gallbladder) may cause it to burst.

SIGNS
The patient is febrile, looks sick, and lies still. There is well-localized tenderness in the right upper quadrant. There may be exquisite tenderness (unlike biliary colic), with guarding and rigidity. Murphy's sign is usually +ve:

MURPHY'S SIGN
Put your hand under the ribs on the right side, and ask the patient to take a deep breath. If he feels pain as the gallbladder moves down on to your hand, the sign is +ve and indicates cholecystitis.

A well-localized mass sometimes forms a few days after the start of the attack, just below the right costal margin. Mild jaundice does not always mean that the common bile duct is obstructed by a stone. If there is jaundice, swinging fever, chills and rigors, however, suspect cholangitis.

SPECIAL TESTS. There is a leucocytosis, unless there is untreated HIV disease. The serum bilirubin and alkaline phosphatase will only be raised if there is obstruction of the biliary tree. The amylase and lipase are raised in pancreatitis.

ULTRASOUND. Gallstones readily show up with an 'acoustic shadow'. With experience you will be able to see if the common bile duct is dilated and if more stones are found inside the duct. The presence of stones may imply cholecystitis, *but does not prove it.* To confirm the diagnosis, you need to see a thickened gallbladder wall and/or fluid around the gallbladder (38.2c).

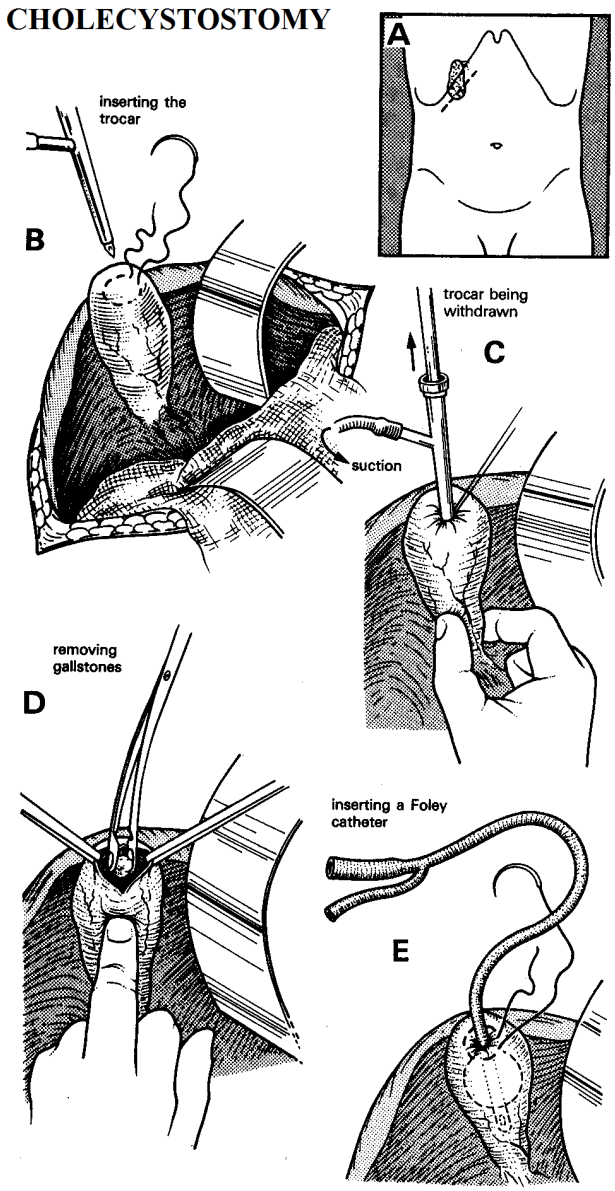


Fig. 15-1 CHOLECYSTOSTOMY.
A, right subcostal (Kocher's) incision. B, insert a purse-string suture. C, aspirate the gallbladder. D, remove the stones. E, insert a Foley catheter.
Kindly contributed by Gerald Hankins.

DIFFERENTIAL DIAGNOSIS OF ACUTE CHOLECYSTITIS
Suggesting liver abscess (15.10): tender hepatomegaly with fever and previous diarrhoea and dry cough.

Suggesting perforated peptic ulcer (13.3): sudden onset of extreme constant pain, with previous dyspepsia.

Suggesting acute pancreatitis (15.13): pain radiating to the back, with alcohol abuse.

Suggesting acute pyelonephritis: pain in the flank associated with urinary frequency, haematuria and previous ureteric colic or *schistosomiasis*.

Suggesting volvulus of the small bowel with strangulation (12.8): initial colicky pain which then became constant, associated with abdominal distension, tenderness and guarding.

Suggesting perihepatitis (Curtis-Fitzhugh syndrome): previous episodes of PID, especially with HIV disease.

Suggesting haemorrhage in a liver tumour: tender knobbly hepatomegaly with weight loss and anorexia.

NON-OPERATIVE TREATMENT FOR ACUTE CHOLECYSTITIS
Make sure of the diagnosis with ultrasound (38.2c) and repeated examination. Perform an OGD (13.2) if you can, to exclude reflux oesophagitis, gastritis or peptic ulceration as differential diagnoses.

Treat pain with enough opioid: *e.g.* pethidine (50-100mg 3hrly). Nasogastric suction is not essential, but it will keep the stomach empty and so relieve nausea and vomiting. Keep nil orally. Correct the initial fluid loss with IV saline.

Antibiotics are less necessary than you might expect, because the inflammation in the gallbladder is primarily chemical. However, they probably reduce complications: treat with chloramphenicol (or gentamicin), ampicillin, or doxycycline. Continue this treatment till the pain and pyrexia settle; then introduce oral fluids and after this allow a fat-free diet. Symptoms should start to improve after 24h, and disappear in 3wks. Advise a low-fat diet, and review after 2-3 months.

If the patient is pregnant, lean towards conservative treatment to allow the pregnancy to reach term or near-term if at all possible.

If symptoms recur, repeat the treatment for acute cholecystitis. When things have settled, think about an elective cholecystectomy, usually six weeks afterwards, if stones are definitely present. *Don't operate for acalculous cholecystitis* unless there is marked tenderness and you fear imminent perforation.

15.4 Empyema of the gallbladder

When cholecystitis gets worse, the gallbladder enlarges and becomes a tense inflammatory mass. This may occur if the cystic duct is obstructed with a gallstone, or secondary to carcinoma. The gallbladder fills with pus and so becomes an empyema; it then may perforate, resulting in septic biliary peritonitis which is frequently fatal.

The patient is sick, pyrexial, lies still and has a painful tender mass in the right hypochondrium below the liver. There may be a known history of gallstones, but usually *not* of jaundice.

SPECIAL TESTS. Leucocytosis progresses from earlier in the disease. Amylase is usually normal.
ULTRASOUND. The gallbladder is filled with turgid fluid, and often gallstones; its wall is thickened (38.2c). Aspiration may relieve some symptoms in a very sick patient, *but is not a lasting solution*.

CHOLECYSTOSTOMY (GRADE 3.3)

INDICATIONS. Drain the gallbladder if:
(1) intense pain persists with swinging fever,
(2) abdominal tenderness gets worse, the area of guarding extends, or the mass increases in size, both suggesting an empyema of the gallbladder,
(3) there is cholangitis with a distended gallbladder (15.7),
(4) the patient is too sick to undergo a cholecystectomy.

ANAESTHETIC. If the patient is very sick or very old, you can operate under LA, especially if the gallbladder is tense and easily palpable under the abdominal wall.

PREPARATION. Make sure your suction is working properly. Treat with gentamicin and metronidazole.

INCISION. Feel for the area of maximum tenderness, an ill-defined mass, or both (15-1A). Centre the incision over this area, and cut through all layers of the abdominal wall 2cm below and parallel to the line of the costal cartilages. You will probably find the gallbladder easily. If you don't find it, carefully separate the adherent omentum and transverse colon by pushing them away with your finger. Pack the rest of the abdominal contents away from the inflamed gallbladder. This will be easier if you tilt the table feet down. *Handle the gallbladder carefully; it easily ruptures and spills infected bile into the peritoneal cavity.*

If the structures below the right lobe of the liver are matted together in an oedematous haemorrhagic mass, so that the gallbladder is difficult to find, insert your hand over the upper surface of the liver, and draw your fingers down until you reach its edge. Then move your hand medially over the convex surface of the liver until you reach the falciform ligament, joining the liver to the diaphragm. At its lower edge is the *ligamentum teres*. About 5cm to the right of this, you should be able to feel the tense, turgid, elongated mass of the fiery-red, acutely inflamed, oedematous, and perhaps partly necrotic gallbladder.

Try to expose enough of the fundus of the gallbladder to allow you to drain it. Use your finger, or a 'swab on a stick' (4-9A), to 'peel' the omentum, the hepatic flexure of the colon, and the transverse mesocolon carefully out of the way. *Avoid sharp dissection*. If there is bleeding, control the haemorrhage with packs.

Put a purse-string suture on the gallbladder fundus (15-1B), incise it and aspirate the pus (15-1C). Then enlarge the opening and extract as many stones as you can (15-1D); if they are very adherent in Hartmann's pouch (15-3M), leave them, rather than perforating the gallbladder. *Don't attempt to explore the common bile duct.*

Place a wide-bore catheter, which you have inserted through a separate stab incision in the lateral abdominal wall, in the gallbladder, and close the opening around the catheter snugly with 2 purse-string sutures, one 5mm away from the other (15-1E). Secure the catheter drain and attach it to a drainage bag. Lavage the abdomen with warm water.

DIFFICULTIES WITH EMPYEMA OF THE GALLBLADDER

If you cannot reach the inflamed gallbladder, extend your incision across the midline as an inverted V.

If you don't find a tense inflamed gallbladder at operation, look for differential diagnoses and act appropriately.

If the gallbladder is not that seriously inflamed, and you are able to do so, perform a cholecystectomy (15.8).

If you rupture the gallbladder at Hartmann's pouch (15-3M) – a mucosal fold between the neck of the gallbladder and the cystic duct, sometimes caused by adhesions between them, where gallstones commonly get stuck (it may be present in both normal and pathological gallbladders) – try to remove any impacted stone (which should be freed up by your manipulations) and, if possible, put a ligature around the cystic duct remnant. Remove as much of the inflamed gallbladder as you can (if the posterior wall is very adherent to the liver, leave it) and insert a wide-bore drain through a separate stab incision in the abdominal wall.

If the gallbladder is so tense and inflamed that a suture causes it to leak profusely, apply suction and remove as much of the inflamed gallbladder as you can, as above.

If the gallbladder has already perforated, there is already effectively a cholecystostomy and friable tissues will make cholecystectomy too difficult. Aspirate septic fluid from the abdomen and pack away the bowels. Remove as much of the gallbladder wall as possible: you can leave the posterior wall adherent on the liver surface. Remove any debris and stones. Try to close the cystic duct with an absorbable suture if you can. Leave a drain brought out through a separate stab incision. Fold some omentum into the bed of the gallbladder.

POST-OPERATIVELY, expect bile to start draining in a day or two. Chart the daily amount of bile draining. Plan to remove the tube in 14 days. If the bile is still discharging after 2wks however, leave the catheter *in situ* for a month at least before removing it. If the bile loss is significant, replace fluid and electrolytes IV. You can try to return the bile to the intestines via a nasogastric tube if the patient will tolerate it. The fistula will slowly close unless a stone has been left in Hartmann's pouch (when a small mucous fistula will result, 15-3M).

N.B. The cholecystectomy needed to cure this problem may be difficult indeed.

15.5 Cholangitis

Ascending cholangitis describes infection in the bile duct, which, if untreated, may be followed by multiple abscesses in the liver, or by septicaemia. IV antibiotics are necessary, but if stones are the underlying cause, the common bile duct should be explored, and any stones removed. This is difficult and needs special instruments and on-table radiography. If it is impractical, and the stone is distal, you may still be able to decompress the common bile duct by inserting a T-tube (choledochostomy), or by opening the duodenum and opening the sphincter of Oddi wide, but this is difficult surgery.

A patient with cholangitis usually has a previous history of biliary colic and cholecystitis. Typically, an attack of colic is followed the next day by fluctuating jaundice, dark urine, pale stools, nausea and vomiting, fever and rigors. The liver may be tender, but the gallbladder is not palpable.

In HIV disease, cholangitis may occur intermittently without the presence of stones, mimicking primary sclerosing cholangitis. In East Asia, liver fluke infestation often causes cholangitis (15.7). In endemic areas, consider hydatid disease.

SPECIAL TESTS. Check if *ascaris* ova are in the stool: this does not necessarily mean that worms are the cause of cholangitis, but strongly suggests it (15.6).

ULTRASOUND (38.2) is very useful to confirm the presence of stones, cysts or *ascaris* and their number and position in a dilated common bile duct.

CHOLEDOCHOSTOMY (GRADE 3.5)

PREPARATION

Treat with IV ampicillin, gentamicin and metronidazole, or substitute ampicillin and gentamicin with a cefalosporin, ciprofloxacin, or mezlocillin.

If he is septicaemic, resuscitate the patient with Ringer's lactate. Add vitamin K 10mg IM. Insert a nasogastric tube. *Don't delay.*

INCISION. Make an upper midline incision and follow the initial steps to find the gallbladder (15.3), then expose the subhepatic area, cystic duct, and common bile duct. (The midline incision is better for exposure of the common bile duct than a subcostal incision.)

Expose the biliary tree as in a cholecystectomy (15.8), but *without grasping the gallbladder.* Make sure you have found the bile ducts before proceeding further. Palpate them to be sure none of them pulsates! If in doubt, aspirate the common duct to make sure it contains bile and not blood. Then expose 2cm of the common bile duct, which will probably be significantly dilated (>5mm) (15-2A), and place two 3/0 stay sutures on its anterior surface about 4mm apart (15-2B). With the tip of the sucker close by, make a longitudinal incision between the stay sutures (15-2C). Suck out all the bile and exudate, and take a swab for culture and sensitivity.

Using Desjardin's stone forceps, gently remove any stones that you can easily see (15-2D). The curve on the forceps may help you: the stones are probably well down the common duct at its lower end, where it enters the duodenum. *Don't prolong this stage of the operation if it is difficult: you can do much harm.* If there is much 'sludge', wash out the common duct by irrigating it with plenty of saline using a plain rubber catheter and a 20ml syringe.

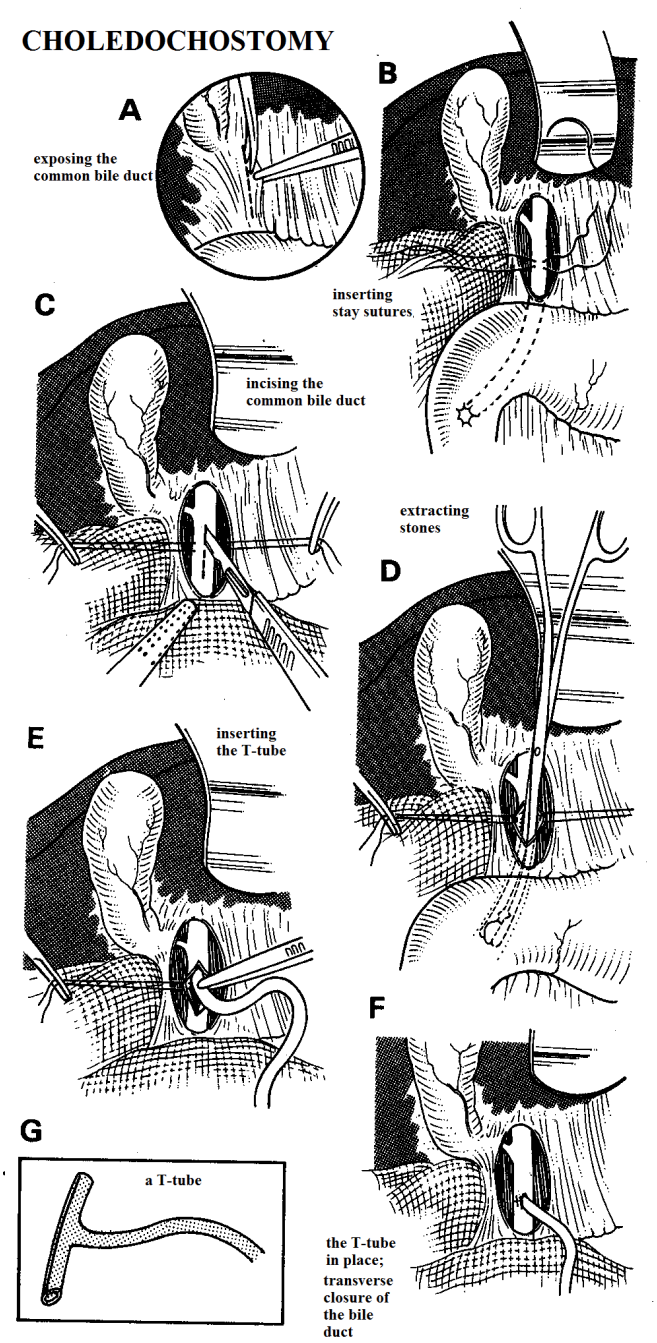


Fig. 15-2 CHOLEDOCHOSTOMY.
A, expose the common bile duct. B, insert stay sutures. C, incise the common bile duct. D, remove the stones. E, insert a Fogarty catheter. F, T-tube sewn in place. G, a T-tube.

EQUIPMENT. *FORCEPS, gallstone, Desjardin's.* Use these long slender forceps for removing stones from the biliary system.

If you don't have Desjardin's stone forceps, a Fogarty balloon catheter pushed past the stone, inflated, and withdrawn is often as effective and less traumatic. You may possibly be able to use a tiny Foley catheter. Find 2 assistants in addition to the scrub sister.

Insert a T-tube (15-2E), and close the opening in the duct snugly round the drainage tube with a transverse absorbable 4/0 suture (15-2F). Bring the tube out through a stab incision, leaving some slack inside, in case it is pulled on. Anchor it securely to the skin with a non-absorbable suture.

(You can make your own T-tube by slitting the end of a piece of ordinary suction tubing, and cutting away ⅓ the circumference of the tubing.)

Close the abdominal wall carefully: the wound is likely to break down or become infected, so consider leaving the skin open (11.10, 11-20).

POST-OPERATIVELY, connect the T-tube to a bedside bottle, and allow it to drain freely until the jaundice and fever subside. Perform a tube cholangiogram 10-14 days post-operatively using 25% sodium diatrizoate ('*hypaque*') or similar aqueous contrast medium diluted 1:2 with 0·9% saline. *Make sure you don't inject any air with the contrast medium.* If you see no stones, and the medium flows nicely into the duodenum, clamp the tube. Provided that there is no pain, fever, or jaundice after 1wk, remove the tube.

If the cholangiogram shows blockage of flow into the duodenum or any residual stones as filling defects, try flushing the duct with 1L of warm saline suspended 1m above the patient, after treating with hyoscine 20mg IM. If this fails, check what pressure builds up. It should not be higher than 8-10cm of water. If after 24h no higher pressure develops, try clamping the tube. Remove it after 2wks if no discomfort develops. If pressure does build up in the tube, *don't remove it.* You may then be able to remove residual stones by dilating the T-tube tract and pulling them out with endoscopy forceps (the Burhenne technique), or they can be removed by an expert by passing a side-viewing fibre-optic endoscope into the duodenum and slitting the sphincter of Oddi, or by opening the duodenum at laparotomy.

TRANSDUODENAL ODDI SPHINCTEROTOMY (GRADE 3.5)

INDICATION. When a stone is impacted at the distal end of the common bile duct; when antegrade extraction or lavage has failed to dislodge a stone.

INCISION. Expose the biliary tree as before; then mobilize the duodenum by the Kocher manoeuvre (13.5). Make a 4cm longitudinal incision in the lateral surface of the duodenum at the junction of first and second parts, and feel the papilla with your finger through the duodenotomy. If you can't find it, pass a bougie or catheter down through the common bile duct. Then insert a fistula probe into the papilla and open it upwards with a #11 blade (4-1) to free any impacted stones. When you see bile flowing freely, you know you have relieved the obstruction. Carefully spatulate the edges of the papilla open with absorbable 4/0 sutures. Be careful not to damage the pancreatic duct (usually visible at the 5 o'clock position). Close the duodenotomy transversely in 2 layers with long-lasting absorbable 2/0 sutures, and cover this with omentum if possible.

If the bile duct has been opened, place a T-tube as above.

15.6 Cholangitis caused by *ascaris*

Ascaris worms sometimes crawl up into the common bile duct and gallbladder, where they can cause biliary colic, acute cholecystitis, obstructive jaundice, cholangitis, and pancreatitis. This most often happens when a child has been given an antihelminthic. So, if a child has cholangitis, or if an adult does not fit the usual clinical picture for biliary disease, suspect ascariasis. Finding *ascaris* ova should arouse your suspicion, but *does not confirm the diagnosis*. Get an ultrasound scan. *Don't operate*, except on the indications below.

Nasogastric suction will empty the upper intestinal tract. Systemic antibiotics will help to control the cholangitis. Later, treat with levamisole 120mg stat, or piperazine 4g stat, or if there is multiple parasitic infestation, mebendazole 100mg od for 3 days, repeated after 15 days.

N.B. Dead ascaris *worms may still block the bile duct!*

SPECIAL TESTS. There is a microcytic hypochromic anaemia, a leucocytosis with >50% eosinophilia; bilirubin is raised in cholestatic obstruction and amylase in pancreatitis. *Ascaris lumbricoides* eggs can be found in the stool.

ULTRASOUND is best to determine if the *ascaris* worm is no longer in the bile duct (38.2c).

INDICATIONS FOR SURGERY. Deepening jaundice, spiking fever, chills and rigors which don't respond to antibiotics, nausea and vomiting, toxaemia, dehydration, tachycardia, and perhaps hypotension; together with a leucocytosis. If there are these symptoms, explore and drain the bile ducts (choledochostomy, 15.5) after appropriate preparation. Remove any worms you find. Avoid a sphincterotomy as *ascaris* may then more easily crawl into the pancreatic duct subsequently.

15.7 Other causes of cholangitis

(1) In East Asia, liver flukes, *opisthorchis*, found in fish and snails, are extremely common. Many thousands of flukes can live for many years in the bile ducts causing inflammation. Resulting fibrosis leads to stricturing and dilation, secondary bacterial infection and stone development. Recurrent cholangitis can lead to biliary cirrhosis, liver failure, hepatorenal syndrome, portal hypertension or septicaemia, as well as pancreatitis. Recurrent inflammation may result in cholangiocarcinoma, and chronic infestation can lead to a *salmonella* carrier state if secondarily infected.

Presentation is usually at 20-40yrs (males more commonly), initially with non-specific malaise but then with a high swinging fever, chills and rigors, a gnawing right upper abdominal pain, and mild jaundice (Charcot's triad), usually with a history of previous attacks. The liver is tender and enlarged and the gallbladder may be palpable. The urine is dark, but the stools are seldom clay-coloured; complete obstruction of the common bile duct is rare.

(2) Around the Mediterranean, in East Asia and in Latin America, sheep liver flukes, *fasciola hepatica*, are found. These flukes are large and tend to remain in extra-hepatic bile ducts. The flukes may actually eat into the wall of the ducts, which leads to haemobilia, presenting as haematemesis and melaena, or more rarely perforate the duct, causing peritonitis. Inflammation leads to similar complications as with *opisthorchis*, but because the extra-hepatic ducts are preferentially involved, gallbladder distension and empyema are more common.

(3) Primary sclerosing cholangitis is an inflammatory condition affecting both intra- and extra-hepatic bile ducts, in men and women equally of 25-40yrs. Fatigue, weight loss, right upper abdominal pain, intermittent jaundice and itching are usual; an acute attack of cholangitis is rare. A similar picture can arise in HIV disease (5.6), with or without papillary stenosis. No medication has been found helpful.

(4) Rupture of a hepatic hydatid cyst into the bile ducts (15.12).

SPECIAL TESTS
There is a leucocytosis (and eosinophilia with fluke infestation); the serum bilirubin and alkaline phosphatase are raised. If infection is severe and liver cells are involved, the transaminases are raised. Measure the serum amylase, because there is a 10% chance that there is also pancreatitis. You may find ova and dead flukes in the faeces and in duodenal aspirates.

RADIOGRAPHS
A plain radiograph may show air in the biliary tract due to an incompetent sphincter of Oddi.

ULTRASOUND often shows dilation of intra-hepatic before extra-hepatic ducts, and may actually detect mobile flukes (38.2c).

NON-OPERATIVE TREATMENT
If the disease is mild, take blood cultures and treat with antibiotics (cefradine or gentamicin, 2.8). Add vitamin K 10mg IM. Start intravenous fluids, restrict oral fluids and aspirate the stomach through a nasogastric tube. Praziquantel 25mg/kg tds for 2 days is the most effective treatment for *opisthorchis* but is ineffective against *fasciola*, for which bithionol 1g tds alternate days for 5 days is 100% effective.

INDICATIONS FOR OPERATION
(1) Failure of non-operative treatment.
(2) A palpable, tender, enlarged gallbladder.
(3) Septicaemia.

LAPAROTOMY. Aim to remove all biliary debris by washing out the extra- and intra-hepatic bile ducts with copious amounts of saline. Prepare the patient (15.5). Perform a choledochostomy (15.5), and insert a T-tube. In the presence of septicaemia and an enlarged gallbladder, perform a cholecystostomy (15.3). When acute symptoms have settled, treat any liver flukes.

15.8 Cholecystectomy (GRADE 3.3)

Removing the gallbladder is the standard method of treating chronic gallbladder disease, but it is not an operation for the occasional surgeon, so unless you are experienced, it is better to treat cholecystitis non-operatively (15.2,3). However, if symptoms and signs get worse, and you cannot refer the patient, and have sufficient experience, it is best to operate early on an acutely inflamed gallbladder than later when it becomes hopelessly stuck down.

If symptoms persist, consider the option of cholecystostomy (15.4) before deciding on cholecystectomy, which can be difficult and where the complications can be serious. The main dangers are bleeding and injuring the common bile or hepatic ducts. *Don't try to remove a fibrotic, contracted gallbladder.* Unfortunately, you will not be able to predict if the operation is going to be easy or difficult. So, be prepared to bail out: abandon the operation, or limit yourself to a cholecystostomy after all.

We describe 2 methods of removing the gallbladder: (i) the retrograde, in which you first dissect and tie its neck, and (ii) the antegrade, in which you start at the fundus.

The commonest cause of an injured bile duct or hepatic artery is an 'easy' operation done quickly. Another cause is anatomical variability (15-3).

ELECTIVE CHOLECYSTECTOMY

INDICATIONS
(1) Gallstones causing several attacks of cholecystitis.
(2) A carrier of *salmonella typhi.*

CONTRAINDICATIONS
N.B. These are relative, but important:
(1) Inexperience.
(2) Uncertain diagnosis and failure to exclude other causes of dyspepsia.
(3) Insufficient symptoms and signs which justify the operation.
(4) Complicating factors, *e.g.* HIV disease or excessive obesity.
(5) The need to bail out, or perform a cholecystostomy, if it is too dangerous to proceed.

ANTIBIOTICS. The main cause of death in gallbladder surgery is post-operative sepsis. Use a perioperative antibiotic (2.9) unless the case is a completely elective one.

PREPARATION
A self-retaining and a Deaver's retractor are almost essential. You will need two assistants as well as the scrub nurse. If you have facilities for radiography in theatre, make sure the patient lies on a suitable table so that radiographs can be taken of the upper abdomen. Check for sickle cell disease if this is common in your area.

INCISION. Make a right subcostal (Kocher's) or midline incision (11-1) extending up to the costal margin.

The Kocher's incision gives better access to the gallbladder itself, but the midline incision better access to the bile duct, and any other pathology that may be present.

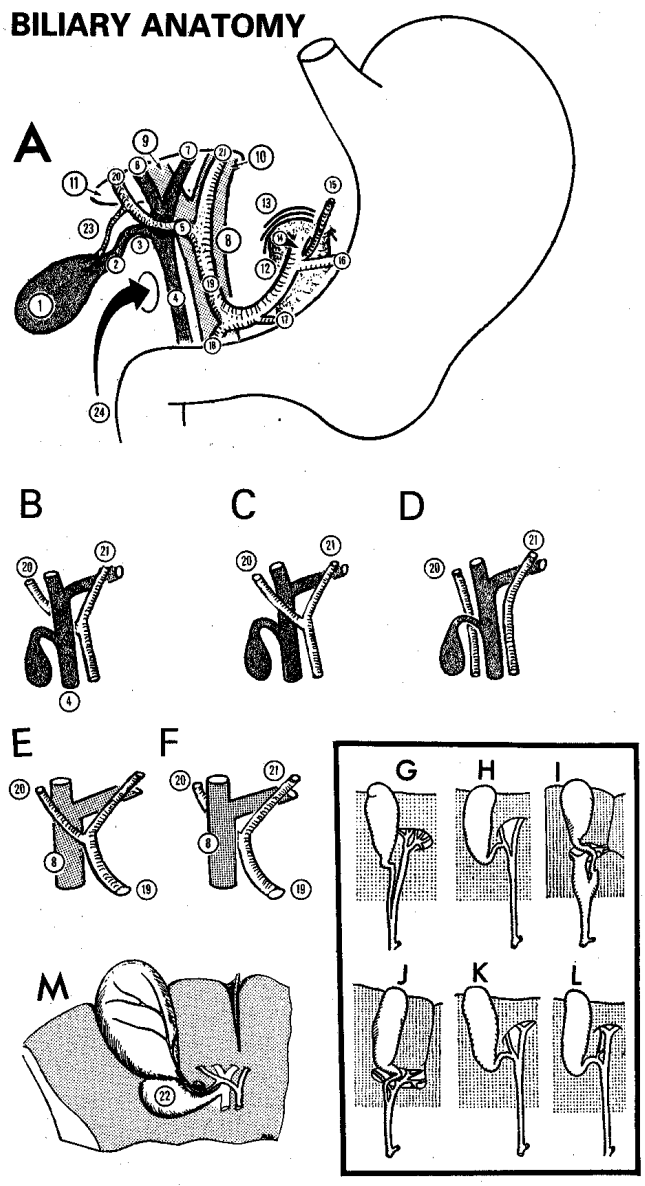


Fig. 15-3 ANATOMY OF THE BILIARY SYSTEM.
A, normal relationships of structures in this region. B-F, relations of the right hepatic artery. In B (and A), it runs posterior to the common hepatic duct (64%). In C, it runs anterior (24%), and in D, it arises from the superior mesenteric artery (9%). In E, it runs anterior to the portal vein (91%) and in F, posterior (9%).
G-L, variations in the bile passages (>50%). Note the accessory hepatic ducts in positions of surgical danger. M, a small pouch (Hartmann's pouch) may project from the right wall of the neck of a diseased gallbladder downwards and backwards towards the duodenum. When it is well marked, the cystic duct arises from its upper left wall and not from what appears to be the apex of the gallbladder.
(1) fundus of the gallbladder. (2) neck of the gallbladder. (3) cystic duct. (4) common bile duct. (5) common hepatic duct. (6) right hepatic duct. (7) left hepatic duct. (8) portal vein. (9) right branch of the portal vein. (10) left branch of the portal vein. (11) porta hepatis. (12) aorta. (13) some fibres of the diaphragm. (14) coeliac artery. (15) left gastric artery. (16) splenic artery. (17) right gastric artery. (18) gastroduodenal artery. (19) hepatic artery. (20) right hepatic artery. (21) left hepatic artery. (22) Hartmann's pouch. (23) cystic artery. (24) epiploic foramen (entrance to the lesser sac).
After Basmajian JV, Grant's Method of Anatomy, Williams & Wilkins 9th ed 1975 with kind permission.

Feel for the gallbladder. Feel for stones in the gallbladder and in the bile ducts. Feel both lobes of the liver to be sure they are smooth and normal. Examine the stomach and duodenum. Feel the pancreas.

If the gallbladder seems far up under the rib cage, run your hand over the right lobe of the liver, divide the falciform ligament across the dome of the liver, and draw it down. Put some large packs between the diaphragm and the liver – *don't forget to remove them afterwards!*

Insert a self-retaining retractor, and try to see the gallbladder. Get the anaesthetist to empty the stomach with a nasogastric tube. Use long tissue forceps to place large moist abdominal packs over the hepatic flexure of the colon, the duodenum, and the stomach. Ask your first assistant to draw these downwards and medially. You should now be able to see under the liver clearly.

Protect the liver with a pack, and ask your second assistant to retract it upwards and laterally with a large Deaver's retractor (15-4B). Look at the gallbladder. Divide any omental adhesions to the gallbladder, if present.

If the gallbladder is acutely inflamed, perform a cholecystostomy (15-1).

If it is very small, shrunken, thick-walled, contains stones, and is firmly stuck to nearby structures, leave it alone, or take out the stones and perform a cholecystostomy. Removing such a gallbladder will be very difficult.

If it looks and feels reasonably normal, apart from a few stones, and is attached by fine adhesions only, it should be safe to proceed.

A. THE RETROGRADE ('DUCT FIRST') APPROACH
Use this if you can readily find the cystic duct, the common bile duct, and the hepatic artery, in the free edge of the lesser omentum. The epiploic foramen (of Winslow) lies behind it; you should be able to pass one or two fingers through it into the lesser sac.
Place a gallbladder clamp or sponge-holding forceps on Hartmann's pouch (15-4C). This is a widened area in the lower part of the patient's gallbladder, just before it tapers off into the cystic duct. Pull gently upwards on these forceps, so as to stretch the tissues and make dissection easier.
Incise Calot's triangle of peritoneum between Hartmann's pouch and the common hepatic duct. This will appear when you apply traction to the sponge-holding forceps on Hartmann's pouch. It is a most important step. Start by making a small nick in the peritoneum with a long pair of Metzenbaum scissors. Carefully insert the tips of the scissors, then, using the 'push and spread technique' (4-9) or a Lahey dissecting swab, open up enough of the patient's peritoneum to expose the deeper structures.

CAUTION! Be careful not to cut any small blood vessels. Bleeding will make the operation difficult. By spreading the blades of the scissors (*but not too far!*) before you cut, or using a Lahey dissecting swab, you should be able to separate peritoneum only.

Take a Lahey swab (15-4C, E), and gently push apart the peritoneum, so that you see the junction of the common bile duct, hepatic and cystic arteries. This is most important. *Don't proceed unless you are certain of the anatomy!*

CAUTION! There are some important anatomical variations:
(1) The common bile duct and the cystic duct may join high or low (15-3G-L). The cystic duct may be very short and the common bile duct is then dangerously tented by traction on the gallbladder.
(2) The right hepatic artery may pass behind the common hepatic duct (15-3A, B, more common) or in front of it (15-3C, less common).
(3) The cystic artery may be closely bound to the common hepatic duct.
(4) The cystic artery usually (64%) arises from the right hepatic artery. It may cross behind (usually) or in front of (unusually) the common hepatic and cystic ducts to reach the gallbladder. Sometimes, it arises from the common hepatic (27%) or the left hepatic artery (5%), or from other arteries in the region (rare).

Be sure of your landmarks before you start to divide anything. Use a Lahey swab and dissect by the 'push and spread' method and thereby find the junction of the patient's cystic and common bile ducts, as described above. Be sure to identify 2cm of the common duct, both proximal and distal to the junction. This will give you an idea of its course and direction. The common bile duct lies to the right of the structures going to the porta hepatis, and is a greenish colour: identifying it is one of the keys to safe gallbladder surgery.

If the cystic artery runs posterior to the common hepatic and cystic ducts (usual), take extra care. Using traction with your left hand on the gallbladder, follow the cystic artery onto the gallbladder. *Don't expect to feel any pulsation in such a small vessel*. If a strand of tissue runs to the gallbladder, assume it is the cystic artery, pass 2 mounted ties around it, and divide between them. Expect to find other branches and deal with them in the same way.
If the cystic artery runs anterior to the common hepatic and cystic ducts (unusual), define it by blunt dissection, and make sure that it is indeed going to the gallbladder.

CAUTION! Don't tie the right hepatic artery by mistake.
If you are sure you have found the cystic artery, tie it doubly proximally and then close to the gallbladder with 2/0 silk (15-4D), and divide it leaving a short cuff of tissue, distal to the tie.
If you have found the junction of the cystic and common bile ducts, and you are sure that what you presume is the cystic duct is going to the gallbladder, and nowhere else, define it further, using blunt dissection (15-4E). This is the time to perform an operative cholangiogram if you can and you are still not sure. Using a long pair of Lahey forceps, gently open up the cleft between the cystic and common hepatic ducts. Pass a mounted tie of '0' absorbable suture through this cleft, and around the cystic duct, but not too close to the junction to cause a kink there. Tie it and for safety's sake, tie a 2nd suture around the cystic duct (15-4F).

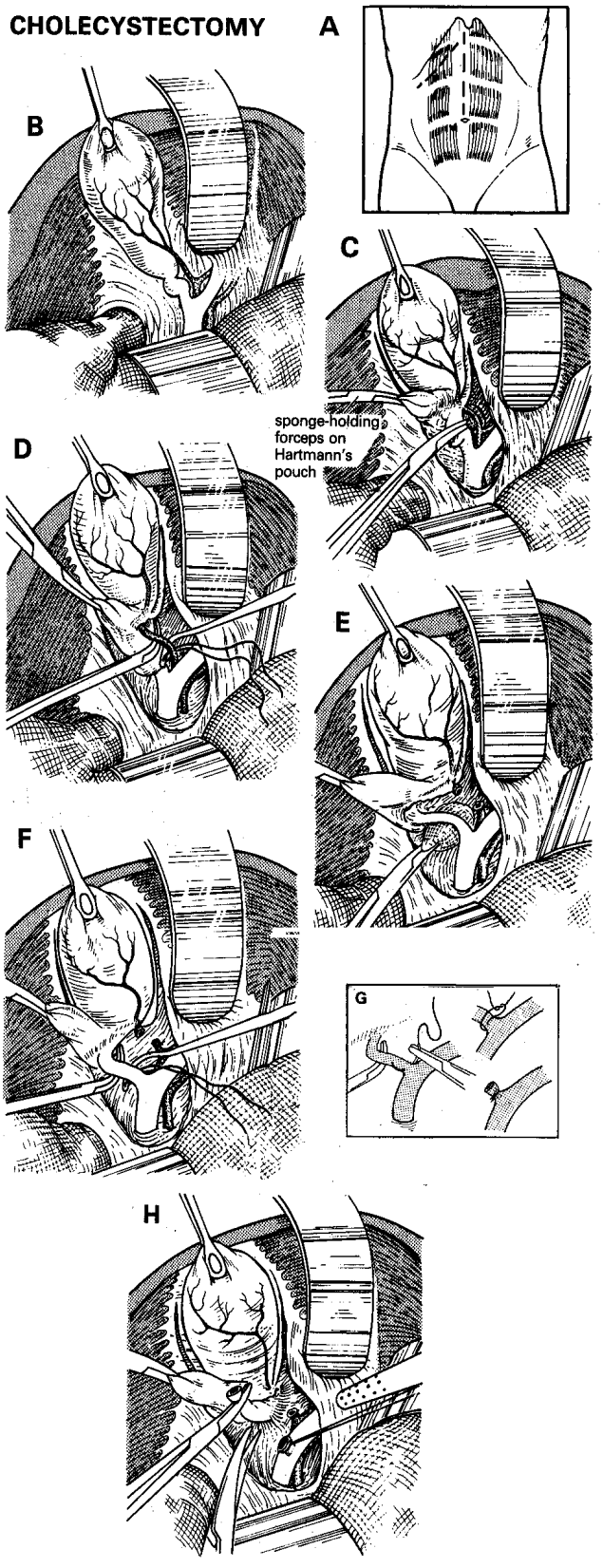


Fig. 15-4 REMOVING THE GALLBLADDER.
A, incisions. B, expose the gallbladder. C, expose the cystic duct. Note that the second forceps holds a Lahey swab. D, tie the cystic artery. E, free the cystic duct. F, tie the cystic duct. G, if the cystic duct is very large and thickened, transfix and tie it like this. H, separate the gallbladder from the liver. *N.B. if the liver bed bleeds, pack it.*
After Rob C, Smith R. Operative Surgery, Butterworth 2nded 1969, Vol4, p.404, with kind permission.

Place another Lahey clamp on the cystic duct, close to the gallbladder, above the ties. Cut the cystic duct just flush with the Lahey clamp, leaving enough tied cystic duct behind, so your ligature will not slip off. If the cystic duct is very large, transfix it (15-4G).

CAUTION! Only divide and tie structures that are passing to the gallbladder. *Don't leave too long a stump of cystic duct behind* as it may form a stone.

You should now be able to strip the gallbladder from its bed by pulling it gently upwards on the clamps. Cut any peritoneal bands that join it to the liver (15-4H), but tie off anything else: there may be a vessel or an anomalous bile duct inside a strand.

If the bed of the gallbladder oozes, press a warm pack into it. If small veins continue to bleed, cauterize them. It is unnecessary and dangerous to close the peritoneum over the bed of the gallbladder.

CAUTION! Check to make sure that the stump of the cystic duct is secure and that no bile is leaking.

B. THE ANTEGRADE ('FUNDUS FIRST') APPROACH
Use this if you cannot readily find the cystic duct, common bile duct, and hepatic artery.

Place a gallbladder clamp or sponge-holding forceps on the fundus of the gallbladder, and divide the peritoneum between the fundus and the liver. You may need to tie any larger vessels that bleed. Mobilize the gallbladder in this way anteriorly and posteriorly, continuing till you reach the neck of the gallbladder. Locate the cystic duct and trace it to the junction with the common bile duct. Then continue as above.

OPERATIVE CHOLANGIOGRAPHY (38.1) is important if there has been a history of jaundice, if ultrasound shows dilated extra-hepatic bile ducts with or without showing a stone, and if a stone is palpable in the common bile duct.

CLOSING THE WOUND. Place a soft rubber drain through a stab wound down to the porta hepatis if tissues are inflamed or you are not certain that the cystic duct ligature will hold: *a controlled bile leak is better than an uncontrolled one!* Close the abdominal wound (11.8); do this in layers if you have used a Kocher incision.

DIFFICULTIES REMOVING THE GALLBLADDER
If you find an adherent inflammatory mass around the gallbladder, withdraw and close the wound. Consider operating later, when the inflammation has subsided. Rarely is this a malignant mass, which is forever inoperable.

If you palpate stones in the common bile duct, or find them on an operative cholangiogram, perform a choledochostomy (15-2) and try to extract them with Desjardin's forceps, or perform a transduodenal Oddi sphincterotomy (15.5). Perform a post-extraction operative cholangiogram, if you can.

If the cystic artery bleeds from the depths of the wound, this can be alarming. *Don't clamp blindly.*

(1) Insert warm moist packs, apply pressure and wait 5mins by the clock. The spurting vessel will then be easier to find and control. Or,
(2) Put your index finger into the epiploic foramen and squeeze the structures (portal vein, bile ducts, and hepatic artery) in the free edge of the lesser omentum between your index finger and your thumb (the Pringle manoeuvre). This will control bleeding from the stump of the cystic artery. When you have suction and instruments ready, remove the packs and try to visualize the vessel, and clamp it. Transfix it carefully with 3/0 silk. *Don't use diathermy in the depths of the wound*, especially if you can't see properly! If exposure is poor, *enlarge your incision.*

If you suspect you have injured the cystic duct early on, make sure your suction is working properly and aspirate bile so you have a good view. If necessary, *enlarge your incision.* Get a cholangiogram if you cannot interpret the anatomy. If the cystic duct is incompletely divided, hold the part near the gallbladder in a Lahey clamp, and pass a mounted tie around it at the common bile duct end and complete the division of the cystic duct. If you injure it very near its union with the common bile duct, transfix it and carefully tie it, dividing it distally. *Make sure you haven't kinked or narrowed the common bile duct.*

If you find that you have damaged the common bile duct, you will have done so in one of three ways:
(1) By a ligature or by a clamp; undo the ligature or take off the clamp and inspect the damage. Perform a choledochostomy (15-2) higher up, and pass a T-tube limb inside through the damaged area.
(2) By partly dividing it; leave a T-tube threaded up and down the duct and proceed as for choledochostomy. Try to repair the hole in the bile duct using interrupted 4/0 absorbable sutures, taking care not to narrow it. Keep the T-tube in for 6wks, and then get a T-tube cholangiogram and remove it if there is free flow into the duodenum.
(3) By completely dividing it; try to drain the bile by passing a tube into the severed proximal end of the bile duct and secure it. You can leave this to drain externally into a bag. Or you can try to insert a T-tube into both ends of the severed duct, tie it so it doesn't slip out and manage the T-tube as before.

If you cannot find the distal end of the bile duct, however, insert the distal limb of the T-tube directly into the duodenum and secure it with a purse-string absorbable suture. (To make a permanent bypass, the definitive operation of choledochojejunostomy-en-Y using a Roux loop is a very demanding procedure.)

N.B. Learn from your mistakes, seek to be able to forgive yourself, and carry on.

DIFFICULTIES AFTER CHOLECYSTECTOMY
If fresh blood discharges from the drain, the pulse rises, the blood pressure falls, and there are signs of a haemoperitoneum, the cystic artery is probably bleeding. Re-open the abdomen, extend the incision, suck out all the blood and insert packs to control the haemorrhage. Then try to visualize the bleeding vessel, clamp it and tie it off.

If bile comes from the drain, with fever, severe pain and a leucocytosis, suspect that infected bile and exudate are pooling under the liver. Treat with gentamicin or a cefalosporin. Perform an ultrasound if you can to locate and quantify the amount of liquid. If there is no improvement, re-open the abdomen, extend the incision, suck out all the bile and inspect the cystic duct stump. If this is obviously leaking and you can hold it in a clamp, transfix it. Usually the whole area is grossly inflamed and you will not be able to identify structures easily; make sure the area is adequately drained.

If the patient becomes jaundiced, suspect the bile duct has been damaged or a stone has lodged in the distal bile duct. Try to confirm this by finding a dilated proximal biliary tree on ultrasound (38.2c). Unless you have access to sophisticated endoscopy, arrange a re-exploration which is difficult, and may mean a choledochojejunostomy-en-Y. If you cannot manage this, you can try to drain the proximal biliary system percutaneously through the liver under ultrasound guidance, or insert a T-tube (15.5) in the dilated part of the duct. Distal stones may pass on their own; a specialized endoscopist may be able to remove them from below.

15.9 **Obstructive** (cholestatic) **jaundice**

When jaundice is due to an obstruction in the flow of bile:
(1) The stools are pale.
(2) The urine is dark, and contains little or no urobilinogen.
(3) The skin itches because of deposition of bile salts.
These features are most marked in complete obstruction, as when carcinoma blocks the common duct. Pain and fever are usually absent. Stones typically cause an intermittent obstruction, and a less characteristic picture. If a stone impacts in Hartmann's pouch (15.4, 15-3M) or in the cystic duct, it causes pain but does not impede the flow of bile down the common duct, so jaundice is absent.

If an older patient has a steadily deepening and usually painless obstructive jaundice, and the gallbladder is palpably enlarged, some tumour is probably obstructing the common bile duct. It is unlikely that there are gallstones (Courvoisier's rule). This is probably incurable, but a cholecystojejunostomy to decompress the gallbladder, by diverting the bile into the jejunum, may make the patient's last days more bearable.

There are several causes, however, of obstructive jaundice:
(1) A secondary tumour in the porta hepatis, usually from a primary in the stomach or gallbladder itself.
(2) Carcinoma of the head of the pancreas.
(3) Cholangiocarcinoma or other cholangiopathies (15.7).
(4) Metastatic liver carcinoma.
(5) Hepatoma (although this is a common disease, presentation as obstructive jaundice is unusual).
(6) Drugs, *e.g.* antibiotics, contraceptives, chlorpromazine, cimetidine, oestradiol, imipramine and many others.
In endemic areas, other causes are liver flukes (15.7) and hydatid disease (15.12).
In neonates, look for a congenital abnormality (33.9).

DIFFERENTIAL DIAGNOSIS. First try to decide what kind of jaundice the patient has.

Haemolytic jaundice. The stools are dark. There is no bilirubin in the urine, but the urinary urobilinogen is increased. The blood shows increased levels of unconjugated prehepatic bilirubin (leading to high readings on the indirect van den Bergh test). The serum transaminases (GPT and GOT) are normal, and so is the alkaline phosphatase. There is a reticulocytosis. Look for evidence of a haemoglobinopathy, especially sickle cell disease, and malaria. Check for consumption of medicines, especially dapsone or sulphonamides. Check for a splenomegaly and insect or snake bites.

N.B. Haemolysis may result in pigmented gallstones!

Obstructive jaundice. The stools are pale (clay-coloured if obstruction is complete), and *show no improvement in colour in 10 days.* There is bilirubin in the urine, but little or no urobilinogen. There is a high conjugated (posthepatic) bilirubin level (giving high readings on the direct van den Bergh test). The alkaline phosphatase is very high. The transaminases are usually normal.

Hepatocellular jaundice. This is commonly viral hepatitis with an obstructive phase lasting 7-10 days, but sometimes much longer. At this stage the stools are pale. The urine contains bilirubin but little urobilinogen. The serum bilirubin is moderately increased (mostly conjugated). The alkaline phosphatase is usually only moderately increased, but if cholestasis is a prominent feature it can rise to levels seen in obstructive jaundice. The transaminases are increased. As the oedema of the cells settles, the stools become normal or even dark, the serum bilirubin falls, the urinary urobilinogen rises or reappears, and the transaminases fall gradually. The return of stool colour is the most important sign. This form of jaundice is not common >35yrs.

CAUTION! You may have difficulty distinguishing the obstructive phase of hepatocellular jaundice from surgical obstructive jaundice. Do all you can to make the distinction. A laparotomy for a stone may be life-saving, but GA (especially with halothane) and the trauma of surgery may cause hepatocellular jaundice to deteriorate, perhaps fatally.

ULTRASOUND (38.2c) is very useful. You need to show extra-hepatic bile duct dilation >7mm to make an operable diagnosis.

Suggesting malignancy:
(1) Relentlessly progressive, steadily deepening obstructive jaundice, weight loss.
(2) A palpable gallbladder which you can feel as an elongated, smooth, non-tender mass, normal in contour, and slightly mobile, which may extend to the umbilicus or even below it. If you can feel a distended gallbladder, it strongly suggests a malignant obstruction at the lower end of the common bile duct, but *its absence does not exclude this*. Aspiration of green bile implies free drainage from the common hepatic duct, but aspiration of 'white bile' (*i.e.* mucus) suggests occlusion of the cystic duct.

Suggesting metastases in the liver or a hepatoma: a large, hard, knobbly liver. A bruit is often present with a hepatoma, ascites is common, and is often blood-stained.

Suggesting a carcinoma of the stomach with metastases in the porta hepatis: pain, anorexia, vomiting, an upper abdominal mass, and the visible peristalsis of pyloric stenosis. Anaemia is common.

Suggesting carcinoma of the head of the pancreas: vague epigastric pain, a palpable gallbladder and weight loss.

Suggesting gallstones: a long history of intermittent varying jaundice, severe intermittent colicky pain, fever, chills and rigors (suggesting cholangitis), little or no weight loss, flatulent dyspepsia. A leucocytosis suggests cholecystitis.

Suggesting tuberculosis: caseating nodes in the porta hepatis, with signs of glandular tuberculosis elsewhere.

Suggesting stenosis of the bile ducts, either malignant or benign: a tender, enlarged liver. A chronic history of repeated attacks of cholangitis. The gallbladder may or may not be palpable. Common where liver flukes are endemic.

Suggesting carcinoma of the gallbladder: an enlarged liver and a hard, irregular mass in the right hypochondrium, usually in a female.

MANAGEMENT. If there are gallstones, the patient needs a choledochostomy (15-2) unless you can remove the gallstones endoscopically. If there is malignant disease with obstruction at the lower end of the common bile duct, a cholecystojejunostomy may help unless you can refer the patient for endoscopic stenting.

CHOLECYSTOJEJUNOSTOMY FOR OBSTRUCTIVE JAUNDICE (GRADE 3.3)

INDICATIONS. In practice the presence of a smooth enlarged gallbladder is the only clear indication to operate. However, you may still achieve a good result in some cases where the gallbladder is not distended.

CONTRAINDICATIONS. Cachexia, debility, a hard irregular gallbladder mass, a hard craggy liver due to metastatic deposits, hepatoma, a large gastric tumour, ascites.

PREPARATION. Confirm suitability for operation by aspirating green bile from the gallbladder (if necessary under ultrasound control). Treat with vitamin K 10mg IM od at least 48h pre-operatively. This will reduce a tendency to bleed. Patients with jaundice are prone to acute renal failure if their glomerular filtration rate falls. So make sure hydration is adequate pre-operatively. Treat with dextrose IV to combat hypoglycaemia when starved pre-operatively.

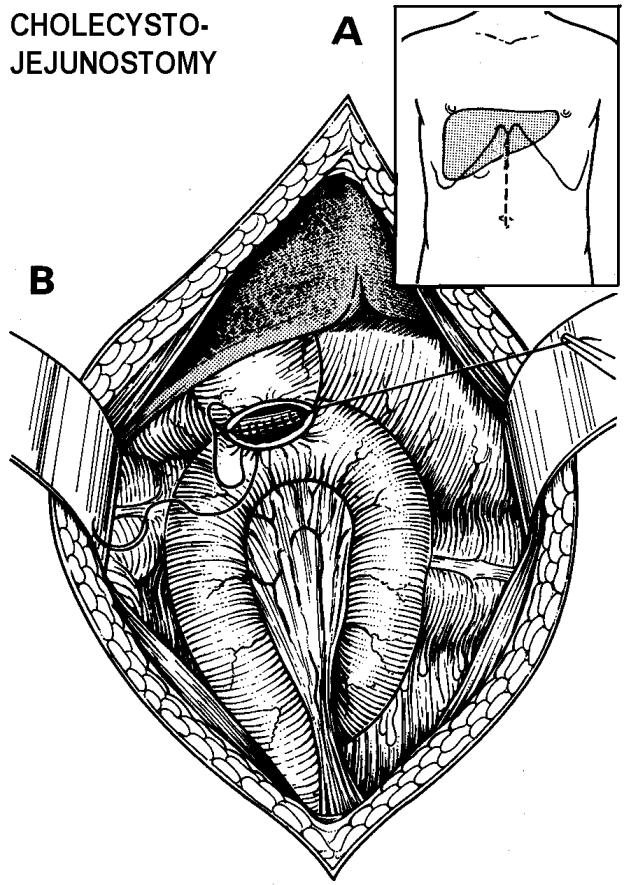


Fig. 15-5 CHOLECYSTOJEJUNOSTOMY.
A, the incision. B, the first layer of the anastomosis.

INCISION. Open the abdomen through an upper midline incision. Expose the liver and subhepatic area (15.8). Good exposure is essential. Inspect and feel the upper abdominal viscera carefully. Is the gallbladder normal in size and appearance? If it is a hard, irregular mass which is fixed to the surrounding organs, it is probably malignant.

If the gallbladder is inflamed, or contains many stones, perform a cholecystostomy (15.3), unless the obstruction in the bile duct is proximal to the cystic duct junction. *Don't try to anastomose bowel to a thick-walled, inflamed, oedematous gallbladder*.

Feel the pancreas, especially its head. Lift the transverse colon upwards and forwards out of the wound with your left hand, while you feel the pancreas at the base of the transverse mesocolon. Its head lies to the right of the vertebral column at this level. A hard, knobbly, craggy mass suggests a tumour. Also feel it from above. Stand on the left side of the table and feel with your right hand while you pull the hepatic flexure of the colon medially. Place your thumb anteriorly and your fingers posteriorly. Feel the head of the pancreas lying in the concavity of the duodenum. If necessary, mobilize the duodenum (15.5), so that you can feel the pancreas properly.

CAUTION! Don't biopsy the pancreas. Unless you use special methods like fine needle aspiration, you may cause pancreatitis and a fistula. However, if there are distant nodules, try to get tissue for histopathology.

Feel the porta hepatis and the structures lying in the free edge of the lesser omentum. Can you feel any craggy, fixed, indurated masses, suggesting primary carcinomas of the bile ducts or secondary deposits? Feel the stomach.

If you find deposits suggestive of tuberculosis (16.5), take a small biopsy of surrounding tissue.

INDICATIONS FOR PROCEEDING FURTHER
Decide what to do next only at this stage.

If there is an enlarged and distended but otherwise normal gallbladder, showing that there is an obstruction in the common bile duct, proximal to or within the head of the pancreas, with no obstruction to the cystic duct, perform a bypass.

If there is 'white bile' (mucus) on aspiration from the gallbladder, the cystic duct is obstructed and a cholecystojejunostomy will be futile.

METHOD
Pack off the distended gallbladder. Decompress it as at cholecystostomy (15-1). Remove the purse-string suture, and extend the opening with scissors to a length of 1·5cm. Apply Babcock clamps to the fundus of the gallbladder about 1cm from each end of the incision. Lift the transverse colon upwards and look for the ligament of Treitz. This is the point where the retroperitoneal 4th part of the duodenum emerges to become the jejunum, slightly to the left of the vertebral column, and distal to the attachment of the mesentery of the transverse colon.

Choose a loop of jejunum 30cm distal to the ligament of Treitz, and draw it up towards the open gallbladder. Apply one or two non-crushing bowel clamps across the jejunal loop. Apply two Babcock clamps 3cm apart on the antimesenteric border of the jejunum, to match those on the fundus of the gallbladder. Bring these clamps alongside one another, making sure that there is no tension on the jejunal loop. Aim to make a 1·5cm diameter stoma.

CAUTION! Make the anastomosis neatly and carefully: biliary peritonitis is a serious complication of a leak.

The end-to-side anastomosis is similar to that for a gastroenterostomy (13.7). Make the seromuscular outer layer of sutures of 3/0 on an atraumatic needle. Insert 5 sutures, which should ideally pick up only the seromuscular layer of the jejunum, but which will probably be of full thickness, in the wall of the gallbladder.

Place them about 2mm away from the cut edge of the incision, and on the bowel side about 2mm back from the antimesenteric border of the jejunum. Incise the jejunum 3mm back from the suture line. Trim away redundant mucosa with fine scissors. Apply Babcock forceps temporarily over any bleeding points. Insert a continuous 'all coats' posterior layer of 3/0 atraumatic absorbable sutures, starting at one end; then continue to close the anterior layer with the same suture. Finally, continue with the previous 3/0 to insert an anterior layer of seromuscular Lembert sutures. Cover the anastomosis with a layer of omentum, and suture this in place.

Be careful to close the abdominal wall soundly (11.8).

DIFFICULTIES WITH CHOLECYSTOJEJUNOSTOMY

If you do find gallstones, make sure that the jaundice is not caused by stones distally in the bile duct. If it is, perform a choledochostomy (15-2), extract the stones, and if you are experienced, remove the gallbladder also. If the gallstones seem an incidental finding, the gallbladder is not inflamed and there is tumour distally, proceed with cholecystojejunostomy as above, but try to remove the gallstones from the gallbladder itself.

If there is gastric outlet obstruction (<10% of patients), shown by an enlarged stomach, and tumour obstructing the duodenum, perform a gastrojejunostomy (13.7) as well. To avoid bile refluxing into the stomach, you can make a third anastomosis, a jejunojejunostomy (part of the traditional 'triple bypass'), but this adds to the morbidity. Remember you are doing palliative surgery only. A Roux-en-Y (15.14) cholecystojejunostomy is probably a better combination with a gastrojejunostomy.

15.10 Liver abscess

A. Amoebic liver abscess: extra-intestinal amoebiasis

When *Entamoeba histolytica* spreads outside the bowel, it usually involves the liver. Here it can cause an 'abscess' filled with liquid necrotic liver. To start with, this is yellow or yellow-green, later it becomes a syrupy dark reddish-yellow. The central area of necrosis is surrounded by zones of progressively less damaged tissue and amoebae. The term amoebic 'abscess' is a bad one, because there is no pus.

There is an 80% chance that the abscess is in the right lobe of the liver, where you will be able to detect it clinically, unless it is very deep. Collections in the liver are sometimes multiple.

Metronidazole usually treats an uncomplicated liver 'abscess' very effectively, but it occasionally (if >5cm diameter) needs aspiration, and rarely (if >10cm) drainage. The major risk is that it will suddenly rupture into the abdominal cavity, or through the diaphragm into the pleural or pericardial space, or even into the lung. Rupture into the abdominal cavity is a dramatic catastrophe, with collapse and peritonitis, like the perforation of a peptic ulcer. Although the contents of an abscess are sterile, they cause an acute inflammatory reaction in the peritoneum, whether this is acute or chronic.

N.B. Common mistakes are:
(1) *Not to perform a sigmoidoscopy.*
(2) *Not to recognize amoebic ulcers when you do see them.*
(3) *Not to remember the existence of acute necrotizing amoebic colitis with perforation.*
(4) *Not to use all the evidence you can to diagnose a liver abscess.*

CLINICAL FEATURES

The patient is usually male (8:1 chance), <30yrs, and may be a child. In endemic areas (*e.g.* Kwazulu in South Africa; Pakistan; Mexico), amoebic 'abscesses' are not uncommon in babies, and also occur in the elderly.

The pain in the right upper quadrant, or the lower right chest, is constant or intermittent, not colicky, and does not radiate. It slowly gets worse, but is seldom severe.

Deep breathing and coughing often make the pain worse. Fever, anorexia, weakness, and loss of weight steadily progress. There is only a 30% chance of having had diarrhoea with blood and mucus during the previous year. Often, there is some associated disease, such as tuberculosis, HIV, malnutrition, or alcoholism.

The liver is tender, smooth, diffusely enlarged, and without an obvious lump. Palpation of the liver may cause much distress. Pressure over the lower 5 ribs in the right anterior axillary line is painful. Examine also for evidence of a pleural effusion.

SPECIAL TESTS. Leucocytosis and anaemia. Raised ESR. Check for hypoglycaemia and hyponatraemia. Look for amoebic ulcers with a sigmoidoscope. You are unlikely to find amoebae in the stools. Use new PCR tests which are very sensitive, if you can.

CHEST RADIOGRAPHY. Look for a pleural effusion, and elevation of the right dome of the diaphragm.

ULTRASOUND is by far the most reliable diagnostic tool. Look for a fluid-filled cavity in the liver (38.2b). Measure how near the abscess is to rupture: *<1cm liver tissue is dangerous!* You cannot differentiate an amoebic from a pyogenic liver abscess except by aspiration.

DIFFERENTIAL DIAGNOSIS includes:
(1) hepatoma,
(2) cholecystitis,
(3) hydatid cyst,
(4) pyogenic liver abscess,
(5) perinephric abscess.

Suggesting a hepatoma (15.11): a hard nodular mass, liver less painful and less tender, no fever or low fever (fever only occurs with very rapidly growing tumours), a bruit, bloody ascites.

Suggesting cholecystitis (15.3), perhaps with spreading suppuration: pain and tenderness is localized to the gallbladder region, there may be a history of intolerance to greasy foods, and presence of jaundice, more commonly in a female.

Suggesting a hydatid cyst (15.12): the mass arises from one or other lobe, rather than enlarging it diffusely; it grows slowly and is largely asymptomatic; it is smooth, tense, and cystic; tenderness is minimal, there is no fever, and the general condition is good. All this may change rapidly if the cyst becomes infected.

Suggesting a pyogenic liver abscess: a short history; severely ill with a spiking fever.

Suggesting a perinephric abscess (6.15): the swelling is low down over the liver on the right; the distinction may be very difficult. Aspiration may establish the site.

MEDICAL TREATMENT FOR HEPATIC AMOEBIASIS
Metronidazole 800mg tds for 5 days, and then
diloxanide furoate 500mg (children: 20mg/kg) tds for 10 days or chloroquine 600mg od for 5 days.

ASPIRATION FOR LIVER ABSCESS (GRADE 1.4)
INDICATIONS
(1) To confirm the diagnosis.
(2) As a method of treatment if the abscess is >5cm diameter, or if the patient fails to improve with medication in 48h, or if the abscess is in the left lobe of the liver, as this may perforate into the pericardium.
(3) If there is on ultrasound scan <1cm liver tissue between the abscess and the liver surface (38.2b).
(4) If there is jaundice, suggesting involvement of the biliary system.

METHOD. Treat with metronidazole for 48h before aspirating. Check the prothrombin index if there is jaundice; and correct a clotting defect with vitamin K 10mg IM. Using LA and full aseptic precautions, preferably under ultrasound guidance, pass a wide-bore (>1mm) long needle on a 50ml syringe, or better, a wide-bore pigtail catheter through healthy skin, over the site of greatest swelling, or maximum tenderness.
If you obtain >250ml, introduce a multi-holed plastic catheter on a trocar in the same direction, attach a 3-way tap, and aspirate until the cavity is apparently empty. Irrigate through the tube daily. Continue until the cavity is empty and then remove the tube.
You have only a c.10% chance of finding amoebae in the fluid. They are more often found in the wall of the cavity.

OPEN DRAINAGE FOR LIVER ABSCESS
(GRADE 2.4)
INDICATIONS
(This is rarely necessary if you have used metronidazole and aspirated the abscess adequately.)
(1) A deep-seated amoebic liver abscess which you have not been able to aspirate, and the patient is deteriorating on medical treatment.
(2) Fluid too thick to aspirate.
(3) Very large abscesses >10cm in diameter, which recur after repeated aspiration.
(4) A large abscess in the left lobe which may perforate into the pericardium.
(5) Abscesses causing marked elevation of the diaphragm.
(6) Secondary infection of the abscess.
(7) A suspicion of a pyogenic liver abscess or an infected hydatid cyst.

DRAINAGE. Make a subcostal incision, pack away the rest of the abdominal contents and insert an aspirating needle directly into the abscess cavity, to identify it. Push an artery forceps into it, open it and suck out the pus. Take some scrapings from the wall of the abscess, and examine a warm wet specimen for *trophozoites*.
If there is much discharge, insert a tube drain, and bring this out through a separate incision on the abdominal wall. The liver may recede from the abdominal wall as you drain it, and end up as a shrunken blob, in the right upper abdomen. Remove the drain in 6-7 days, to minimize the risk of secondary infection.

DIFFICULTIES WITH EXTRA-INTESTINAL AMOEBIASIS
If there is a sudden pain like with a perforated peptic ulcer, the abscess has probably ruptured into the abdominal cavity. Resuscitate with IV fluids and start IV metronidazole. As soon as the general state is satisfactory, perform a laparotomy.
Make a midline incision. Explore the liver as best you can, and look for the site of the rupture: a ragged area with chocolate-coloured fluid pouring from it. Suck out as much of this as you can. Mop up what you cannot aspirate. Irrigate all the crevices of the peritoneum with several litres of warm saline or water. Ensure a thorough lavage, and close the abdomen. Treat post-operatively as peritonitis (10.1).

If there are tender ill-defined masses in the abdomen, suspect that the abscess has leaked into the abdominal cavity. Start intensive medical treatment, and monitor the abdomen closely. Mark the outline of any mass, and check its size regularly. Perform a laparotomy if it gets bigger.

If an abscess presents on the chest wall, it will ultimately rupture through the skin, which may become infected with amoebae, and form a chronic ulcer (14-5A). Treat with metronidazole.

If a severe cough and dyspnoea develop, this may, or may not, mean that there is a pleural effusion. If the effusion is small, it will resolve as the liver abscess improves. If it is large, insert an underwater seal chest drain.

If there is an ulcerating skin lesion round a sinus from the liver, caecum, or anus, remember the possibility of CUTANEOUS AMOEBIASIS. Metronidazole cures cutaneous amoebiasis so fast that you can use it as a diagnostic test. In the perianal area, ulcerating skin lesions are usually non-specific. Untreated, the lesions spread fast; biopsies show up the *trophozoites*.
N.B. Tuberculosis, malignancy, and fungi are often commoner.

If the patient coughs up dark reddish-yellow fluid, the liver abscess has ruptured into a bronchus. Although this may drain the abscess, it may also fatally flood the bronchial tree. Get a chest radiograph and treat with IV metronidazole, aspirate the liver abscess, and use postural drainage for the lung (11-24).

If the liver abscess discharges into the pericardium, it usually does so from an abscess in the left lobe. Pericardial rupture is not uncommon in endemic areas, is often not recognized, and is usually fatal. Watch therefore for abscesses in the left liver lobe, and aspirate them. Try to aspirate the pericardium (9.2).

If there is no adequate response (<5%) to metronidazole alone, treat with chloroquine 600mg loading dose and 150mg bd for 21 days, and if necessary, dehydroemetine 65mg od IM for 10 days. This drug is cardiotoxic, so you cannot use it in heart failure.

If an epileptic fit ensues, there may be cerebral amoebiasis. If there are no focal neurological signs, treat with diuretics and steroids, as well as second-line therapy; if there are, suspect a brain abscess.

B. Pyogenic liver abscess

A large pyogenic liver abscess is usually much less common than an amoebic 'abscess'. Organisms spread mostly from the biliary tract, or *via* the portal vein from the bowel (*e.g.* appendix abscess or intestinal salmonella carried with chronic *Schistosoma mansoni* infestation or HIV disease) or *via* the hepatic artery from a remote focus (*e.g.* pyomyositis). Multiple small abscesses can result from *staphylococcal* septicaemia or ascending cholangitis. Occasionally the abscess may arise from penetrating trauma, or infection in a hydatid cyst (15.12).

The history is usually shorter than with amoebiasis. Jaundice suggests infection from an obstructed bile duct. Although signs of a large abscess are like those of the amoebic variety, much more commonly there is a fever of unknown origin. Ultrasound is the easiest way of making the diagnosis, and aids in liver aspiration.

Until cultures are available, use broad-spectrum antibiotics: metronidazole, gentamicin, cloxacillin and ampicillin.
Try to aspirate the abscess completely; if you fail, proceed to open drainage (as above). Open a large abscess to prevent rupture into the abdominal cavity!
Test for sickle cell disease, *schistosomiasis* or melioidoisis, if these are common in your area.

15.11 Hepatoma (Hepatocellular carcinoma)

Hepatoma is one of the world's common malignant tumours, mostly because it is so common in China, Southeast Asia, the Amazon region and Peru, and sub-Saharan Africa, where it is even more common than secondary tumours of the liver. In Africa, there is a 90% chance that a liver with a hepatoma is also cirrhotic, due to the strong association of hepatoma with hepatitis B, C and D infection, and with the *aflatoxin* produced by the *aspergillus* fungus growing in damp stored food. Prevalence of hepatitis B is also high in Greenland, Alaska and Northern Canada, and hepatitis C is a global problem.

A hepatoma often arises simultaneously as multiple nodules in many parts of the liver. Sometimes, a huge mass deforms one lobe. The other primary liver tumour, cholangiocarcinoma, is a problem in Southeast Asia.

The patient is usually male (8:1), and 30-50yrs. He complains of pain, anorexia, weight loss, or a mass; jaundice is a late sign. The pain is usually constant, and sharp or burning, and is in the upper abdomen, usually on the right. It is commonly made worse by food, which causes an inappropriate feeling of fullness after only a small meal. Typically, the pain is made much worse by alcohol, so much so that it may have caused abstinence.

Usually, presentation is late with a large, or even a huge, firm, irregular, tender liver. Quite a small tumour may cause prostration but normal activity may continue with a large tumour. There may be cachexia, but not always. Look for ascites and a large spleen. Look for collateral veins running vertically over the anterior costal margin, or parallel to the spinous processes. Listen over the tumour for a bruit or friction rub; this may be intermittent, so listen on several occasions.

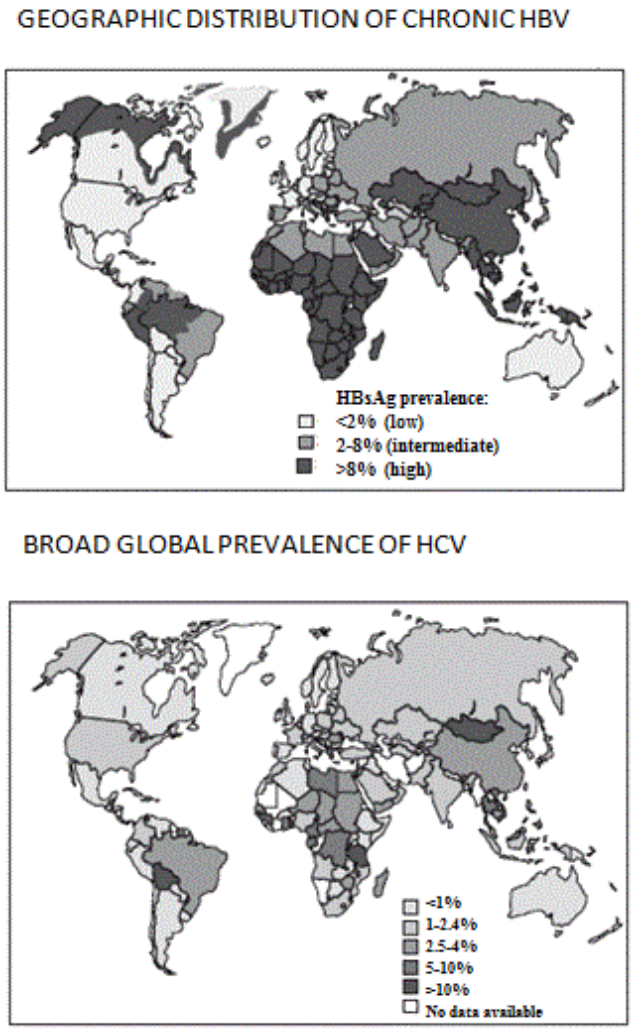


Fig. 15-6 HEPATITIS B, C AND HEPATOMA.
The geographical distribution of the hepatitis B virus, based on incidence of Hb_sAg in blood samples, correlates with hepatoma incidence. Hepatitis C has a much broader spread in the world.

If there is any room for doubt, do all you can to confirm the diagnosis. Several diseases present as swellings of the upper abdomen. Try to distinguish those you can treat, such as liver abscesses, hydatid cysts, and tuberculosis, from those you can only palliate.

The best and most non-invasive tool by far is an ultrasound: do all you can to get such a machine and get acquainted with it (38.2b). If you have any hope of sending specimens for cytology, use this method. Otherwise you need to biopsy the liver to get histology. This can be dangerous, so use ultrasound guidance if you can. If you cannot get histology, aspiration with a fine needle may still be useful to detect pus or tuberculosis.

In children the primary liver malignancy is hepatoblastoma. Usually the child is severely anaemic. The liver may rupture with a minor injury. Chemotherapy is the only option.

SPECIAL TESTS. Serum α-fetoprotein >500μg/L is present in 70-80% of cases. *Though this test is unlikely to be available locally, it may be easier to get a laboratory to do it rather than get histology on a potentially dangerous liver biopsy.* The bilirubin rises late, and is often not raised on presentation. If it is >40μmol/L, life expectancy is weeks only. The bilirubin and alkaline phosphatase levels rise parallel with one another. The transaminases are normal, unless there is also chronic active hepatitis. If there is ascites, aspirate a sample of the fluid and examine it. Blood-stained fluid supports the diagnosis of malignancy, not necessarily of the liver. Anaemia is seldom a marked feature.
Don't forget to test the bleeding and clotting times.

RADIOGRAPHS. Chest radiography usually shows that the right lobe of the diaphragm is raised. Metastases rarely show up but are present in 50% of patients postmortem.

ULTRASOUND is extremely useful and will show solitary or multiple lesions, or fluid cavities (38.2b). Use it to help guide a needle biopsy.

NEEDLE, liver biopsy, Menghini, 1·6mm, normal Menghini point, with adjustable stop.
NEEDLE, liver biopsy, Vim-Silverman, adult size, 23mm x 88.5mm, with Franklin modification.

NEEDLE BIOPSY OF THE LIVER (GRADE 1.4)
INDICATIONS
An enlarged liver when the diagnosis is unknown.

CONTRAINDICATIONS
(1) *Don't do this if your histology service is dubious in quality, or if the results are unlikely to arrive for weeks!*
(2) Deep jaundice, severe anaemia, or any bleeding tendency, as shown by petechiae, ecchymoses, or haemorrhages.
(3) Hydatid disease, where needle biopsy may lead to fatal anaphylaxis or dissemination.
(4) *Don't attempt this if you cannot transfuse blood in an emergency. Correct coagulation problems first!*

CAUTION! Measure the bleeding time (normal <3mins) and clotting time (normal <8mins) before taking a biopsy and treat with vitamin K 10mg IM till the bleeding and clotting times are normal.

ANAESTHESIA. Use LA in an adult; a child may need ketamine.

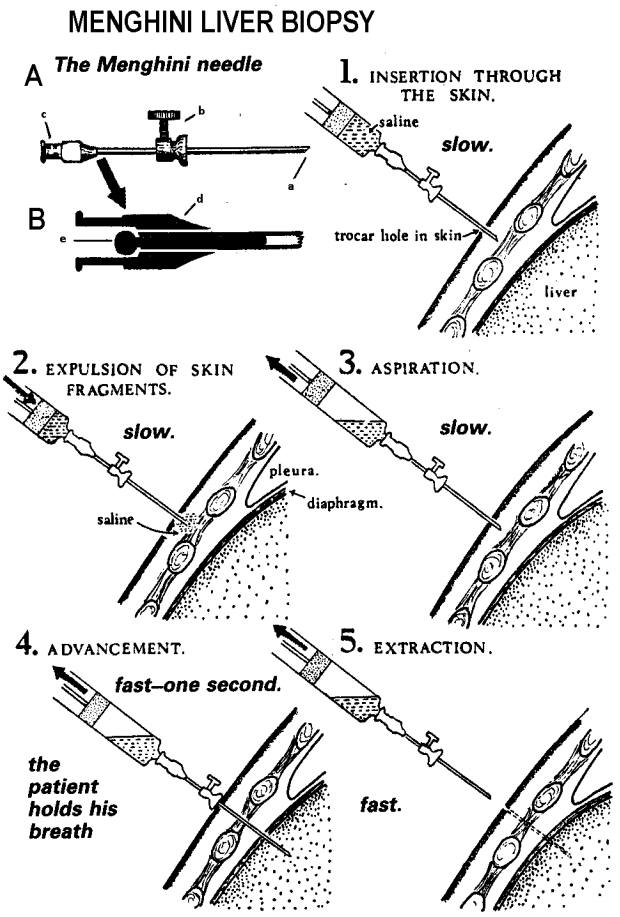


Fig. 15-7 MENGHINI LIVER BIOPSY.
A, Menghini needle. This has a sharp stout end (a), with an adjustable guard (b), and a Luer lock (c). B, viewed in cross section, the assembly has an obturator outside the needle, and a blocking pin, which slips loosely inside the needle and prevents the biopsy fragment from falling back and breaking up in the syringe. To take a biopsy using a Menghini needle: (1) insert it through the skin, (2) expel skin fragments, (3) aspirate, (4) advance the needle into the liver, with the patient holding his breath, and gently suck up tissue by holding the syringe steady, (5) extract the needle whilst gently maintaining suction.
After King M. Medical Care in Developing Countries, OUP 1967.

(A) MENGHINI NEEDLE
Fit the Menghini needle to a well-fitting 10ml syringe, set the guard at about 4cm, and draw up 3ml of sterile saline. Pass the needle point through the anaesthetized track through the intercostal space. Inject 2ml of saline to clear the needle point of any skin fragments.

CAUTION! Now, ask the patient to hold his breath in expiration.
Start to aspirate, and while continuing to aspirate, rapidly push the needle into the liver perpendicular to the skin, then immediately pull it out again. Apply pressure to the site of the biopsy. Continue aspirating until you have placed the needle point under some saline in a glass dish. Discharge the saline remaining in the syringe. The biopsy specimen will appear. Rescue it and transfer it to formol saline. Clear the needle with the obturator.

(B) VIM-SILVERMAN NEEDLE
Fit the inner obturator into the sheath. With firm but well-controlled pressure, push the needle through the abdominal wall while the patient holds his breath. You will feel the peritoneum 'give' as you go through it.
Don't push the needle in too far at this stage. Ask the patient to take a deep breath. If the needle moves with respiration, its tip is already in the liver; if not, ask him to hold his breath again, and gently push it 3cm further in, or until it moves.
The needle has hollowed-out biopsy jaws, a sleeve which fits over them, and an obturator to discharge the specimen. The inner jaws grasp a core of tissue, after which you slide the sleeve over them to trap it.
Surgeons vary in the needle they prefer. A Menghini needle is in a patient's liver for a shorter time, so it is safer. Its disadvantage is that it is less likely to withdraw a satisfactory specimen in a cirrhotic. This is important because cirrhosis is common in areas where hepatoma is common.

CAUTION! Don't manipulate the needle during regular breathing, or you may tear the liver.
Ask him to hold his breath, then remove the inner obturator and replace it with the biopsy jaws. Steady the needle with your left hand, and push the jaws with your right hand into the needle as far as they will go.
Ask him to hold his breath again. Hold the biopsy jaws firmly with your right hand, and slide the outer jaws 3cm further into the liver. This will wedge the jaws and the tissue firmly in the outer sheath. Rotate the needle once or twice, to break off any tissue which is attached at the tip, and then quickly withdraw it. Ask him to breathe again. You can do everything in a few seconds. *Long breath-holding is unnecessary*. Slide the sheath over the biopsy jaws and open them. Use a fine needle to remove the core of tissue from the jaws into formol saline.

METHOD. *Before you use either needle for the first time, try it out by doing the biopsy on a mango* (for example), *and if possible, practise on a cadaver.*

CAUTION! To avoid tearing the liver, tell the patient to hold his breath when you are pushing the needle in, pushing it in further, or pulling it out. There are also times when you will want him to breathe deeply to check the position of the needle. Before you start, make quite sure that he understands what is being done. If you are using LA, get him to practise holding his breath. This is important, because you must perform the puncture itself while he holds his breath at the end of expiration.

Lay the patient supine near to the right side of the bed, and place a firm pillow against the left side in the hollow of the bed. Place his right arm behind the head, and turn his face to the left.

Choose a point in the mid or anterior axillary line in the 8th, 9th or 10th intercostal spaces, or over the palpable mass that you want to biopsy. Clean the skin with iodine, and anaesthetize the chosen site with LA solution, down to the parietal peritoneum. Pierce the anaesthetized area with a scalpel (or with the special trocar).

If you have not succeeded, try again in a different place. If you fail after several attempts, there is probably no solid tissue that can be biopsied. If the needle goes in without any resistance, attach a 20ml syringe to it and aspirate: you may withdraw pus of a liver abscess (15.10), or blood from a haemangioma, or clear fluid of a hydatid (15.12).

POST-OPERATIVELY (both methods), keep the patient supine for 12h. Monitor the pulse and blood pressure during this time, just in case there is bleeding into the peritoneal cavity. A hepatoma is very vascular, and occasionally bleeds when you biopsy it.

DIFFERENTIAL DIAGNOSIS. Try to exclude treatable diseases. Some of these are inflammatory, and so produce fever, but this also occurs in primary hepatoma (8%), so it is not a reliable guide.

Suggesting secondary carcinoma of the liver: a hard nodular liver, evidence for a primary tumour. In carcinoma of the stomach there may be a separate mass, dyspeptic symptoms, or symptoms of pyloric obstruction.

Suggesting cholangiocarcinoma: deep jaundice, no bruit, and a liver which is less big and irregular than with hepatoma.

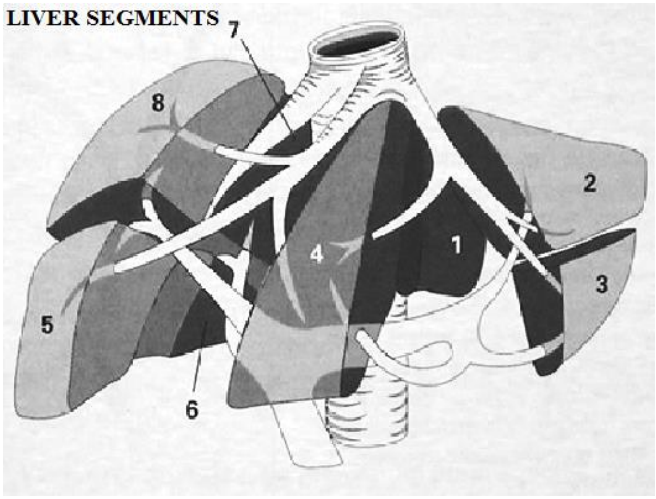


Fig. 15-8 LIVER SEGMENTS.
Each side of the liver is divided into 2 sectors: paramedian and lateral. *N.B. The falciform ligament dividing the liver into traditional right and left lobes does NOT equate with the right and left vascular divisions.* Each of the 4 sectors is further divided superiorly and inferiorly, hence forming 8 segments. However, the left paramedian sector is not formally divided, though known often as segments 4A and 4B (or 9). The caudal lobe is its own segment (1). Thus a right hepatectomy consists of removing segments 5,6,7,8 and a left hepatectomy segments 2,3,4 whilst a left hepatic lobectomy removes only segments 2+3.

Suggesting carcinoma of the head of the pancreas: deepening jaundice, little or no pain, the absence of bile pigment in the stools, a gallbladder which is usually palpable, no bruit.

Suggesting amoebic abscess: fever, a smooth, diffusely enlarged, tender liver with no obvious lumps, no jaundice; tenderness, especially intercostal tenderness.

Suggesting gallstones: severe colicky pain, biliary dyspepsia, little or no weight loss.

Suggesting hydatid disease, with cholangitis: contact with dogs, a tense, almost painless, longstanding (years), smooth, rubbery mass, commonly in the right upper quadrant; little weight loss, general condition good.

Suggesting a subphrenic abscess with downward displacement of the liver: fever and an acute or subacute illness, cough and chest signs on the right side, shoulder tip pain on the right side.

Suggesting tuberculosis of the liver: a hard irregular liver, often with no fever, no jaundice, pain is not marked, especially in an immunocompromised patient. When there is no jaundice, you cannot distinguish hepatoma and secondary carcinoma from tuberculosis, except by needle biopsy, which is one reason why it is so useful.

Suggesting intrahepatic stones: fever, right upper quadrant pain, rarely jaundice in a patient in East Asia.

PROGNOSIS. Few patients survive >6 months.

MANAGEMENT. Cytokine inhibitors may palliate usefully, but they are enormously expensive. There is little you or anyone else can do for the patient, with the rare exception of a single tumour confined to one lobe which can be resected in specialist centres.

Try to promote hepatitis B vaccination in your area if hepatoma is common.

15.12 Hydatid disease

The *Echinococcus granulosus* tapeworms in their larval stages cause uni- or multi-locular or multiple hydatid cysts; in their adult forms they inhabit the gut of dogs, wolves, coyotes and foxes. Man is infested in the same way as sheep, horses, oxen and cattle, by ingesting ova from canine faeces (either by hand-to-mouth transfer or from contaminated drinking water). Hydatid disease is widespread in Turkey, Rio Grande do Sul in Brazil, as well as Australia, North Africa and Southern Africa; but also in Saudi Arabia, Siberia, Northern China, Japan and the Philippines. Turkana in north-west Kenya had the highest prevalence, but now only <4% of the population are infected. Programmes to control hydatid infection have been among the most successful measures of their kind.

If hydatid disease is endemic in your area, you may find hydatid cysts in the liver (80%), spleen (7%) or the other parts of the abdomen (14%), or occasionally in the lungs, brain, bones, or indeed almost anywhere. If there is a peripheral cyst, expect that there is at least one in the liver too.

The presentation is typically of a tense, almost painless, longstanding, smooth, rubbery, mobile cyst in the upper right quadrant, often of several years' duration, with little fever or malaise: it may be enormous, and may be multiple. A hydatid cyst usually contains several litres of clear highly antigenic fluid under pressure. Around it there is a thin, tough, fibrous ectocyst. Inside this, and separated from it often by an easy plane of cleavage, is a thick, yellowish, slimy, gelatinous endocyst, which the parasite forms, and which tends to split and curl up on itself when you cut it. Scolices, which are tiny, barely visible, white granules, and daughter cysts, like grapes or soap bubbles, float free in the fluid of the cyst.

Echinococcus vogeli, a parasite found in Central and South America, produces multiple cysts in the liver. Likewise, *Echinococcus multilocularis*, found in Central Asia, produces multiple cysts, but also inflammatory infiltrations that mimic tumour, and can spread to the lungs in 30%.

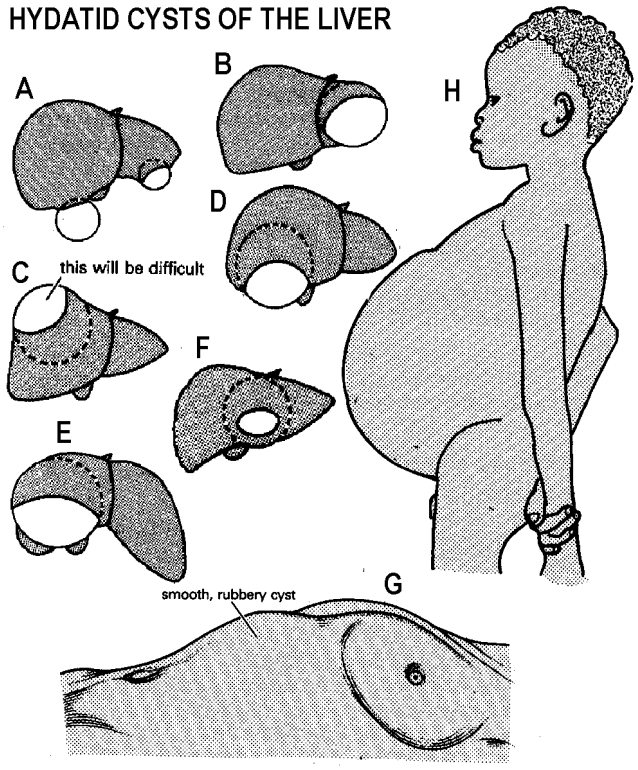


Fig. 15-9 HYDATID CYSTS OF THE LIVER.
A-F, some of the cysts you may find. C will need a thoraco-abdominal incision. G, a patient with a single cyst. H, abdominal distension due to multiple hydatid daughter cysts throughout the peritoneal cavity. *After Pelissier G, Goinard P, Peguilo J. Le kyste hydatique, Masson, Niort 1960.*

A cyst in the liver may rupture into:
(1) the bile ducts and cause cholangitis,
(2) a serous cavity, where it may cause a hypersensitivity reaction, varying from urticaria to anaphylactic shock.

The diagnosis is difficult without ultrasound, and the disease may be so rare that you forget it as a possibility. There is hardly ever any hurry to operate, so investigate it as fully as you can.

Surgery is likely to be difficult, so treat it medically before you operate or aspirate. The method of puncture, aspiration, injection (of scolicide) and re-aspiration (PAIR) is safer than surgery in most cases. You may be able to do this under ultrasound control if you have had some experience.

If you intend to remove a hydatid cyst surgically, do so in the plane between the tissues of host and parasite: leave the ectocyst, and don't try to remove the entire cyst intact, unless you can do this easily, as in the ovary or spleen.
Instead:
(1) Gain access to the cyst, and pack it off, so that if any infective hydatid fluid does escape, it will not contaminate the peritoneal cavity; 1ml can contain thousands of scolices.
(2) Aspirate some of the fluid, and inject a scolicide.

(3) After 10mins, aspirate as much remaining fluid as you can.
(4) Scoop out the endocyst and the daughter cysts.
(5) Close the cavity, so that it does not leave a sinus or a fistula.

In a child, the cyst will probably be monolocular, so that aspirating it will not be too difficult. But in an adult, it is likely to be multilocular, so that:
(1) the fluid will be difficult to aspirate, because the daughter cysts block the needle, and
(2) the scolices will be difficult to sterilize, because the scolicide cannot penetrate in to them.

The great danger is that you will spill the fluid, which may:
(1) allow daughter cysts to establish themselves in the peritoneal cavity, and
(2) cause anaphylactic shock. The recurrence rate is usually at least 25%.

PREVENTION. If yours is an endemic area, do all you can to educate the local population, especially about washing hands, eliminating stray dogs and regularly deworming their domestic dogs, not feeding them uncooked offal and burning refuse from slaughtered animals.

SPECIAL TESTS. An absolute eosinophilia is present in 30% of patients. *The Casoni test gives false -ve and +ve results in c.20% of cases, and is no longer used*. Other antibody tests, such as the ELISA test, may also give false negatives, particularly for bone and brain cysts.

RADIOGRAPHS. Take a chest film: there may also be cysts in the lungs. A cyst in the liver may raise the right lobe of the diaphragm. An old aborted cyst may leave a calcified shadow.

ULTRASOUND is the best way of detecting hydatid cysts (38.2b). A lamellated membrane, detachment of the membrane from the cyst wall, or the presence of daughter cysts confirms the diagnosis. You should be able to differentiate a hydatid from a simple liver cyst by its having a double wall.
DIAGNOSTIC ASPIRATION. *Don't try to do this*, because this may make the cyst leak, and may be fatal. Aspiration is part of treatment. *Don't try to tap a hydatid cyst merely to make it smaller.*
If you are aspirating what you think is an abscess, and you aspirate clear fluid, it is highly likely to be a hydatid cyst. Continue aspirating to dryness, before you remove the needle. Then inject a scolicide, wait, re-aspirate and then quickly withdraw the needle.

DIFFERENTIAL DIAGNOSIS
Suggesting simple liver cyst: smooth hepatomegaly, with single cyst wall on ultrasound.
Suggesting liver abscess: fever, tender smooth hepatomegaly, with short history of illness.
Suggesting hepatoma: irregular huge liver with ascites and weight loss; audible bruit.
Suggesting secondary carcinoma of the liver: hard knobbly enlarged liver with weight loss.

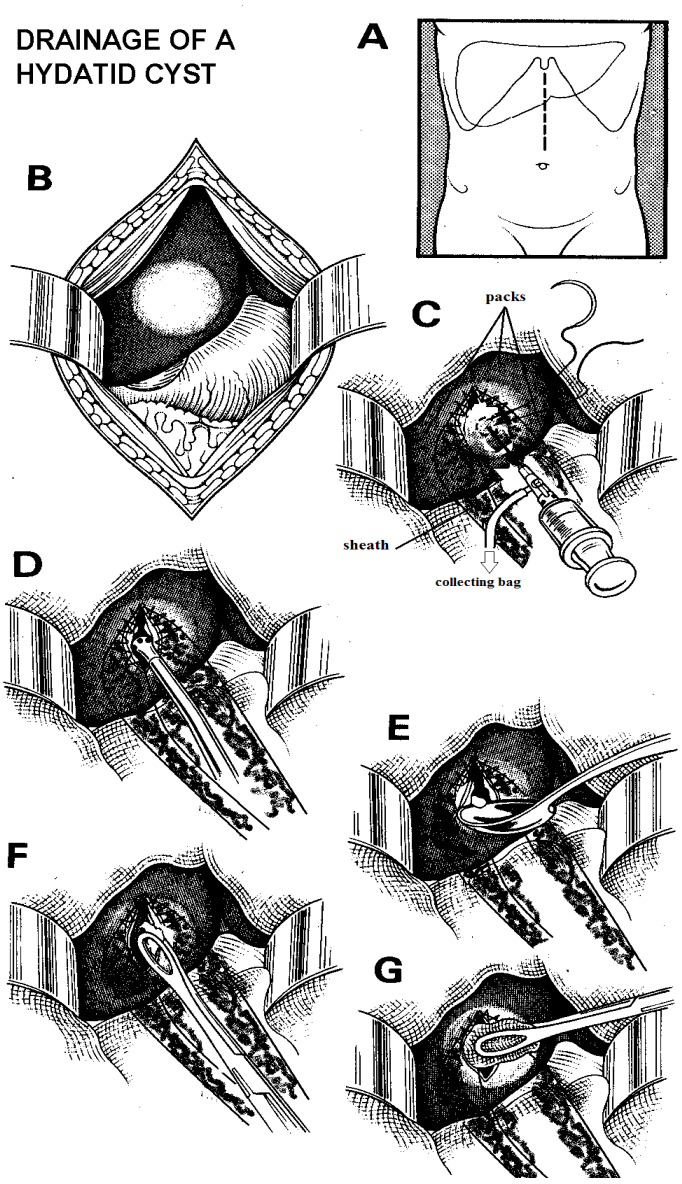


Fig. 15-10 OPERATION FOR HYDATID CYST OF THE LIVER.
A, high midline incision. B, expose the cyst. C, isolate the cyst with scolicide-soaked packs, and suture a tubular sheath to the pericyst. Work within this sheath to avoid spillage. Aspirate 50% of the fluid into a bag of scolicide. Leave the needle in place and inject 50-100ml scolicide till it becomes tense. Tie the purse string around the puncture site and wait for 10mins. Re-aspirate the cyst till it is flaccid. D, then incise the pericyst and suck out the rest of the fluid. E, remove the daughter cysts with a spoon. F, remove the endocyst completely with sponge forceps. G, swab out the cavity with scolicide.
Partly after Rob C, Smith R. Operative surgery: Part 1: Abdomen, Rectum and Anus, Butterworth 2nd ed 1969 p.322 with kind permission.

TREATMENT
Treat with praziquantel 15-25mg/kg bd for 7-14 days, pre-operatively. This kills the proto-scolices. Small cysts may disappear without surgical intervention. Albendazole in high doses (10mg/kg od for 28 days) damages the germinal membrane and so is complementary to praziquantel. You should therefore use both! Repeat the dose 4 times after a rest interval of 15 days. It is contra-indicated in pregnancy. Mebendazole 600mg tds for 3wks is an alternative, but only effective in c.40%. Use cimetidine 400mg bd in addition.

PERCUTANEOUS ASPIRATION, INJECTION AND RE-ASPIRATION (PAIR)
Treat medically first for a minimum of 3wks.

If there is a single cyst <5cm diameter, withdraw as much fluid as you can under ultrasound guidance through a wide-bore needle, and inject, preferably through a T-connection piece, an equivalent volume (or slightly more) of a scolicide (preferably chlorhexidine 0·04%, which is safer and more effective than other scolicides such as cetrimide, 70% alcohol, 1% povidone iodine, 0·5% silver nitrate, hydrogen peroxide or hypochlorite). Start slowly by injecting a small volume; if there is any pain, abandon the procedure. If not, continue, and re-aspirate after 1hr.

CAUTION! Don't use formalin or hypertonic saline as scolicides.
Don't use PAIR if the cyst communicates with the biliary tree. Remember that the liver moves with respiration and so even then a needle will not stay immobile.

LAPAROTOMY FOR HYDATID CYSTS OF THE LIVER (GRADE 3.4)

INDICATIONS
Large, symptomatic, subcapsular cysts in young patients; large especially peripheral viable cysts, prone to traumatic rupture.
Don't attempt operation if the cyst is high on the right, under the diaphragm, or there are multiple, deep-seated cysts, or if there is a communication with the bile ducts because extensive mobilization of the liver will be necessary.

PREPARATION. Kill the hydatids and make dissemination less likely by using praziquantel or albendazole.
Treat with hydrocortisone 100mg IM 12h before the operation, so as to minimize the danger of anaphylaxis when you open the cyst and reduce the risk of laryngeal spasm.
Have ready: An aspirator, or a large-bore syringe with a needle and 3-way tap; a long pair of sponge forceps, and a sterile kitchen spoon to scoop out the cyst; coloured packs which will let you see the brood capsules and scolices more easily (soak the packs in scolicide); 2 suckers, one for the trocar (if you use one), and another ready for any spills; a sterilized 6x15cm tubular polythene sheath; and a scolicide.

INCISION
Make a midline incision unless the cyst is very peripheral. If necessary, extend this laterally in a J-shape, so that you have adequate exposure. Another way of getting better access is to stuff packs, soaked with scolicide, into the right subphrenic space, so as to push the liver down. If you know exactly where the cyst is, a subcostal incision may be even better.

The cysts present as smooth white swellings. Look and feel carefully to find how many there are. The rest of the liver is usually large, smooth, and distended.

Select a place where the cyst is close to the surface, and isolate it with carefully placed scolicide-soaked packs. Suture a tubular polythene sheath 6x15cm with 3/0 silk to the pericyst without puncturing the cyst. Aspirate through the polythene sheath at least half the fluid with a 1·5mm needle (15-10C).

If the needle blocks, clear it by injecting a little scolicide. If needle aspiration does not work, use a trocar and cannula. If necessary, try introducing a multi-holed catheter through the cannula. This will however increase the risk of spillage. If many daughter cysts block the sucker, it is probably multilocular.

Collect the fluid in a bag containing an excess of scolicide. *Don't use an open dish as fluid may spill!* Keep a record of the volume you withdraw, as a guide to how much scolicide you will need to inject. Leave the needle in place and inject 50-100ml scolicide back into the cyst until it becomes tense. Then tie a purse-string suture snugly round the puncture site, and remove the needle. Wait for 10mins for the contents of the cyst to become sterile.

Re-aspirate the cyst till it becomes flaccid and open it through the part which presents most easily. Insert the tip of the sucker, and suck out the remaining fluid. Record-sized cysts have had over 40L! Using a kitchen spoon, remove all the daughter cysts, sediment and debris (15-10E). Then, using finger dissection, find the natural plane of cleavage between the ecto- and the endocyst.

To get adequate exposure, you may have to incise the cyst across the full width of its bulge, and deroof it. Remove the yellow laminated membrane of the endocyst completely, piece by piece, with sponge forceps (15-10F). There will be little bleeding.

CAUTION!
(1) Try not to rupture the daughter cysts as you remove them, because their contents are probably still infective.
(2) *Don't try to remove the ectocyst*; it is tightly stuck to the liver, and will bleed.

Swab the inside of the cyst with packs soaked in scolicide, and explore it for secondary cavities. If you find any, repeat the process of aspiration and injection.

If the cavity is small, you may be able to bring its edges together with mattress sutures.

If the cavity is large, you can:
(1) Suture any obvious small bile duct openings, and leave it alone, which is probably the wisest method.
(2) Fill it with ordinary 0·9% saline, and suture it.
(3) Saucerize it by excising the protruding portion. The difficulty with this is that the cut surfaces of the liver will bleed.
(4) Fill it with a graft of omentum. Dissect a strip of omentum on a vascular pedicle and stitch this into the cavity. The omentum will swell to fill the space and absorb the fluid from the cavity.
(5) Bring the walls of the cyst together with multiple absorbable sutures, so that the cavity is obliterated.
Drain large cavities for about 10 days, especially if you have not been able to remove all the endocyst.

CAUTION!
(1) *Don't let the untreated cyst fluid spill*, or daughter cysts will form in the peritoneal cavity.
(2) If the needle hole leaks, insert a purse-string suture.

If the cyst is a simple non-hydatid cyst, you can simply deroof it and obliterate the cavity with omentum. Make sure you control bleeding from the cut cyst edge with a running suture or diathermy.

DIFFICULTIES WITH HYDATID CYSTS

If there are signs of peritonism with an anaphylactic response, shock, and dyspnoea, the cyst has ruptured into the peritoneal cavity. Use hydrocortisone 0·5-1g IV, and adrenaline 1:1000 1ml SC.

If the blood pressure falls alarmingly and there is no other reason for it, the cause is probably an anaphylactic reaction. Treat as above. Alternatively the cyst may have eroded into a major vessel such as the inferior vena cava or portal vein: apply pressure with your fingers and get suction, bulldog clamps *etc* as for a vascular injury.

If there is generalized abdominal swelling, consider the possibility of HYDATIDOSIS OF THE PERITONEAL CAVITY. At operation you may find the peritoneal cavity distended with hydatid cysts, and remove bucketfuls of them. Try to remove as many of them as you can. Then flood it with liberal quantities of 0·04% chlorhexidine solution as a scolicide. Aspirate this and then irrigate it with saline to wash out the remaining scolicide. The surface of the bowel may be seeded with small white nodules. Make sure you start praziquantel and albendazole unless already started.

If there is a cyst in the omentum, try to remove it entire, with part of the omentum. If you cannot do this, aspirate and inject scolicide, as for the liver.

If there is a cyst in the spleen, perform a splenectomy (15.17).

If there is a cyst in the kidney, aspirate under ultrasound guidance, and introduce scolicide as above.

If there is obstructive jaundice, perhaps with cholangitis, aim to drain the common bile duct (15.7). Remove all hydatid sludge, debris, and daughter cysts from the duct, and irrigate it thoroughly. Drain the common bile duct with a T-tube.

If a cyst is leaking into the peripheral bile ducts, sterilize, evacuate, and drain the cyst. An external biliary fistula may develop, which may improve cholangitis which is likely to be present.

If a fever develops post-operatively, the cyst cavity is probably infected. Treat with gentamicin or chloramphenicol.

If there are features of a liver abscess – a swinging fever, anorexia, and increasing pain – suspect that a hydatid cyst has become infected. Open it, saucerize it, and drain it (15.10). Infection will have destroyed the hydatids.

If there are recurrent cysts, they will probably take about 3yrs to develop, be multiple, and be in the abdominal cavity. Distinguish recurrent cysts from the manifestation of an unsuspected second cyst. Start medical treatment.

If there are cerebral cysts also, use praziquantel in higher doses (60-90mg/kg bd for 30 days), together with dexamethasone 4mg qds.

If a cyst is calcified and asymptomatic, leave it alone.

15.13 Pancreatitis

Both acute and chronic pancreatitis are not uncommon; rarely a pancreatic pseudocyst (15.14) or abscess (15.15) develop as complications. If the patient does not recover 1wk after an acute episode, suspect such complications.

Pathologically, **acute** pancreatitis varies from oedema and congestion of the pancreas to its complete autodigestion, with necrosis, haemorrhage, and suppuration. Less severe forms may go on to form a tender, ill-defined mass in the epigastrium.

The main symptom is pain, which can vary from moderate epigastric discomfort to an excruciating, penetrating agony, which bores through to the back, and needs high doses of opioids to relieve it. There is tenderness in the epigastrium, perhaps with guarding. Vomiting, paralytic ileus and the outpouring of fluid into the retroperitoneum may result in severe fluid loss. If you see bruising around the umbilicus or in the flanks, this indicates intra-abdominal haemorrhage and means the pancreatitis is severe.

The underlying cause is usually:
(1) alcohol abuse (especially spirits), or
(2) gallstones (or 'sludge'), and occasionally,
(3) *ascaris* worms,
(4) liver flukes,
(5) leptospirosis,
(6) HIV disease (including treatment with ARV therapy),
(7) mumps or *coxsackie* virus,
(8) hypercalcaemia, hyperlipidaemia,
(9) systemic lupus or cystic fibrosis,
(10) trauma (including endoscopic instrumentation),
(11) drugs such as sulphonamides, thiazides, œstrogens,
(12) poisons such as organophosphates,
(13) *tityus* scorpion envenomation (in Trinidad, Brazil and Venezuela),
(14) typhoid (rarely).

You may diagnose pancreatitis clinically, or from laboratory tests, or only when (*in error*) you perform a laparotomy for an acute abdomen. Estimating the serum amylase is not difficult, and your laboratory should be able

to do it, because *you should avoid a laparotomy for pancreatitis at all costs!*

Chronic relapsing or recurrent pancreatitis is one of the causes of a severe chronic upper abdominal pain. It is quite common in alcoholics anywhere, and there is a calcific type found much in Kerala and Orissa in India, but also in Japan and sub-Saharan Africa; the pancreas calcifies and its endocrine function deteriorates. A longitudinal pancreatico-jejunostomy is helpful if the pain is debilitating, but this is formidable surgery indeed. Chronic pancreatitis may also be due to tuberculosis (16.5).

ACUTE PANCREATITIS

DIFFERENTIAL DIAGNOSIS includes:
(1) gastritis,
(2) perforated peptic ulcer (13.3),
(3) acute cholecystitis (15.3),
(4) rupture of a liver abscess (15.10),
(5) strangulating upper small bowel obstruction (12.2).

SPECIAL TESTS. Serum amylase rises within hours of the start of the pain, and remains high for about 2 days. A level of >1,000 Somogyi units is almost diagnostic. A peritoneal tap in the right lower quadrant will confirm the diagnosis: the aspirate may be straw-coloured, or reddish-brown, but its amylase is always high.

In severe acute disease (Ranson's criteria):
(1) the white count is >16000/mm³
(2) the glucose is >10mM; after 48h,
(3) the haematocrit drops >10%,
(4) the urea rises >10mM,
(5) the serum calcium drops <2mM,
(6) the pO_2 drops <60 mm Hg with a metabolic acidosis.

Hyperglycaemia is common with chronic calcific pancreatitis.

(The Balthazar score gives the severity of the pancreatitis in terms of the degree of inflammation and necrosis seen, but this needs contrast-enhanced CT scanning.)

RADIOGRAPHS are not very helpful but may show pancreatic calcification, if there have been previous attacks; and a left pleural effusion, or distended loops of bowel from ileus.

ULTRASOUND demonstrates oedema, and fluid collections (or even necrosis) of the pancreas (38.2d). You may see gallstones, and especially 'sludge' in the common bile duct, or a completely different diagnosis!

TREATMENT
If pancreatitis is mild, it will settle by a regime of restricting oral intake, with nasogastric drainage, analgesia and IV fluid replacement.
If pancreatitis is severe, replace fluid loss energetically with large volumes of 0·9% saline, Ringer's lactate, or a plasma expander. Correct electrolyte losses. Monitor the urinary output, the haematocrit, and if possible, the central venous pressure. Monitor glucose; start an insulin sliding scale. The pain may be overwhelming: treat with large doses of opioids, supplemented by diazepam or promethazine. Administer oxygen by mask; consider mechanical ventilation. Prophylactic antibiotics, especially against *staph aureus*, are advisable. You may need blood transfusion later.

If you are sure of the diagnosis, *don't operate*; *but it is better to operate unnecessarily than not to operate on a case of strangulated bowel, for example.* The amylase level may rise with necrotic bowel. Severe cases may benefit from lavage with saline, *via* tubes which can be inserted in the flanks under LA (13.3), but attacks tend to recur. Formal open lavage is then better.

If you do open the peritoneum, you will know that there is pancreatitis when you see areas of whitish-red fat necrosis on the transverse mesocolon, or omentum, and the exudate described above. The pancreas feels swollen and oedematous, and may contain greenish-grey necrotic areas. The fat necrosis may be confused with TB, so take a biopsy. Drains by themselves don't help, but you may use them for continuous peritoneal lavage.

If the abdomen is difficult to close, leave it open as a laparostomy putting on a vacuum dressing (11.10, 11-20) or make fasciotomies in the rectus sheath.

DIFFICULTIES WITH ACUTE PANCREATITIS
If you find that there are also gallstones, consider doing a cholecystectomy (15-4) later. *Don't be tempted to remove the gallbladder in the acute phase*.

If, during the course of 2-3wks, septicaemia develops, suspect that a pancreatic abscess (15.15) is developing, especially if there is haematemesis.

If deterioration continues, your only chance will be to refer to a centre where CT scanning is available: a pancreatic necrosectomy may be life-saving.

If respiratory or renal failure develop, usually in the first 48h (5-10% chance), death is likely. There is little you can do except treating with oxygen, plenty of IV fluids, and plasma expanders. Monitor the CVP, ventilate mechanically, add broad-spectrum antibiotics and nutritional support.

15.14 Pancreatic pseudocyst

A large watery pancreatic exudate sometimes collects in the lesser sac. This has no epithelial lining, hence the term 'pseudocyst'. It usually presents >3-4wks after an abdominal injury, or an attack of acute pancreatitis, with a mass in the abdomen and epigastric discomfort or pain. There may be toxaemia with fever and tachycardia, but the degree of sickness is much less than in acute pancreatitis, or a pancreatic abscess. The mass usually distends the abdomen: it may extend right across the epigastrium, and

reach down to the umbilicus or beyond it. It is not usually tender, but may be quite tense and not fluctuant if very large. Sometimes, there are symptoms of pancreatic insufficiency, with steatorrhoea.

You can drain the cyst into the stomach, jejunum, or percutaneously into a bag. The correct timing of these procedures is important because the cyst has to mature to hold sutures. It may not do so properly in HIV disease. Draining it is less urgent than draining a pancreatic abscess (15.15), and there is less chance of complications. External drainage usually results in leakage and skin excoriation, *and should be used only as a last resort*; cystogastrostomy is simple but cystojejunostomy is the definitive operation, and you should be able to manage this if you are confident with bowel anastomoses.

SPECIAL TESTS. The amylase level in the cyst fluid is grossly raised.

ULTRASOUND shows a large cyst easily, though you may have difficulty differentiating it from a liver cyst (38.2b,d).

RADIOGRAPHS are less useful: you can make the diagnosis by adding oral contrast: a lateral film of barium in the stomach will show a mass bulging into the contrast from behind. There is gross widening of the normal contour of the duodenum. You may see patches of calcification in the pancreas. Or, insert a nasogastric tube and inject 200ml of air into the stomach and take a lateral supine view of the abdomen. In a pancreatic pseudocyst the stomach is displaced forwards; in a liver abscess, backwards.

DRAINAGE FOR PANCREATIC PSEUDOCYST (GRADE 2.4)
TIMING
Don't operate until >6wks after an attack of pancreatitis, by which time the cyst wall will be mature enough to take sutures. Once a pancreatic pseudocyst is palpable, it rarely disappears spontaneously. Operate as soon as possible after 6wks; if you leave it too long, it may bleed, rupture, become infected, or destroy much of the pancreas.

DIFFERENTIAL DIAGNOSIS includes:
(1) liver abscess (15.10),
(2) hepatoma (15.11),
(3) hydatid cyst (15.12),
(4) renal cyst or hydronephrosis (27.14),
(5) gastric outlet obstruction (13.7),
(6) Burkitt's lymphoma (17.6),
(7) abdominal tuberculosis (16.1),
(8) aortic aneurysm (35.8).

Rarely, a pancreatic carcinoma can form a cyst.

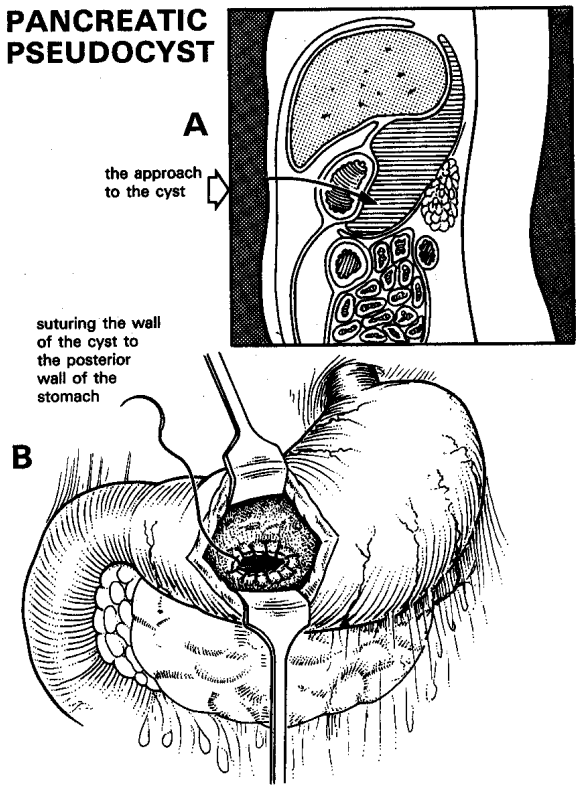


Fig. 15-11 PANCREATIC PSEUDOCYST.
A, approach to the cyst through the anterior and posterior walls of the stomach. B, suture the wall of the cyst to the posterior wall of the stomach to control bleeding.
After Maingot R, Abdominal Operations, HK Lewis 4th ed 1961 p.557 Fig. 26, permission requested.

It may be necessary to drain the cyst earlier if it is about to rupture, if it causes persistent intractable vomiting, or has started to bleed.

You can drain the cyst percutaneously under LA; make sure you put two purse strings to hold the drain and invert the cyst around the drainage tube as for a gastrostomy (13.9). The problems are leakage and resulting skin excoriation. External drainage is a temporary measure if you are unable to perform a formal internal drainage procedure.

CYSTOGASTROSTOMY FOR PANCREATIC PSEUDOCYST (GRADE 3.2)
PREPARATION
In dehydration, anorexia, or toxaemia, administer IV fluids and high-calorie, high-protein enteral feeding for a few days pre-operatively. Insert a nasogastric tube the evening before the operation, and wash out the stomach thoroughly.

INCISION
Make a midline incision. Choose an area on the anterior wall of the stomach that is overlying the cyst. Use a knife or diathermy to start a 4cm incision in the long axis of the stomach between 2 Babcock forceps. Enlarge it with scissors. Clamp any briskly bleeding vessels, and retract the edges of the incision, so that you can inspect the posterior wall of the stomach. Suck it empty.

After opening the stomach, aspirate through its posterior wall into the cyst. Expect to find a mildly opaque straw-coloured or murky brownish fluid. If so, insert a small haemostat through the hole in the stomach into the cyst, and open it so as to enlarge the opening to 3cm. Suck out the fluid: expect to aspirate up to 4L.

CAUTION! Don't incise the cyst widely; it may bleed severely.

N.B. **If you aspirate fresh blood**, the 'cyst' may be an aortic aneurysm: *stop!* Get an ultrasound scan (38.2e).

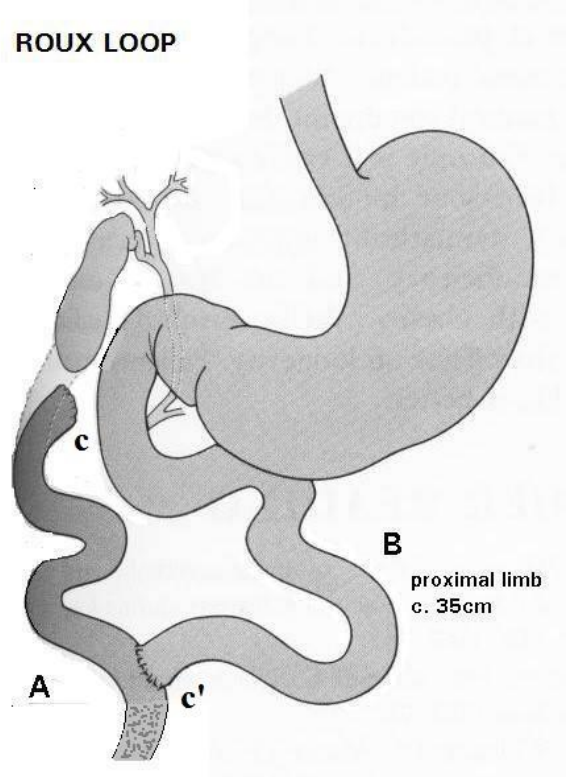


Fig. 15-12 THE ORIGINAL ROUX LOOP.
A, the distal afferent (Roux) loop, which you can use to drain the gallbladder, bile duct, pancreatic cyst, or stomach. B, the proximal efferent loop. The small bowel has been divided at c, and the proximal part anastomosed end-to-side at c'. Here the plan is to anastomose the Roux loop to the gallbladder.

If you have waited the 6wks for adhesions to form and the cyst wall to mature, there will be no need to suture the stomach wall to the cyst, as they will already be tightly stuck together. But control brisk bleeding from the stomach edge; so quickly oversew the opening all round with a continuous interlocking haemostatic suture of 2/0 non-absorbable. *Don't use absorbable suture,* because pancreatic juice digests it.

When you are sure the posterior opening in the stomach is no longer bleeding, close the anterior opening in two layers, the first a full-thickness haemostatic continuous layer of 3/0 absorbable sutures, and the second a seromuscular Lembert layer of continuous non-absorbable suture. Close the abdominal wall in the usual way.

CYSTOJEJUNOSTOMY-EN-Y FOR PANCREATIC PSEUDOCYST (GRADE 3.5)

INCISION. This is the definitive operation. Proceed as above; after opening the cyst, lift up the proximal jejunum and look at its blood supply; select a point 30cm from the ligament of Treitz at which to divide the bowel so that the distal loop easily reaches the pseudocyst. Divide the bowel between non-crushing clamps. You will need to divide some of the vascular arcades in the mesentery to mobilize the distal bowel (the Roux loop) adequately. When you are satisfied there is no tension, make an end-to-side anastomosis of the loop to the opening in the pseudocyst. Then anastomose the proximal cut end of jejunum to the right side of the Roux loop some 20cm along its length with an end-to-side anastomosis, making sure the bowel lies comfortably. Close any mesenteric defects carefully avoiding damage to the fine mesenteric vessels.

SIMPLE CYSTOJEJUNOSTOMY FOR PANCREATIC PSEUDOCYST (GRADE 3.4)

INCISION. This is a simpler, but less satisfactory, procedure. Make a midline incision. Open the lesser sac to get access to the pancreatic pseudocyst; check that its wall is thick enough to hold sutures. Then pack off the abdominal contents with large swabs, open the cyst by 2cm only and drain its contents. Bring a loop of proximal jejunum up to the cyst and make a side-to-side anastomosis (as for a gastrojejunostomy (13-16)).

POST-OPERATIVELY, restrict oral intake with nasogastric suction for 3-4 days, until flatus is passed; then start oral fluids, followed by a soft diet.

15.15 Pancreatic abscess

This is a dangerous complication of acute pancreatitis (15.13) or perforated duodenal ulcer. Occasionally it results from severe pancreatic injury. A collection of pus, necrotic tissue, and clot fills the lesser sac; it enlarges behind the peritoneum, it expands anteriorly to obliterate the lesser sac, and it pushes the stomach and transverse colon forwards. It may present with a catastrophic haematemesis.

If the abscess develops during the course of an attack of pancreatitis, the diagnosis is usually obvious, but it may be difficult otherwise. So if ever a severely sick patient has an ill-defined deep-seated epigastric mass, remember that there might perhaps be a pancreatic abscess.

SPECIAL TESTS. The urinary and serum amylase are usually high. Check the glucose.

ULTRASOUND. A fluid-filled cavity is seen adjacent to the pancreas, as a pancreatic pseudocyst (38.2d). *Aspiration is difficult and hazardous, so is not recommended*.

LAPAROTOMY FOR PANCREATIC ABSCESS (GRADE 3.4)

PREPARATION. Resuscitate with adequate IV saline or Ringer's lactate. Correct hyperglycaemia with insulin. Administer oxygen, bicarbonate and calcium in seriously ill patients. Catheterize the bladder. Treat with gentamicin, ampicillin and metronidazole. Cross-match blood. Decompress the stomach with a nasogastric tube.

EXPLORATION. Make an upper midline incision from the xiphisternum to beyond the umbilicus. Open the abdominal cavity with care, because the mass, or the stomach or colon, may have stuck to the abdominal wall. You may find it difficult to know what you are seeing. Dissection is difficult and dangerous, because the tissues are so vascular and oedematous. Lift and free the abdominal wall from the organs under it, and insert a self-retaining retractor. Feel for the upper border of the abdominal mass. Try to find a place where you can incise it without injuring anything. This will usually be through the lesser omentum, or better, the transverse mesocolon to the left of the ligament of Treitz. When you have decided where to drain, seal the area from the rest of the peritoneum with large moist packs. Using a syringe and a large needle, aspirate the place where there seems to be the thinnest layer of tissue between the abscess and your finger. Take pus for culture. If you find pus under pressure, decompress the abscess with suction. Enlarge the abscess so that you can insert two fingers, but *don't try to dissect further*. Wash out any floating solid matter.

CAUTION! Don't disturb the necrotic pancreatic tissue at the bottom of the abscess – it will bleed!

Place 2 catheters in the abscess cavity, and bring them out through stab wounds. Bring one out anteriorly, and the other as far back as possible, in the most 'dependent' position. Use these to irrigate the abscess cavity continuously (about 2L in 24h). Make a feeding jejunostomy (11.7), because oral feeding will not be possible for 3wks, and you will probably be unable to feed parenterally. Feeding through a jejunostomy results in less secretion of gastric juice than feeding through a gastrostomy.

POST-OPERATIVELY
Continue nasogastric suction, fluids, and antibiotics until the temperature is normal. Chart the daily drainage output. *Don't be in a hurry to remove the drains*, even if leaving them in does seem to increase the risk of a fistula. Allowing pus to collect again is a greater risk. If the wound is looking fairly clean, close it by secondary suture in 7-10 days.

CAUTION! This is heroic surgery! A pancreatic abscess carries a 30-50% mortality, and often reforms, even with adequate drainage. If so, be prepared to re-operate 3 or 4 times if necessary. However, if you don't operate, death is inevitable!

DIFFICULTIES WITH PANCREATIC ABSCESS
If pancreatic juice is still discharging after 2wks, leave the catheters *in situ* for a month and then withdraw them slowly 5cm per day.

If a pancreatic fistula develops, collect the juice in a stoma bag with the orifice carefully cut to size to prevent skin excoriation. This can be mixed with jejunostomy feeds if malabsorption is a problem.

If bleeding becomes severe, try pinching the vessels in the free edge of the lesser omentum between your fingers and pack the pancreatic area. Leave the packs for 48h and remove them at a second laparotomy, when bleeding should be much less.

15.16 Pancreatic carcinoma

If the carcinoma is in the head of the pancreas (70%), it may obstruct the common bile duct, so that presentation is with painless progressive obstructive jaundice (15.9). If it is in the body of the pancreas (30%), presentation is with upper abdominal and back pain and general symptoms of malignancy. Spread to the lymphatics and surrounding structures is early, and 10% of patients develop ascites. Thrombophlebitis migrans (thrombophlebitis in any superficial vein appearing, resolving, and then appearing again elsewhere) may occur with any malignant tumour, but is particularly common with this one.

Radiotherapy, chemotherapy, and surgery are of little value, but differentiation with tuberculosis is important. Taking a biopsy of the pancreas itself may cause pancreatitis and worsen the condition, so try to get a piece of tissue from an adjacent node or piece of omentum. Sometimes carcinoma of the pancreas cannot be easily distinguished from chronic pancreatitis (15.13) or tuberculosis.

15.17 Surgery of the spleen

If you operate on the spleen, you often end up removing it. The indications for doing so (apart from trauma) must be good, because the spleen of a tropical patient is commonly large, and may be so firmly stuck to the diaphragm that:
(1) exposing it is difficult.
(2) bleeding is likely from the vascular adhesions that join it to the diaphragm.

If there is portal hypertension, the vena cava carries high pressure venous blood which escapes into the systemic circulation.

Remember the spleen protects against subsequent infection, especially *pneumococcal* pneumonia, malaria and dog and cat bites with *Capnocytophaga canimorsus* (a common commensal) which may rapidly be fatal. Infection is even more common if HIV is present.

INDICATIONS (OTHER THAN FOR TRAUMA) FOR SPLENECTOMY

(1) **Spontaneous rupture**
This is rare, but may occur typically in massive malarial (*not bilharzial*) splenomegaly; though it may be truly spontaneous, *e.g.* in mononucleosis, the patient may not remember any trauma because it was slight. It may occur after colonoscopy. Signs are of shock, left upper abdominal pain and abdominal distension, although initially there may be felt a *reduction* in abdominal girth as a large spleen bleeds.

(2) **Hypersplenism**
A big spleen may be responsible for removing all the blood cell lines, resulting in anaemia, leucopenia and thrombocytopenia of varying severity, though the thrombocytopenia is commonest. The problem is that the same picture can be shown by HIV disease, which itself can give rise to splenomegaly. Thrombocytopenia is a serious hazard in surgery and so you need to have some experience before attempting a splenectomy without platelets available for transfusion. However, your hand may be forced if a patient continues to bleed elsewhere because of the thrombocytopenia, particularly if there is portal hypertension.

If the underlying cause is HIV disease, blood counts will improve with ARV therapy (5.8) without splenectomy. You should anyway be wary of removing a spleen when there is HIV disease because of the risks of infection; you may be able to make the distinction between hypersplenism and HIV-pancytopenia by looking at a bone marrow film: this is normal or *hyperplastic* in hypersplenism, but *hypoplastic* in HIV disease.

(3) **Chronic idiopathic thrombocytopenia**
Here the low platelet count is *not* associated with an enlarged spleen but through destruction of platelets by antibodies. There are often ‘megathrombocytes’ in the peripheral blood film and raised numbers of megakaryocytes in the bone marrow.

It occurs more often in females than males (4:1); there is usually quite a good response to prednisolone, but eventually this option becomes undesirable in the chronic case.

However, splenectomy may not benefit 20% of cases. *Don’t do it if systemic lupus erythematosus or rheumatoid arthritis is the cause of the chronic thrombocytopenia*. *Never do it for the acute thrombocytopenia, and especially not where thrombocytopenia is due to drug sensitivity* (especially quinine). If you have access to radiotherapy, this may be the safer option.

(4) **Haemolytic anaemias**
There are several haemoglobinopathies, including sickle cell trait (*never sickle cell disease*) thalassaemia, and spherocytosis for which splenectomy may be beneficial, but their selection needs the advice of an expert.

(5) **Splenic torsion** or **wandering spleen**
These are both very rare and arise when the spleen has an abnormally long pedicle and no adhesive attachments. You are unlikely to make the diagnosis before you operate. Torsion of the spleen tends to occur during pregnancy when it is confused with an ovarian tumour!

(6) **Hydatid cyst** (15.12).

(7) **Malignancy**
HIV-ve lymphoma, *Schistosomiasis*-related giant follicular lymphoma, chronic myeloid or lymphatic leukaemia, or fibrosarcoma affecting only the spleen, but you should get advice from an expert.

KASHY (20yrs) complained of a swelling in the right iliac fossa. Ordinarily, it was painless but during attacks of 'fever' it became painful and tender. At laparotomy, the whole spleen was found to be in the right iliac fossa, but the splenic vessels crossed the abdomen to their normal position. The 'wandering spleen' was removed easily.
LESSON Some rare conditions have easy solutions.

CONTRAINDICATIONS TO SPLENECTOMY

(1) **Splenic abscess** (15.18) occurs occasionally. It starts acutely, it may become chronic, and it shows up radiologically as a fluid level in an irregular space. Ultrasound shows the spleen to be filled with dense fluid. The pus is sterile usually, but may be secondary to amoebiasis, brucellosis, salmonellosis, leptospirosis or candidiasis. There may be sickle cell disease, acute myeloid leukaemia or pancreatitis. Carefully pack away other abdominal viscera, and drain the abscess: don’t try to remove the spleen.

(2) **Tropical splenomegaly syndrome**
This is an immune response to recurrent attacks of malaria, and is responsible for nearly all large spleens in malarious areas. It responds to long courses of antimalarials. *Don’t remove such spleens unless hypersplenism is a complication.*

(3) **Splenic tuberculosis** (16.5)
Anti-TB therapy should make splenectomy superfluous; fibrosis during healing will shrink the spleen. HIV disease is particularly likely.

(4) **Leishmaniasis** (34.7). Medication should likewise make splenectomy unnecessary.

(5) **Acute diseases**, *viz.* leptospirosis, acute thrombocytopenia.

(6) **HIV disease** (5.6)
Whilst not an absolute rule, the risk of infection increases, and so the indication must be really good. Institute ARV treatment first.

CAUTION!
(1) *Don’t operate lightly*; your only absolute indications for doing so are the first three.
(2) If the spleen is huge, think seriously about operating because it may need a thoraco-abdominal approach.

SPLENECTOMY (GRADE 3.4)

PREPARATION
Make sure you have a wide-bore cannula in place for fluid replacement; have blood cross-matched (but don't be in a hurry to raise the haemoglobin if this has been chronically low), and a nasogastric tube to decompress the stomach. Provided there is no allergy, always give prophylactic penicillin perioperatively. Place a sandbag or pillow behind the left thorax to rotate it to the right.

INCISION. Make a midline or left subcostal incision: this will give better direct access to the spleen, and you can extend this as a Chevron with a right subcostal incision. Alternatively for a very big malarial spleen, you can make a left thoraco-abdominal incision, because this allows you to see vascular diaphragmatic adhesions directly (11.1). Or you can turn the patient to the right, by propping up the left flank; this allows a huge spleen to 'fall out' of the abdomen more easily when mobilized.

Doing an elective splenectomy is different from an emergency splenectomy in that you can approach the spleen in a relaxed manner. Explore the abdomen, noting the condition of the liver and presence of any lymph nodes, as well as the mobility of the spleen. If it is adherent, and large, it is wisest to open the lesser sac and tie off first the splenic vessel artery and then the splenic vein before proceeding.

You can improve access by placing a pack behind the spleen if there are insignificant adhesions. Otherwise divide the peritoneal attachments. If bleeding is a problem at this stage, deliver the spleen, rotate it forwards to the right and tie a thick ligature right round the entire splenic pedicle. This is safer than trying to grasp it with a large clamp. As you do so, try not to damage the stomach, and try to cause the least possible damage to the pancreas. When you have controlled bleeding, proceed to tie the vessels individually.

Enter the lesser sac by opening a window in the greater omentum, and lift up the stomach off the pancreas by dividing the adhesions between them a short distance. Then feel for the splenic artery along the upper border of the pancreas. Incise the peritoneum over it, pass an angled haemostat underneath it and pass ligatures round it.

Carefully expose the splenic vein by dissecting away the fatty tissue in the pedicle. Then tie off the splenic artery, tie the vein and divide the splenic pedicle. For extra security, apply a second set of ligatures at the same point before the vessels divide. *Don't leave a long splenic vein stump which may thrombose!*

CAUTION! Make sure you pass the ligatures and tie them before dividing the vessels. If you use haemostats and the cut vessel drops off and is lost in a pool of blood, you may never find it again.

If you haven't been able to draw the spleen forwards, do so now, if necessary by dividing adhesions to the parietal peritoneum. Then divide the peritoneum lateral to the spleen (the lienorenal ligament); put your finger into the opening and gently free the spleen. You can now bring it well outside the abdomen. Free the splenic flexure of the colon from the spleen and separate the tail of the pancreas from the splenic vessels. Free the spleen from its attachments to the greater curvature of the stomach by dividing the short gastric vessels individually in the gastrosplenic ligament.

CAUTION! Don't include an area of stomach wall with your ligatures, especially at the upper margin of the spleen.

Remove the spleen, keeping it in saline for possible autotransplantation (see below), and put a big dry pack in the splenic bed. Leave it there for 5mins; then remove it and look for any bleeding vessels and tie them off. If the operative site is not absolutely dry and you are uncertain about an injury to the tail of the pancreas, place a large drain in the splenic bed.

SPLENIC IMPLANTATION. *This is controversial and may not work.* Keep some slices of spleen, say 5x3x0·5cm, and implant them under the peritoneum in the side wall of the abdomen, or in the anterior abdominal wall. Or, place some 1cm cubes of spleen within a sandwich of omentum. They will usually take, and may reduce the severity of attacks of malaria and perhaps the danger of septicaemia, but will not fully protect against *pneumococci*.
The risk of adhesions is much less than the risk of life-threatening infections. You can check if the splenic slices are functional after 4wks if there are no Howell-Jolly bodies and no target cells in the peripheral blood film, and the platelet count is normal.
Don't do this with splenectomy indications 4,5,6 or if there is already an accessory spleen.

SEGMENTAL SPLENECTOMY (GRADE 3.5)
Preserving a functional remnant of c.20-30% of the spleen avoids septic and immunological problems also; it can also be used in hypersplenism. You need to devascularize anatomical segments (especially middle and lower portions) of the spleen, by ligating branches of the splenic artery at the hilum.

Wait for 5mins to observe a line of demarcation, and cut it along this line in a V-shape whilst squeezing the proximal portion manually. A vascular clamp across the pedicle will reduce bleeding. Then sew over the remnant an omental patch, bringing the edges of the 'V' together. This is not easy surgery.

DIFFICULTIES WITH SPLENECTOMY
If you have damaged the stomach or the colon, close the perforation in two layers with long-acting absorbable suture; make sure nasogastric suction is in place. Start gentamicin and metronidazole.

If you have damaged the pancreas, suture a piece of omentum over the damaged segment and leave a drain. Try to monitor amylase levels of fluid draining.

If bleeding from the diaphragm persists, insert one or two dry packs for 10mins by the clock; then come back and remove them to see if you can identify the bleeding vessels to tie them. If you still fail to control bleeding, leave packs

tightly *in situ*, close the abdomen, and return after 48h to remove them, by which time the bleeding will almost certainly have stopped.

If shock suddenly develops post-operatively, a ligature has probably come off. Re-open the abdomen immediately, with good suction available, to try to find the bleeding vessel, and tie it off.

If the wound sloughs and there is a fluid discharge, the tail of the pancreas or stomach may have been injured. Re-open the wound, and suture a piece of omentum over the damaged viscus; *don't try to ligate the pancreas or suture the gastric perforation* as sutures will probably cut out and cause more damage.

If there is fever with no obvious cause, and you have given penicillin, check for a subphrenic abscess (10.2).

If there is rapidly developing haemolytic anaemia and fever, suspect malaria or babesiosis, a tick-borne illness usually affecting domestic or wild animals, and get both thick and thin blood films. Use quinine 650mg orally tds and clindamycin 600mg orally tds for 5-10 days.

If there is respiratory distress post-operatively, think of a pneumothorax.

POST-OPERATIVE CARE AFTER SPLENECTOMY
Treat with penicillin or ampicillin prophylactically intra- and post-operatively for 2wks, and longer in children <2yrs. Follow protocols for *pneumococcus*, *haemophilus* and *meningococcus* vaccine if possible. *Insist on malaria, meningitis and tick-bite fever prophylaxis in endemic areas, including in the perioperative period.*

15.18 Splenic abscess

This starts acutely, though it may become chronic. It occurs in sickle cell disease, acute myeloid leukaemia, HIV+ve patients with candidiasis, and secondary to amoebiasis, brucellosis, salmonellosis, leptospirosis and occasionally as a result of acute pancreatitis or perforation from a gastric ulcer. The danger is rupture into the peritoneal or pleural cavity which may be accompanied by disastrous bleeding.

There is left upper abdominal pain, a dragging sensation in the abdomen, discomfort after meals, anorexia, and a palpable tender mass extending down from the left upper quadrant towards the right lower quadrant. This might be huge. There is usually anaemia and a low-grade fever. The mass may be tympanic because it usually is filled with gas, arising from oxygen liberated by red cells in the spleen, and carbon dioxide from tissue metabolism rather than gas-forming organisms.

SPECIAL TESTS. Leucocytosis and anaemia are usual, but the white count may not be raised with HIV disease. In Southeast Asia, check for melioidosis (6.10).

CHEST RADIOGRAPH. An erect film will show a gas bubble and fluid level below the left hemidiaphragm, pushing it upwards.

ULTRASOUND. Look for a fluid-filled cavity in the area of the spleen (38.2g). Much of the splenic tissue may be destroyed. Aspiration will confirm the diagnosis, but beware of causing severe haemorrhage or rupturing the colon. You may be able to aspirate a splenic abscess completely, and avoid surgery. Treat with antibiotics 48h beforehand and then follow a procedure as for a liver abscess (15.10).

LAPAROTOMY FOR SPLENIC ABSCESS (GRADE 3.1)

INDICATIONS. Essentially all splenic abscesses need surgery.

PREPARATION. Cross-match blood and start pre-operative penicillin and metronidazole.

EXPLORATION. Make a left subcostal incision or Chevron (inverted-V double subcostal) for very big abscesses. Pack away the rest of the abdominal contents. Drain the abscess and leave a large tube or catheter in the space, passed through a separate stab incision in the lateral abdominal wall. Lavage the abdomen with warm saline or water.

DIFFICULTIES WITH SPLENIC ABSCESS
If access is difficult and you have made a midline incision not expecting a splenic abscess, extend it as an inverted-L laterally with a left subcostal arm, as a left-sided Kocher incision (11-1).

If serious bleeding from the spleen ensues, pack the area and press firmly. Get assistance. Place a further pack (or two) above and behind the spleen to lift it forward, and perform a splenectomy (15.17). If you are experienced, this is the definitive operation rather than simple drainage.

If you find a gastric perforation, the tissues will be very friable. *Don't attempt to close the perforation with sutures*, but stuff a segment of omentum into the hole, and fix it there with absorbable sutures. Make sure you drain the stomach with a nasogastric tube. You are unlikely to be able to save the spleen because unravelling the inflamed tissues will damage it and result in considerable bleeding. Grasp the hilum of the spleen, place a pack or two behind it, and perform a splenectomy (15.17).

16 Abdominal tuberculosis

16.1 Introduction

Abdominal TB is common and becoming more so, especially with HIV disease. Extra-pulmonary TB will suggest HIV disease in 90% of cases where TB was previously not so common. It is responsible for about 10% of bowel obstruction in some parts of the world, *viz.* India and Nepal. You may see it when you are expecting something else.

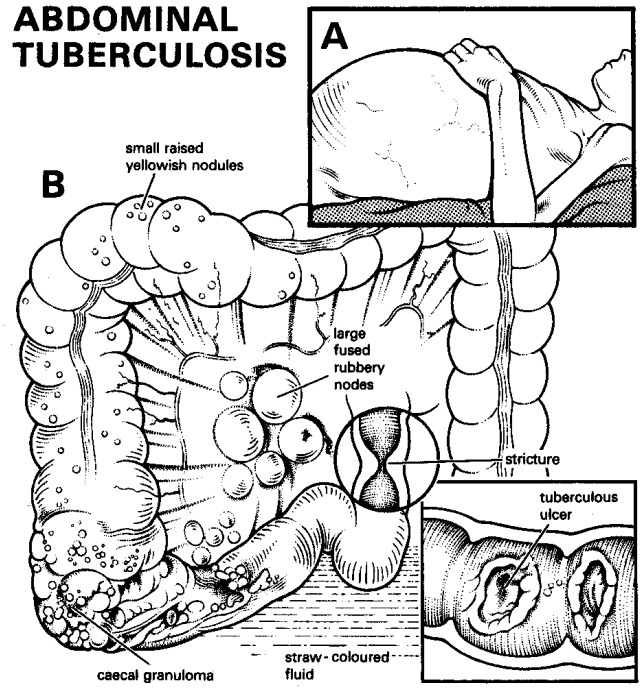


Fig. 16-1 ABDOMINAL TB can present in many ways. Patient A's abdomen is distended with ascitic fluid. You may not be able to diagnose some of the other forms of tuberculous peritonitis until you do a laparotomy.
Kindly contributed by Gerald Hankins.

There are 4 main types:
(1) The ascitic type.
(2) The plastic type, which causes intestinal obstruction, and may affect the gynaecological organs (23-3D).
(3) The glandular type, which involves the mesenteric nodes.
(4) Strictures anywhere in the bowel, but usually in the caecum and distal small bowel, where they are caused by contracture of a tuberculous ileocaecal mass to form a fibrous constriction. Strictures may be multiple, and are then highly likely to be due to TB.

Less commonly, you may also find:
(5) Tuberculous ulcers which can occur anywhere in the bowel, but are most often seen in the ileum, caecum, rectum, or sigmoid colon. In the small bowel, a tuberculous ulcer can cause diarrhoea. A tuberculous ulcer may perforate the bowel, or bleed; because this occurs distally, bleeding is usually from the rectum.
(6) Tuberculous sinuses or fistulae.
(7) An isolated tuberculoma, usually in the right iliac fossa, or other masses.
(8) Intussusception affecting the ileocaecal segment.
(9) Tuberculous appendicitis, presenting just as acute appendicitis.
(10) TB of the liver, spleen or pancreas.
(11) Tuberculous gastric or duodenal ulceration.

All this pathology can present in so many ways, and with so few distinguishing signs, that diagnosis is difficult. A patient loses his appetite, loses weight, and feels ill, just as with TB elsewhere. There is vague abdominal pain and tenderness, and maybe vomiting. Depending on the type of TB, there may be symptoms of abdominal swelling (the ascitic type), obstruction (the plastic type or tuberculous strictures), abdominal masses (the glandular type), bleeding or perforation (tuberculous ulcers). There may also be symptoms of chronic PID (gynaecological TB, 23.1). Not infrequently, abdominal TB presents after a laparotomy for another condition, *e.g.* a Caesarean section.

Unless there is obstruction or bleeding, you can treat the TB medically. But operate if the bowel obstructs completely (the plastic or stricturing type) or, rarely, if bleeding from tuberculous ulcers persists.

Start standard TB therapy (5.7) as soon as oral intake resumes.

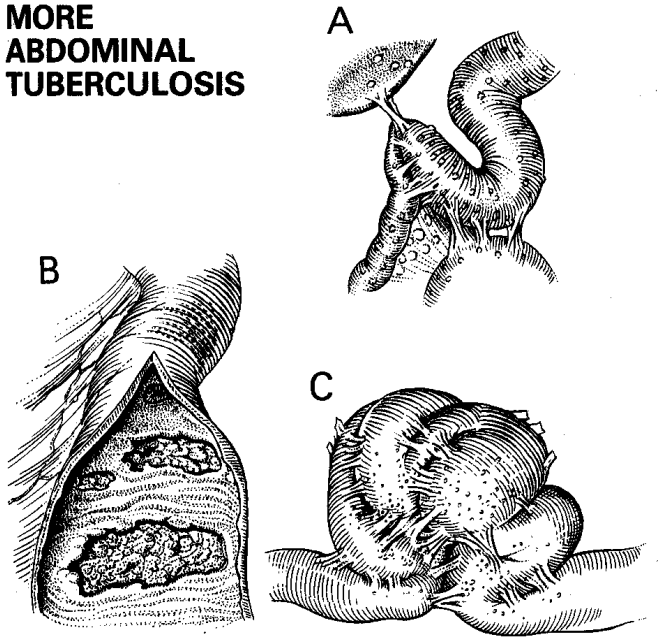


Fig. 16-2 OBSTRUCTING AND ULCERATIVE ABDOMINAL TB. A, adhesion causing obstruction. B, tuberculous ulcers. C, coils of bowel matted together.
Adapted from a drawing by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

16.2 The ascitic type

Tuberculosis may be responsible for 80% of all your cases of ascites. Presentation is with a swollen abdomen containing many litres of straw-coloured fluid. A child with advanced abdominal TB typically has 'a ballooned abdomen and matchstick legs', but in many children the diagnosis is far from obvious. The fluid accumulates as a result of large numbers of exudative miliary tubercles on the peritoneum. The only certain way to make the diagnosis is to perform a mini-laparotomy, or laparoscopy, which will also enable you to diagnose cirrhosis, periportal fibrosis (due to *Schistosoma mansoni*), carcinomatosis of the peritoneum or hepatoma. You can usually diagnose miliary TB with your naked eye; but you can be wrong, so take a biopsy of the parietal peritoneum and/or the liver. (The small seedlings may be cryptococcal in HIV+ve patients.) Occasionally the ascites may be encysted and be confused for a giant ovarian cyst.

In the Concato syndrome, there is a pleural and pericardial effusion as well; this may be due to polyserositis, but in such cases, you should start anti-TB therapy (and prednisolone 120mg od, tapering off over 4wks) immediately and not intervene surgically: the patients are just too ill for that! *Only stop TB treatment if you can really prove its absence!*

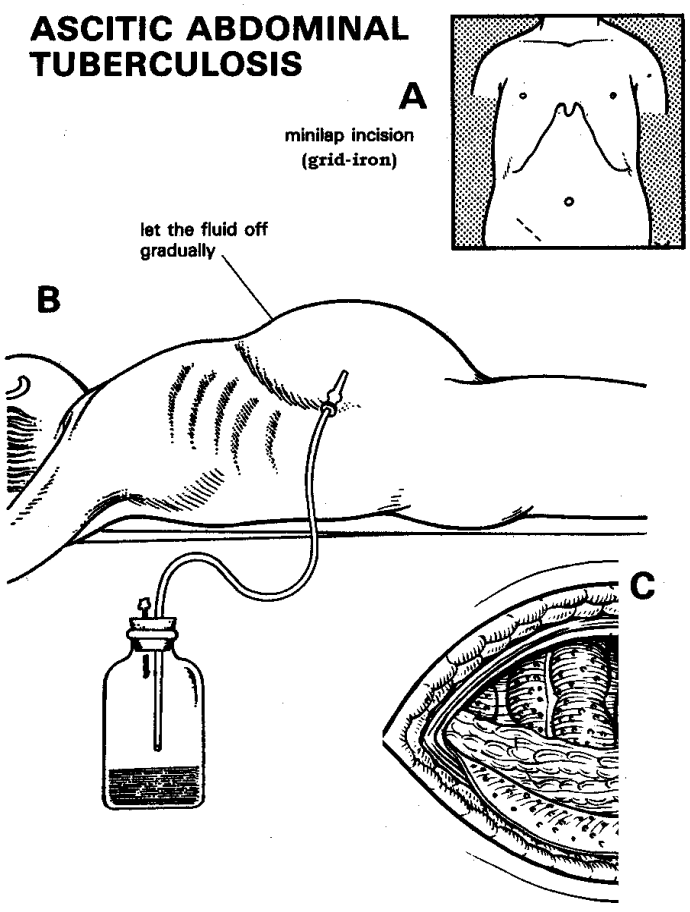


Fig. 16-3 ASCITIC ABDOMINAL TB.
A, mini-laporotomy (gridiron) McBurney incision. B, draw off the fluid slowly before you start. C, miliary tubercles of the parietal peritoneum and bowel.

(a) Special tests. Check the Hb, ESR and HIV. Get a chest radiograph and examine sputum for AAFB. Do an abdominal ultrasound scan to look for lymphadenopathy, and check the condition of the liver and kidneys.

Examine the ascitic fluid: if the white cell count is over 25/µl, with at least 70% lymphocytes, tuberculous peritonitis is fairly likely. If the fluid has fewer lymphocytes than this, the ascites is more likely to be caused by cirrhosis, periportal fibrosis or bacterial sepsis. Your lab will be unlikely to find any AAFB in it, because they are very sparse. Use GeneXpert polymerase chain reaction (PCR) testing if possible (5.7).

Measure the protein in the peritoneal fluid. In tuberculous peritonitis it is usually 4-10g/L, but it may be up to 20g/L or higher. Most patients with ≥20g/L have carcinomatosis. If it contains >4g/L of protein, it is likely to be an exudate. If it contains <4g/L, it is likely to be a transudate as found in cirrhosis or periportal fibrosis.

A more accurate test is the albumin gradient (*i.e.* the difference between serum and ascitic albumin): this is >1·1g/L in cases of portal hypertension, and significantly less than 1g/L in tuberculosis.

If you can do it, measuring adenosine deaminase is highly specific: levels >33 units/L indicate TB in >95%. Note that in the presence of cirrhosis, the value may be lower. You may be able to get special dipsticks for ascites which, though expensive, are very useful.

Blood in the fluid usually suggests malignancy.

(b) The differential diagnosis of ascitic TB
Suggesting ascitic TB: miliary nodules on the peritoneum, each about 1-2mm in size, slightly raised and whitish. The nodules of carcinomatosis, which is the main differential diagnosis, are larger, usually >3mm, more vascular, and more irregular. You will soon learn to distinguish them. Illness is not as severe as would be with a malignant effusion of the same size.

Suggesting ascites secondary to liver disease: the liver may be enlarged, hard, and irregular, or small and hard to feel; the spleen is usually large; there is usually <4g/L of protein in the peritoneal fluid.

Suggesting the nephrotic syndrome: the ascites is less marked than the generalized oedema. If there is ascites, there is usually also marked oedema of the abdominal wall. There is usually <4g/L of protein in the peritoneal fluid.

Suggesting nutritional oedema (hypoproteinaemia): other signs of protein deficiency, but these may also be present in TB. There is usually <4g/L of protein in the peritoneal fluid.

Suggesting heart failure leading to cirrhosis and ascites: a raised jugular venous pressure, and other signs of heart failure; <4g/L of protein in the peritoneal fluid.

Suggesting carcinomatosis affecting the peritoneum: hard deposits in the pouch of Douglas, umbilicus or rectovesical pouch; usually >20g/L of protein in the peritoneal fluid.

Suggesting pancreatitis: epigastric and back pain, persisting after an acute episode, often related to alcohol excess or gallstones; the ascitic fluid usually has an amylase >2,000 IU/L.

(c) Intervention to diagnose the cause of ascites
MINI-LAPAROTOMY OR LAPAROSCOPY (GRADE 2.3)

INDICATIONS. Ascites of uncertain cause. A patient can have more than one diagnosis; for example, cirrhosis *and* tuberculous peritonitis. Ascites predominating over other signs usually requires a mini-laparotomy. It is seldom indicated when the ascites is not predominant, as in the generalized oedema of heart failure, or renal disease. Check the blood urea before you proceed.

CAUTION!
(1) *A mini-laparatomy is NOT suitable for exploring the abdomen*.
(2) You can diagnose tuberculous abdominal glands this way, but look for lymph nodes easier to access elsewhere, for example in the axilla. *You should not perform a mini-laparotomy to take a liver biopsy:* it might bleed catastrophically and you will then have inadequate exposure and/or anaesthesia to control it.
(3) Laparoscopy (19.5) is useful if you have the means but the same comment with regard to liver biopsies applies.

DRAINING THE ASCITES. If there is more than mild ascites, draw off most of the ascitic fluid slowly before you begin. If it all escapes suddenly, as you open the abdomen, the circulation may collapse. So draw off 1L every 2h, starting 48h pre-operatively, to a maximum of 6L. If there is still significant ascites after you have withdrawn 6L, wait until the next day before you draw off more. Use a wide-bore intravenous cannula, a drip set, and a sterile bottle. *Re-examine the abdomen once the ascites is drained away*; you may be able to feel a liver, spleen or other abdominal masses previously obscured.

CAUTION! To avoid possible injury to a large spleen, which may be difficult to feel because of the ascites, drain the fluid from the right lower abdomen.

ANAESTHESIA. Use LA in an adult or ketamine in a child. Avoid GA, because there may be cirrhosis.

INCISION. Make a 5cm right iliac fossa incision as for appendicectomy (14.1). This will allow you to see and examine a tuberculoma, and will be less likely than a midline incision to leak ascitic fluid post-operatively.

Look for miliary tubercles and secondary deposits on the peritoneum. Tubercles are remarkably uniform in size, and fairly uniform in appearance (like salt grains). Biopsy the peritoneum by removing an elliptical piece of the parietal peritoneum 2x0·5cm, from the edge of the abdominal incision. Close in the usual way, but *don't insert a drain*, as it will leak continuously.

16.3 The plastic peritonitic type

This is the result of a tuberculous granuloma, which causes the omentum, and the other structures in the abdomen, particularly loops of the distal small bowel, caecum, and ascending colon, to stick together with many adhesions. The affected coils of bowel are thick and rubbery, with characteristic transverse lesions on the small bowel.

Loops of small bowel may obstruct, and be difficult to separate. Carcinoma, *amoeboma*, and Crohn's disease can all cause a plastic peritonitis, but TB is more common than all these others combined. Amoebiasis makes loops of small bowel stick to the descending colon, without causing a true plastic peritonitis.

The obstruction in the bowel is commonly incomplete, so that symptoms are subacute or chronic, and may have lasted months or years. The adhesions which stick the loops of the bowel together are extensive and difficult to separate, so manage them non-operatively if you can. Start anti-TB treatment, and oral fluids only if tolerated, before proceeding to normal diet. A tuberculous granuloma of the small bowel usually resolves without a stricture; but in the ileocaecal area fibrosis and stenosis often follow.

Occasionally, you may have to operate for persisting complete obstruction. Even then, if you know TB is the cause, you will be wise to try non-operative treatment for a few days first provided there is no strangulation. When you do operate, you may find that there is no stricture in the wall of the bowel, and that you can relieve the obstruction by dividing adhesions only, but *don't divide adhesions which are not causing obstruction*. Try to avoid opening the bowel, because there is always a danger that a fistula may follow. If you have to open bowel, you have a choice between:
(1) A 'stricturoplasty', if there is a narrow stricture in the small bowel (16-4A).
(2) A small-bowel resection.
(3) An ileocaecal resection. *Avoid bypass procedures such as ileo-transverse colostomy or entero-enterostomy*: they result in blind loops, malabsorption and further obstruction; use these only when extensive matting of bowel prevents you from reaching the site of obstruction, but knowing that another laparatomy will be necessary later!

Avoid these common mistakes:
(1) *Don't try to make a diagnosis without understanding the nature of the disease*. Weigh up the signs and symptoms carefully.
(2) *Don't be too eager to start a therapeutic trial without confirming the diagnosis*: there may be some other disease. A mini-laparotomy under LA (16-3) is almost always possible.
(3) If you cannot make a diagnosis, *don't wait too long before exploring the abdomen*.
(4) If there is chronic obstruction which does not respond to non-operative treatment, surgery is mandatory.
(5) If the patient is desperately ill, *don't make meddlesome and dangerous attempts to resect grossly scarred bowel, nor to free difficult adhesions.*

(a) Symptoms

Weight loss (all cases), may be excessive.

Weakness, malaise, fatigue, and anorexia (75%) with also nausea and vomiting, fever and night sweats (60%).

Abdominal pain (90%) is usually constant, central, and not severe. If it is in the right lower quadrant, it suggests ileocaecal TB. With ascites pain is often mild, and may be absent.

Alternating constipation and diarrhoea, cramps, and gurglings (30%). Typically, a description of a 'ball of wind' moving in the abdomen.

Rectal bleeding (5%) may be severe. Vaginal discharge and bleeding may be marked.

Steatorrhoea with pale, bulky, and offensive stools (5%).

N.B. A chronic cough, blood-stained sputum, and TB elsewhere may not be present.

(b) Signs

Abdominal tenderness (60%) is ill-defined, and is usually maximal in the middle of the abdomen. There is often a peculiar 'doughy' feel (20%) of the abdominal wall.

An abdominal mass (40%) may be present. There may be some well-defined tender rubbery masses, either in the ileocaecal region, at the base of the mesentery, or in the adnexa or pelvis in a female. A mass is unusual in the ascitic type.

Signs of obstruction to the lower small bowel may be acute or subacute.

Anaemia is usually moderate.

Dependent oedema as the result of hypoproteinaemia is common.

Lymphadenopathy: look carefully for enlarged axillary or cervical lymph nodes; if you find one, biopsy it. An inguinal node is less likely to be diagnostic, unless it is very large (17.4).

Perianal sinuses or fistulae may be present: *don't forget to look at the anorectum!*

(c) Special tests. Check the Hb, ESR and HIV, and examine the stools. Get chest and erect and supine abdominal radiographs, and examine the sputum for AAFB. *Avoid a barium follow-through in the acute stage: it may make an incomplete obstruction complete. However, you can use water-soluble contrast, and this may also relieve an incomplete obstruction through its osmotic effects.*

Get an abdominal ultrasound to look for lymphadenopathy, thickened bowel, peritoneal nodules, areas of calcification, intestinal stones, a mass or 'cold' abscesses. You may be able to aspirate for AAFB.

Perform a sigmoidoscopy (26-3) if there is rectal bleeding; if this is unhelpful, try to get a barium enema radiograph (38-3). All parts of the colon may be involved with segmental hypertrophic, annular or ulcerative lesions 5-7cm long.

The appearance may look like a carcinoma; TB can indeed be present with a carcinoma, so operation is usually necessary unless you can get a biopsy endoscopically.

(d) Differential diagnosis of plastic abdominal TB

Suggesting *ascaris* infection: a child with vague abdominal pain and subacute obstruction but no weight loss or fever. Tenderness is not constant, and palpable masses of worms are unusual (12.5).

Suggesting an appendix mass: a short history, and an acute onset.

Suggesting amoebiasis: a history of passing blood and mucus rectally, and *trophozoites* in the stools.

Suggesting carcinoma of the colon: an urban lifestyle. It does occur in villagers but is unusual.

Suggesting cirrhosis or a liver tumour: an irregular firm or hard liver, prominent ascites, and a large spleen, a previous attack of hepatitis, or chronic alcohol abuse. A bruit is often present (15.11).

Suggesting Crohn's disease: loss of weight and diarrhoea are the main symptoms. The differential diagnosis may be impossible until tissues are examined histologically. The disease is very rare outside the West.

Suggesting *oesophagostomiasis*: multiple nodules in the colonic wall, which may become confluent and thickened, associated with *O. bifircum* eggs in the stool, especially found in Northern Ghana and Togo.

(e) Non-operative treatment for abdominal TB

INDICATIONS

(1) You are reasonably certain of the diagnosis, and feel a therapeutic trial is reasonable.
(2) There is incomplete obstruction.
(3) There are no signs of strangulation.
(4) There is tuberculous ulceration without perforation or severe bleeding.

Start standard anti-TB therapy (5.7). In an adult, the abdominal symptoms and masses are unlikely to respond for about 2 months, although a child may respond sooner. However, fever, anorexia and malaise will subside sooner. If there is no response to TB treatment, consider the possibility of another diagnosis.

If there is partial obstruction, pass a nasogastric tube, and resuscitate with IV fluids (12.4). If the obstruction passes off, you may not need to operate.

(f) Operative treatment for abdominal TB

DIAGNOSTIC LAPAROTOMY FOR PLASTIC ABDOMINAL TB (GRADE 3.3)

The mini-laparotomy for ascitic TB (16-3) is not suitable for the plastic type of TB; a standard laparotomy through an ordinary incision is, however, a more extensive procedure, which may involve you in further surgery. Do this, however, if there is a persistent vague abdominal pain, perhaps some intestinal symptoms, weight loss, and a raised ESR, and especially if there is a mass.

Open the peritoneal cavity through a midline incision, mostly below the umbilicus, and look for the signs of TB. Biopsy a lymph node. If the site of the biopsy bleeds, control it with packs or with a 3/0 figure-of-8 suture which runs under the bleeding point on both sides.

If you cannot find peritoneal tubercles or rubbery lymph nodes easily, take a biopsy from the parietal peritoneum.

If you find a firm mass at the ileocaecal junction, perhaps with adhesions to adjacent structures and a normal peritoneum, the diagnosis is more difficult. Cut across an enlarged node. If you see caseous areas, you have confirmed the diagnosis. Even so, take a specimen for histology. *Avoid taking a biopsy from the wall of the bowel:* this may lead to a fistula. If the nature of the ileocaecal mass is uncertain, but is probably tuberculous, leave it if it is not causing obstruction. In this case, perform an ileocaecal resection (12.7) or, if this is too difficult, an ileo-transversostomy side-to-side. If it might be neoplastic, and mobile, try to perform a right hemicolectomy (or ileocaecal resection if there are metastases); if immobile, biopsy an enlarged node nearby and await the histological report.

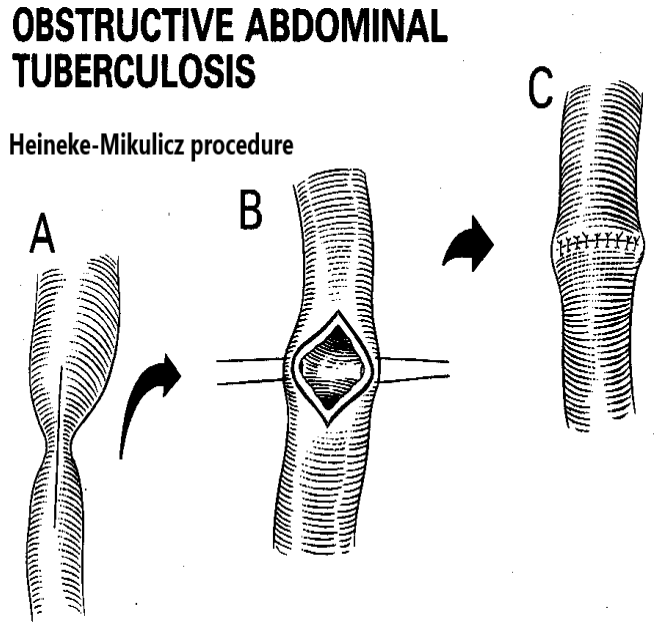


Fig. 16-4 STRICTUROPLASTY FOR OBSTRUCTIVE ABDOMINAL TB. A-C, Heineke-Mikulicz procedure for a stricture of the small bowel. A, incise the bowel longitudinally. B, insert stay sutures beside the middle of the incision, pull them out, and suture the bowel transversely. C, completed procedure.
Kindly contributed by S. Nandy.

If you find a large 'cold' abscess, biopsy and drain it.

If you find an inflamed appendix, excise it taking care that the base of the appendix is closed securely.

If you find a thick mobile fibrotic segment of small bowel and the ileocaecal region is normal, resect it and anastomose the ends (11-7). If it is very short, perform a stricturoplasty (16-4A).

If a tuberculous ulcer has perforated the terminal ileum, oversew and patch it, as you would with a typhoid perforation (14.3). Treat it by resection, if the perforation is large and friable.

If a tuberculous ulcer of the colon has fistulated into the bladder or vagina, make a defunctioning colostomy (11.6); *don't attempt resection until treatment has made the patient better.*

If loops of the bowel are severely stuck down by plastic peritonitis, *don't do too much dissection:* the risks of creating fistulae are too high. Instead, if obstruction is definite, perform a simple side-to-side ileo-transverse colostomy or entero-enterostomy (11-10) without resecting any bowel. This will bypass the diseased segment and avoid much stressful surgery but creates a 'blind loop'. However, a repeat laparotomy later will probably be needed after anti-TB treatment is completed in order to correct malabsorption problems of the blind loop.

16.4 The glandular type

This presents as irregular lumps in a child's or young adult's abdomen, sometimes with ascites, and with little tendency to obstruct. The mesenteric nodes are large, and not very mobile. They may be so large that you can feel them through the abdominal wall. They are matted together, and firm to hard, with characteristic pale yellow areas of caseation on their cut surfaces. There is often hepatosplenomegaly, moderate anaemia, dry depigmented skin, hypoproteinaemic oedema and low-grade fever.

(a) In gastroduodenal TB, lymph node involvement is common; for stricturing in the duodenum, perform a gastrojejunostomy (13-16). Occasionally enlarged lymph nodes in the *porta hepatis* cause obstructive jaundice (15.9) or portal hypertension.

(b) Sinuses or fistulae often develop from deep-seated infected nodes or tuberculous abscesses, especially in HIV+ve patients. You find them in the iliac fossae, in the suprapubic area, or at the umbilicus. Unfortunately biopsy of the track only demonstrates TB in 20%, so look for evidence of TB elsewhere.

Often you can be fairly certain of the diagnosis. If many lymph nodes are involved, biopsy one from the neck, axilla, or groin (17.4). Non-specific adenitis is common in the groin, so only biopsy an unusually large one. You may see enlarged nodes in a chest radiograph. If you cannot establish the diagnosis in any other way, you may need to perform a laparotomy and take a node for biopsy. Lymphoma is the important differential diagnosis (17.6).

With reasonable clinical suspicion, a trial of anti-TB therapy is justified.

16.5 Hepatic, splenic and pancreatic tuberculosis

Tuberculosis of the solid abdominal organs is uncommon (1%) and often, but not always, found only when there is TB elsewhere. Where it affected the adrenal gland in Addison's original case, it resulted only in endocrine disorders. In the liver, there may be multiple granulomas, a single mass with cavitation, or abscess formation; in the spleen, an enlarging semi-solid mass with cavitation. In both forms there is enlargement of the organ but otherwise symptoms and signs are non-specific. Diagnosis is easiest with ultrasound and needle aspiration. Other causes of hepatic granulomas may be leprosy, brucellosis, syphilis, lymphoma, and drug damage.

Surgical intervention is not necessary. You should avoid ethambutol in treatment as this may further damage the liver.

TB affecting the pancreas resembles chronic pancreatitis and can only be differentiated by needle aspiration cytology at operation or biopsy of an adjacent lymph node. *Don't try to biopsy the pancreas itself* for fear of causing a pancreatic leak. HIV disease can cause pancreatitis on its own, but if an ultrasound shows a mass, abscess or cyst, especially in the head of the pancreas, you are probably justified in starting anti-TB therapy.

16.6 Small and large bowel tuberculosis

Any part of the bowel may be involved with TB, but especially the ileocaecal junction. Isolated segments, though, usually 5-7cm long, resulting in hypertrophy of the wall, ulceration or stricturing are not that uncommon.

Perforation, fistula formation and intussusception may result. The appearances are virtually indistinguishable from Crohn's disease, which is very rare outside the Northern hemisphere.

Ileocaecal TB may look like an adnexal mass, an amoeboma, a mass of *ascaris* worms, an intussusception, a caecal carcinoma, a lymphoma, an ileal phytobezoar, a bilharzioma, actinomycosis, or angiostrongyliasis, but unlike in malignancy there is no history of weight loss and anorexia.

(a) Special tests
Barium enema (38.1) is especially useful for ileocaecal or colorectal disease; initially there is hyperirritability of the bowel so there is flocculation of contrast throughout the small bowel with absence of contrast in the diseased segment. The loops lose their mobility and then strictures form with proximal dilation (which may be massive) and filling defects or ring shapes due to enteroliths. The caecum becomes contracted and conical in shape with filling defects, and the ascending colon stenosed and shortened, pulling up the ileocaecal junction. This results in a widened ileocaecal angle (the 'goose-neck' deformity). Colonoscopy can confirm the diagnosis with biopsies.

In intussusception there is a crescent-ring filling defect, or 'watch-spring spirals' of barium seen as contrast is held up or seeps past the intussuscepting bowel. Ultrasound scan appearance is typical (12.7).

Colonic lesions look like carcinomas with 'apple-core' deformities.

(b) Management
Ileocaecal resection, stricturoplasty, entero-enterostomy or ileo-transverse colostomy are the options if surgical intervention is necessary (16.3).

Involvement of the appendix (2%) may be primary or as a result of ileocaecal infection; unless you send the appendix for histology, you may miss the diagnosis!

Colorectal involvement usually results in bleeding, which may be severe. The sigmoid and rectum are the commonest sites. Check the stools for *trophozoites* to exclude amoebiasis. You may not be able to distinguish colonic lesions from carcinoma, which may exist simultaneously, so a resection may still be best, if it is possible.

16.7 Urological tuberculosis

If there is persistent cystitis, which fails to respond to antibiotics, with pus cells and red cells in the urine, but no bacteria are cultured from it by routine methods, there may be TB of the urinary tract. There may also be HIV cystitis, or this together with TB! You will see this usually in a young adult without signs of TB elsewhere. Treatment of the TB is usually effective if the disease is not too far advanced and the treatment is taken conscientiously. Improvement may be dramatic, especially if you can start early, and even strictures of the ureter have been known to heal. So watch for urological TB, and be prepared to treat on suspicion alone. The surgery needed for late presentation is complex. Unfortunately, the disease starts so insidiously that there may be no complaints till late.

Bacilli reach one of the kidneys (usually only one, but sometimes both) in the blood, after which caseation slowly destroys it. Only when the disease has eroded into its calyces do bacilli spread in the urine down to the ureter and bladder, and cause frequency and pyuria. Eventually, most of the kidney is destroyed, after which the disease may spread beyond, to form a palpable mass in the loin, perhaps with a discharging sinus.

TB inflames the mucosa of the bladder and forms tubercles which may ulcerate, coalesce, and form shallow ulcers, especially round the orifices of the ureters and on the trigone. Ultimately, much of the wall of the bladder is destroyed, so that it ends up scarred, red, and contracted. A ureter which drains a tuberculous kidney is flooded with bacilli, and becomes thick, fibrosed and strictured, usually in its lower third. Above this, the urinary tract dilates to form a hydro- or pyo-nephrosis.

Presentation occurs with:
(1) Symptoms of chronic cystitis: frequency and dysuria. This later progresses to the burning nocturia and strangury (slow and painful discharge of urine, drop by drop) of a small shrunken bladder, which may become secondarily infected. These symptoms make the bladder appear to be the cause of the disease, rather than the kidney.
(2) Painless intermittent microscopic haematuria or rarely obvious bleeding. (In contrast, a renal carcinoma usually presents with macroscopic haematuria.)
(3) Dull discomfort in the loin, which gets steadily worse, especially when TB is complicated by a pyogenic infection (20% of cases). The kidneys are not enlarged or tender, until late.
(4) Malaise and the usual general symptoms of TB.

(a) Signs. The kidneys may be palpably enlarged. Several parts of the urinary tract and genitalia may be involved at the same time: a non-tender irregular and boggy prostate, *not* usually enlarged; thickened oedematous spermatic cords with vesicles thick and boggy; thick, woody, and craggy epididymes, which may caseate, and form sinuses, or may involve the testis, and cause a secondary hydrocoele.

(b) Special tests. Urine with pus cells and red cells, but no bacteria on standard culture (unless there is secondary infection), is strongly suggestive. However, recurrent *E. coli* urinary infection occurs in 20%.

A 24hr urine, or a freshly voided clean-catch early morning urine specimen (after a period of dehydration), may show AAFB in a stained film. Repeat the examination 3-5 times. This needs little equipment, but it does require considerable skill, and much patience. You will probably have to rely on finding pus cells and red cells only. If possible, culture the urine for AAFB. Fine needle aspiration from the epididymis may be more helpful.

Ultrasound may show an irregular shrunken bladder or deformed kidney, but is not that useful.

Perform a cystoscopy before an intravenous urogram (IVU, 38.1a), which is much more expensive and will only show changes if disease is advanced. This may show 'moth-eaten calyces', and dilatation of the renal pelvis, and ureter. There may be extravasation of contrast. Cavitation in the kidney is highly suggestive of TB. If it is very advanced, the kidney will not be functioning. There may be multiple long ureteric strictures; a corkscrew appearance and beaded ureter strongly suggest TB. The bladder is small and contracted.

(c) Differential diagnosis. In endemic areas *schistosomiasis* is much the most common cause of pain on micturition with pus cells and red cells in the urine, compared with urinary TB, which is uncommon or rare.

Suggesting *schistosomiasis*: small 3-5mm nodules in the epididymis, nearly always in the tail, and calcification of the bladder wall, as shown by a line in the shape of the bladder, which collapses after micturition.

(c) Cystoscopy (27.3) will confirm the diagnosis, show the degree of involvement of the bladder, and exclude *schistosomiasis*. You may see a 'golf-hole' ureteric orifice.

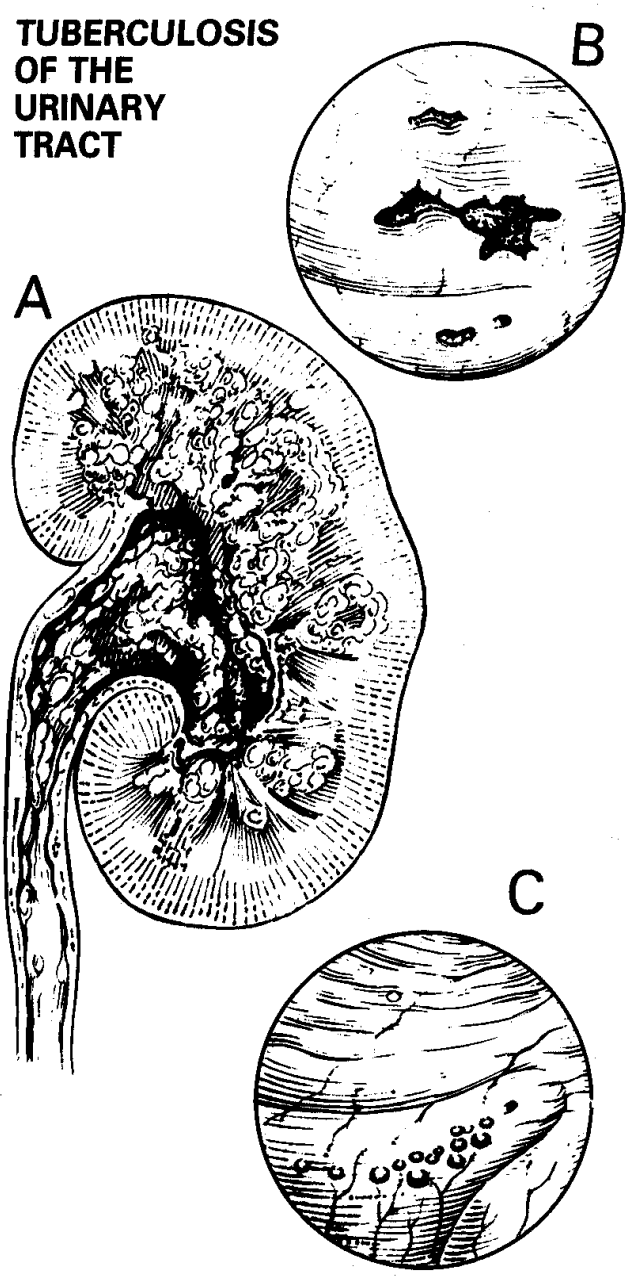


Fig. 16-5 TUBERCULOSIS OF THE URINARY TRACT.
A, TB of the kidney involving the pelvis and the ureter. B, tuberculous ulcers of the bladder. C, tubercles near the orifice of the ureter.
Adapted from a drawing by Frank Netter, with the kind permission of CIBA-EIY Ltd, Basle, Switzerland.

(d) Treatment of renal TB
Treat on an outpatient basis, with standard TB therapy (5.7). If renal function is impaired, avoid streptomycin, or ethambutol, or use them intermittently. Rifampicin, isoniazid and pyrazinamide are safe. Review every 2 months for regular assessment, including the examination of the urine.

If there is a relapse, and you think that drugs have not been taken faithfully, consider changing to a regime using second-line drugs. Check the HIV status if this has not already been done, or was previously -ve.

If you are in an endemic area and routine examination shows no ova of *Schistosoma haematobium*, examine the deposit from a specimen passed at midday (the time when most ova are passed) on 3 consecutive days.

Check the Hb (low), ESR (raised) and HIV status.

(1) **Ultrasound** may show caseation in the pelvis of the kidneys, hydronephrosis, hydro-ureter, and a shrunken bladder, but will only show advanced pathology.

(2) **Radiographs.** On plain abdominal films, look for the outline of an enlarged kidney, diffuse calcification, and obliteration of the psoas shadow. Get a chest film.

(3) **Indications for surgery.** Operations for renal TB are complicated and need an expert.

If the IVU shows no function, or has a moth-eaten appearance, with flecks of calcium, a nephrectomy is needed. Hypertension is an additional reason. If the patient is toxic and febrile, suggesting a pyonephrosis, or a perinephric abscess, these need urgent drainage (6.15, 27.14).

If there is a ureteric stricture, ideally a ureterogram is needed. If you have drained a pyo- or hydro-nephrosis externally, you can inject contrast through the nephrostomy tube. For upper ureteric strictures, a pyeloplasty is needed; for lower ureteric strictures, a re-implantation of the ureter or bladder flap.

N.B. In endemic areas, schistosomiasis is a common cause of a lower ureteric stricture.

If there is still extreme frequency and dysuria after 6 months of treatment, suspect that there is a small contracted bladder. Confirm this by ultrasound or cystoscopy and/or a cystogram. Surgery may be possible to augment the size of the bladder.

N.B. Don't forget gynaecological TB (23.1).

17 Lymph nodes & salivary glands

17.1 Lymphadenopathy in HIV disease

Lymph node enlargement is one of the commonest presenting signs of HIV disease; typically the nodes are small, rubbery and symmetrical in the early phases. Epitrochlear lymphadenopathy is virtually diagnostic of HIV disease. If you biopsy these nodes, the histological changes show follicular hyperplasia only. You will probably see so many patients with lymphadenopathy that you cannot reasonably biopsy them all! Also it will not be very helpful. The femoral nodes are often enlarged, especially if no shoes are worn and the feet are bare, and will not give a useful result unless obviously grossly abnormal. See if the nodes are matted together, firm or hard, asymmetrical, and >2cm diameter; then the diagnostic yield will be significantly greater.
The main causes will be:
(1) tuberculosis;
(2) Kaposi sarcoma;
(3) lymphoma; and
(4) in Latin America, *histoplasmosis*;
(5) in South America, *leishmaniasis* or *paracoccidiomycosis (blastomycosis)*;
(6) in East Asia, *penicillium marneffei* infection.

Cystic degeneration occurs particularly in the parotid and submandibular regions, and so it becomes very difficult to differentiate lymph node pathology from salivary gland disease; the two are often anyway mixed. When cysts become large, unsightly and painful, you can aspirate them: usually you find a straw-coloured fluid. Excising cysts may not be easy, especially in the neck: they may be deeper than you think, and more cysts will probably appear elsewhere later! If they occur in the parotid, you may damage the facial nerve by operating.

Excision biopsy of a lymph node in the neck may not be easy, and it will be worthwhile to develop a cytology service if possible. If general cytology is not feasible, you can at least look for AAFB.

Macroscopic examination of a lymph node is useful if no histology is available. Angiofollicular dysplasia (Castleman's disease) occurs more commonly with HIV and principally affects neck and intra-abdominal nodes. Try to excise all such nodes.

Don't forget that not every swelling in the neck is a lymph node! It might be a branchial (congenital) cyst, neuroma, lipoma, lymphangioma, salivary gland tumour or even a large sebaceous cyst.

17.2 Fine needle aspiration (GRADE 1.1)

To obtain a cytology specimen, insert a sterile 20 or 22 gauge needle attached to a 10ml syringe into a superficial lymph node (or tumour) and withdraw the plunger sharply and so some cells into the syringe; then expel the cells onto a dry glass slide without having let go of the plunger beforehand. It is important to examine the cells immediately; diagnosis depends much on their architecture, and you have the risk of losing the specimen in transport. Dry the slide but *don't smear it because this might destroy the cells*. Fix it in methanol, or absolute ethanol, for 3mins, and stain it with Field's stain, or better, any Romanowsky blood stains, such as Leishman, Wright, or 10% Giemsa buffered in a pH6.8 solution.

In about 40% of cases, you will be able to see caseation in a tuberculous lymph node with the naked eye, and in 70% of cases by microscopy.

You may be able, by experience and reference to standard atlases, to make more sophisticated diagnoses, but it would be wise to confirm these, if at all possible, by histology.

The cells of Burkitt's lymphoma (17.6) are fairly uniform in size. The cytoplasm of the cells forms a thin eccentric rim around the nucleus, is basophilic, non-granular, and usually contains some small vacuoles. The nucleus is slightly indented, and has 2-5 nucleoli, evenly distributed chromatin, and occasionally mitoses. You will not see the 'starry sky' appearance, because this is a histological, not a cytological, finding. In HIV-related disease, the cells are larger with plasmacytoid features.

17.3 Lymph node biopsy

You will often be able to get extremely important information from examination of lymph nodes. Significant lymphadenopathy is matted, firm or hard, >2cm in diameter and asymmetrical. If you don't get a conclusive clinically realistic answer from aspiration cytology, you should go ahead and excise a node. *You should not use a trucut biopsy needle* (24-3) *for a neck node.*

Most useful biopsies come from the neck or axilla; the groin often has low-grade infection and fibrosis and unless the node is obviously abnormal, it will not be worth removing. A node may feel quite superficial, but then on exploration be under important structures, so *familiarize yourself with the anatomy* of the region you are operating in, especially the nerves. (If necessary, have an anatomy book open in theatre next to you, to give you guidance!) Although you can remove a superficial lymph node in the neck quite readily under LA, it may be helpful to add pethidine or ketamine to ease the patient's discomfort. *Don't try any node excision in children or an axillary node excision under LA.*

Remember that the neck and axilla are very vascular areas with much 'clockwork', so you must be able to see what you are doing! Therefore, make sure you have a good operating light, and preferably diathermy available. If not, be certain to have sufficient haemostats, gauze and a suction that is working available.

If you cannot remove the whole lymph node, cut it clean and take a representative sample: *don't try to be heroic and excise a huge node without seeing what is underneath!* Try to handle the node as gently as possible because you distort the architecture if you are rough, and this makes histological interpretation difficult.

(a) Indications for lymph node biopsy

(1) Tuberculous lymphadenitis (if fine needle aspiration is uncertain and to confirm the diagnosis).
(2) Matted, firm or hard lymphadenopathy >2cm in diameter.
(3) Suspicion of metastatic disease with the primary tumour unknown.

METHOD FOR NECK NODE BIOPSY (GRADE 1.5)

Position the patient with the head tilted up and turned to the opposite side. Make an incision over the node in the direction of Langer's skin lines, and extend it 1cm to either side of the node. (*Don't try to remove a node through a keyhole incision.*) Deepen the incision and make sure of haemostasis. Keep the wound edges apart with a self-retaining retractor. If there is now too little room to operate, your incision is too small: go back and increase its size.

If there is only fascia covering the node, incise this and carefully extract the node with scissors using the 'push and spread' technique (4-9B). If the node lies deep to other structures (muscle, artery, vein or nerve), retract these out of the way. You may need an assistant to do this. Dissect gently down to the node, dividing only what you can see. There is usually a fairly large artery feeding the node behind it, so you should push the node to one side to see this to tie it off, or diathermy it. Carefully free the node, or cut off a part clean if it is too big to remove *in toto*. Ensure there is no bleeding; if there is some oozing, close the wound with interrupted non-absorbable 3/0 sutures around a small Penrose drain (4.9).

METHOD FOR AXILLARY NODE BIOPSY (GRADE 1.5)

Position the patient with the arm flexed under the head, which is turned to the opposite side. Make a transverse incision at least 5cm long and deepen it through the axillary fascia. Keep the wound edges apart with one or two self-retaining retractors. This exposes the fat of the axilla and the tail of the breast; here are the pectoral group of nodes. There are more nodes adjacent and behind the axillary vein which is at the upper border of the axillary fat; these nodes may be lateral, central or apical and are also close to branches of the brachial plexus. So you need to operate carefully here making sure you can see properly; gently separate the node from surrounding structures and control bleeding as you go.

Once you have sampled the node, ensure there is no bleeding and close the wound as above.

METHOD FOR GROIN NODE BIOPSY (GRADE 1.5)

Make sure the patient is bathed and clean. Position him supine; if the hair is likely to be in the way, shave it but *take care not to cut the skin*. Make a 5cm incision in the groin crease or over the node, away from anywhere where the underlying pathology is infiltrating the skin *because the wound may then not heal.* Deepen the wound through the fascia. Keep the wound edges apart with self-retaining retractors. There are many superficial veins; tie these or diathermy them to keep a dry field. There are superficial nodes just below the inguinal ligament and around the long saphenous vein. If the enlarged node is one of these, gently separate it from surrounding tissues and remove it as above. If the long saphenous vein is involved or damaged, clamp it and ligate it. There are deeper nodes in the femoral canal; palpate the femoral artery and so locate the femoral vein just medial to it. The nodes are more medial still. *Don't attempt to remove one of these unless you are prepared for a block dissection* (17.8); satisfy yourself with a biopsy of a part only. Close the wound with a drain if you have dissected deeply.

(b) Examination of the node

A normal node has a pale colour and is uniform; you may be able to distinguish the cortex and medulla with the naked eye. It may show abnormalities nonetheless on histology and so you should not assume it to be normal; however, direct examination of nodes can give valuable instant information.

Under a good light, look for caseation or tuberculomas which are present in 75% of tuberculous nodes. Caseation may occur with lymphoma, *histoplasmosis*, *blastomycosis* and tularaemia, but these are relatively uncommon, and where tuberculosis is endemic, you should start treatment whilst waiting for the histology result.

Pus within the node or showing purulent necrosis suggests TB in over 90%, especially when surrounding cellulitis is absent. You should examine the pus and a smear of the cut lymph node on a glass slide by microscopy and ZN staining for AAFB.

If you see hypervascular nodes, especially with a purplish colour, this is likely to be Kaposi sarcoma.

(c) Difficulties with lymph node biopsies

If you cannot control the bleeding, *don't plunge haemostats blind deep into a cavity.* Pack the cavity and get the suction ready. Then carefully remove the pack under a good light so you can see properly, and apply a haemostat to the bleeding vessel. If the bleeding is coming from a major vein or artery, and you cannot control it, pack the wound again and press on it. Get more assistance, put up an IV infusion of saline or Ringer's lactate and anaesthetize the patient.

Open a major dissection instrument pack, preferably with vascular clamps. For an axillary biopsy, position the

patient with the head tilted up and turned away from you. *When everything is ready*, extend the incision to get good exposure and slowly remove the pack using suction. Put vascular clamps (or haemostats) on either side of the bleeding vessel, and tie them off. *Don't be tempted to use diathermy in this situation.* If all this fails, tightly pack the wound, wait 24h and re-explore the next day.

If the node is too big to remove safely, cut part of the node off clean and leave the remainder.

If the node is actually an abscess, clean the cavity out and pack the wound; if it is rather large, insert a Penrose drain, and close the wound with interrupted sutures. *Don't forget to take some tissue for biopsy, and a swab for culture and AAFB.*

If the node is actually not a node but a tumour (benign or malignant), proceed as above to try to excise it if you can. If it is stuck, just take part of it for biopsy, and close the wound.

If the node is cystic, try to excise the cyst complete with its lining. If it bursts, try to remove as much of the lining as you can to prevent its recurring.

If you find malignant melanoma (34.6) **in the node**, take a biopsy and prepare for a block dissection within a week. Look for the primary, and make sure that is widely excised then, if necessary with skin grafting of the defect.

If the 'node' is actually an incarcerated femoral hernia, proceed to hernia repair (18.7) with bowel resection if necessary (18.8): *don't abandon the procedure!*

17.4 Tuberculous lymphadenitis

Widespread tuberculosis of the lymph nodes is not uncommon in many areas. It usually involves the nodes of the neck, or less often those of the axilla, iliac region, or groin, mainly in children and young adults, although no age is exempt. All 4 triangles of neck may contain matted masses of glands. If these are not treated, abscesses may form and discharge through the skin, to leave sinuses which may become secondarily infected. After many months, these abscesses may heal spontaneously, to cause severe fibrosis and lymphatic obstruction in the leg, arm, breast (34.12), or vulva (23.17).

Establish the diagnosis by fine needle aspiration (17.2) or lymph node biopsy (17.3). *Send samples NOT in formalin.*

If you have found evidence of TB, start anti-TB treatment (5.7).
Don't excise the enlarged nodes. Don't be alarmed if they enlarge temporarily during TB treatment or, rarely, after it, without microbiological relapse. This is due to hypersensitivity to tuberculoprotein. All nodes become smaller in time.
Check the Hb level and HIV status. Antiretroviral therapy usually best waits till after TB treatment is finished (5.8).

17.5 Salivary gland enlargement

A very noticeable facial feature is bilateral parotid gland enlargement; this may be endemic especially in malnourished patients with vitamin deficiency, suffering from multiple parasitic disease, but is also a common feature of untreated HIV disease and almost diagnostic where this is common. Parotid enlargement occurs even when CD4 levels are normal, so there may be no other overt signs of HIV infection. Initially the parotid enlargement may be unilateral, but it almost always becomes bilateral with time. The parotids may feel lumpy or cystic or both; aspiration produces a yellowish fluid, occasionally opaque. It is rarely necessary to repeat aspiration once you have started ARV treatment: superadded infection is not uncommon. The parotids may become quite grossly enlarged, particularly with tuberculosis or lymphoma; fine needle aspiration is then helpful to get a diagnosis, but operative intervention is neither useful nor safe. The enlargement will recur and facial nerve palsy is a real hazard.

(a) Bilateral parotid enlargement may be caused by:
(1) chronic recurrent sialadenitis,
(2) Sjøgren's syndrome,
(3) sarcoidosis,
(4) sialectasis in Chagas disease,
(5) swelling from excessive pipe, glass or trumpet blowing,
(6) leukaemia.
None of these causes warrant surgical intervention.

(b) Acute bilateral parotid enlargement is usually viral (mumps, Coxsackie or CMV) or bacterial from acute sialadenitis due to poor oral hygiene (for example, in patients with head injuries in whom mouth care has been neglected, or in patients with dental caries or ill-fitting dentures).

(c) Chronic unilateral parotid enlargement may be due to:
(1) tuberculosis,
(2) actinomycosis,
(3) tumours (17.7).
The distinction may be difficult and fine needle aspiration is very helpful.

(d) Acute unilateral parotid enlargement may be due to:
(1) an abscess,
(2) acute sialadenitis due to a stone in the parotid duct: this is rarer than a stone in the submandibular duct.

Submandibular gland enlargement arises from the same causes, but because the salivary secretion is more viscous than from the parotid, stones are more likely to form.

SIGNS
Facial nerve palsy implies malignancy of the parotid (or previous surgery). Check if a parotid swelling occupies the whole gland, or only part of it.

Look at the parotid duct opening (opposite the second upper molar tooth) and the submandibular duct opening (at the side of the frenulum of the tongue); look and feel for redness, pus or a stone. If it looks like there is an obstruction, give the patient a sweet to suck and watch the gland becoming tense and painful. Probing the duct gently may produce a gush of saliva or pus!

SPECIAL TESTS
A plain radiograph will show up a stone; you can cannulate the duct and inject contrast to get a sialogram (38.1i) to demonstrate a stricture or impalpable stone.

If you aspirate pus, drain the parotid abscess (6.10).

PAROTID SIALOLITHOTOMY (GRADE 2.2)
Removing a stone from the parotid duct may be simple, but can get complicated. Remember the facial nerve lies close to the duct, so don't pass sutures round it as you can do for the submandibular duct.

Ask the anaesthetist to pass a nasotracheal tube to keep the mouth clear; otherwise the endotracheal tube has to be strapped well out of the way on the opposite side of the mouth. Put in a pharyngeal pack with a long thread hanging out of the mouth, so it is not forgotten! Keep the mouth open with a gag and get an assistant to hold the tongue out of the way by pulling on it with a towel clip.

Find the parotid duct opening and pass a probe along it; put a 2/0 stay suture 5mm above and below the papilla and then cut along the duct starting at the orifice till you reach the stone. Lever it out gently. If it is not palpable, insert the probe into the duct and cut down onto it. Open the duct orifice wide by suturing together the duct wall and mucosa of the mouth, and keep it like that. Then try to manipulate the stone out with your fingers, and it is more likely a stone will pass naturally.

SUBMANDIBULAR SIALOLITHOTOMY
(GRADE 2.1)
Only try to remove a stone in the submandibular duct if it is easily palpable anteriorly in the mouth; otherwise it is better to remove the whole gland with the stone. Remember the stone may fall back down the duct, so pass a ligature round the duct proximal to the stone to prevent this.

Prepare the mouth as for a parotid sialolithotomy. Put a stay suture vertically into the floor of the mouth medial to the duct opening; pass a probe through this and then cut directly onto the stone and lever it out. Leave the duct open and don't disturb the orifice so it does not become stenosed later. If the stone is right at the orifice, push it back a bit (but not too far!) and continue as before.

DIFFICULTIES WITH SIALOLITHOTOMY
If the stone disappears into the gland, perform a submandibular gland excision (17.7) but *don't try to remove the parotid*. Instead, cut back on the parotid duct as far as you can, and introduce an embolectomy catheter down it and try to manipulate the stone out by distending the catheter balloon distal to the stone. If this fails or you don't have an embolectomy catheter, try to crush the stone by passing a haemostat down the opened duct and exerting pressure on the parotid gland outside the mouth.

17.6 Lymphomas

(a) Endemic Burkitt's lymphoma

Burkitt's lymphoma is a non-Hodgkin's lymphoma, but because it behaves differently from the other lymphomas in this group, it has to be considered separately. There are three types:
(1) The type which is endemic in highly malarial areas, mostly in Africa, and which is associated with the Epstein-Barr virus (EBV).
(2) The immunodeficiency-associated type, associated with HIV disease.
(3) The rare sporadic type seen all over the world, which is not associated with EBV. This is more common in Yemen, Somalia and Ethiopia owing to frequent chewing of *qhat*.

Burkitt's lymphoma is a high-grade tumour of B lymphocytes, and is the fastest-growing tumour in man: it can double in size in 24h. It afflicts children from 2-16 (mean age 7), is unknown under 1, and is rare >20yrs. It is the commonest children's cancer in Africa, and in some areas it is as common as all other childhood cancers combined. In endemic areas the genome of the Epstein-Barr virus is present in 100% of tumours; in Egypt this is reduced to c.75%.

It presents as:
(1) Swelling of the mandible or maxilla (1-4 quadrants): the commonest presentation in Africa. The earliest sign is loosening, or displacement, of a child's molar or premolar teeth.
(2) Proptosis, which may be marked, but is usually not painful.
(3) Intra-abdominal tumours, especially of the retroperitoneal lymph nodes or ovaries.
(4) Extradural lesions causing spinal cord compression and paraplegia.
(5) Enlargement of the parotid glands, breasts (usually both), testes, thyroid, and kidneys, all of which are uncommon. Lymphomatous masses can also occasionally occur in skin or bone.
(6) Lymph node enlargement is also uncommon, except in the abdomen.
(7) Firm, painless, non-tender swellings, sometimes of huge size, anywhere in the body (rare).
In spite of all this, the child's general health is usually remarkably good. When the bone is involved, radiographs show osteolytic lesions.

Burkitt's tumour is a malignancy that can be cured by chemotherapy alone. Even involvement of the central nervous system is compatible with long survival. It responds so dramatically that it should have the highest priority for chemotherapy: the prognosis is inversely

proportional to the volume of the tumour, hence the importance of:

(1) **Early urgent treatment.** If you can treat before the central nervous system is involved, there is about a 50% chance of surviving 4 more years, and probably long-term.

(2) **Tumour debulking.** If presentation is late, try, if possible, to remove the large bulk of the tumour surgically first. If you can remove more than 90% of it, you will double the survival time. Unfortunately, you are unlikely to be able to do this, except perhaps by removing a girl's ovary, or her breasts, or a mass in the mesentery. If you try, spilling the tumour is unimportant, because there will be a malignant ascites anyway.

So, if a child is to be treated early, and perhaps to be treated at all, you will probably have to treat him yourself. There is no time for a leisurely workup: try to start treatment in 24-48h.

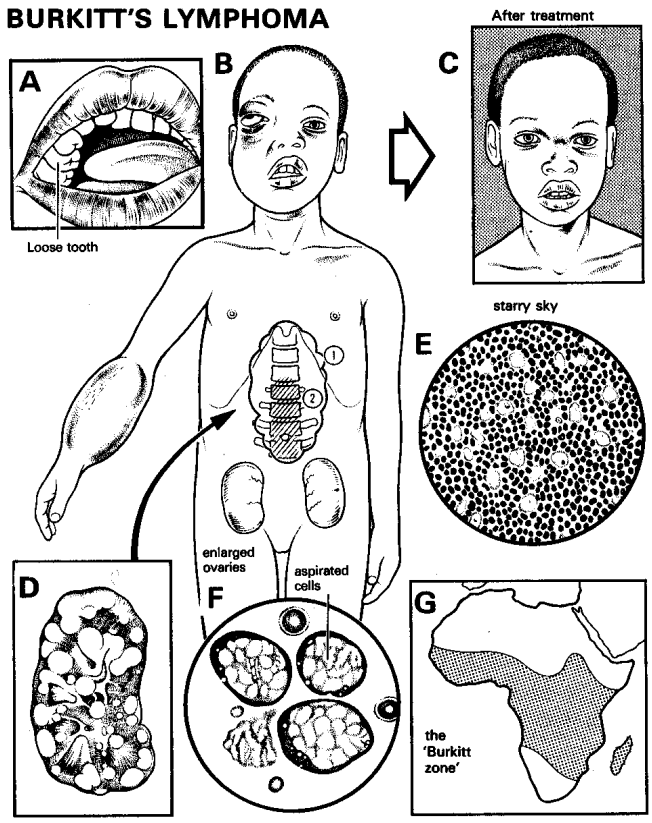


Fig. 17-1 BURKITT'S LYMPHOMA.
A, a child's first complaint may be a loose tooth or displaced teeth, and the first radiological sign, an erosion of the compact bone lining the alveolus, the lamina dura of a tooth. B, child with Burkitt's lymphoma, showing proptosis and swelling of both the upper and lower jaw on the right side. C, prompt chemotherapy is effective in >50% of cases. D, massive involvement of a kidney. E, characteristic 'starry sky' histology of Burkitt's lymphoma. F, if you aspirate a tumour and stain the cells with a Romanowsky stain, you may see cells like this. G, the 'Burkitt zone' in Africa, which is coincident with that of holoendemic or hyperendemic malaria. (1) mass of retroperitoneal lymph nodes. (2) spinal mass causing paraplegia.

There are 2 important complications:
(1) The acute tumour lysis syndrome is a combination of lactic acidosis, hyperkalaemia, and hyperuricaemia, and is the result of the rapid destruction of a large mass of tumour. Resection of most of the tumour before chemotherapy reduces this risk. This syndrome, and the post-surgical complications of far advanced disease, are the common causes of an early death.
(2) The excretion of cyclophosphamide in the urine can cause a haemorrhagic cystitis.
You can reduce both these dangers by maintaining a high urine output and by alkalizing the urine with bicarbonate.

Diagnosis. The fact that you are in an endemic area, the child's age, and the site of a rapidly growing tumour should alert you to the diagnosis. Get radiographs. Carry out a fine needle aspiration for cytology (17.2). Take a standard biopsy, or a smear biopsy. If possible, send a bone marrow smear for histology, and examine the CSF for malignant cells. Check the HIV status.

Differential diagnosis: this varies with the site.
A swelling of the jaws: an infected tooth socket (6.9), perhaps with osteomyelitis, injury, maxillary sinusitis (29.8), a dentigerous cyst or an adamantinoma (31.6), or a nasopharyngeal carcinoma (29.16). Burkitt's lymphoma usually displaces the teeth.
Proptosis: retinoblastoma (28.16) affects the eye itself whereas Burkitt's lymphoma usually displaces the orbit.
An abdominal swelling: tuberculous lymph nodes (17.4), other lymphomas, a nephroblastoma (27.35).

Staging. Prognosis is directly related to stage:
Stage A Single site (often the jaw).
Stage B Two sites excluding abdomen, thorax, CNS or bone marrow.
Stage C Abdomen or thorax tumour, excluding CNS or bone marrow.
Stage D Involving CNS and/or bone marrow.

Prognosis. Both cyclophosphamide alone, and the 3-drug regime described below, give a complete response rate of 95%. Cyclophosphamide alone gives a relapse rate of 50-60% (*but only 10% in stage A disease*); the 3-drug regime reduces this to 30%. These figures are for all stages combined. Stages A and B have a better prognosis. Relapse in HIV disease is almost certain; ARV treatment (5.8) is necessary after the chemotherapy course is completed.

If there is no relapse within 6 months of starting treatment, there is a >90% chance of a complete cure. Relapse after 1yr is unlikely, if there is complete response to the initial treatment.

Chemotherapy for Burkitt's lymphoma
If you cannot obtain very rapid histological or cytological confirmation, and the clinical presentation is typical, start treatment without them: the tumour may grow dramatically in a few days. *Don't delay!* Prepare for chemotherapy (37.4).

If there is a large intra-abdominal mass, or an accessible mass elsewhere, try to resect it urgently. If this is impractical, start chemotherapy alone. *Don't delay chemotherapy if early resection is impossible*.

Triple therapy reduces the relapse rate at all stages, especially in less favourable cases (stage D), or when there is meningeal involvement, or in stage C when there is a very large tumour. Stop treatment at the end of the course: maintenance therapy is of no value. *If you only have cyclophosphamide, it is still very worthwhile as a single-agent therapy.*

CYCLOPHOSPHAMIDE. Use cyclophosphamide 1g/m² IV stat, and repeat this every 2-3wks for at least 2 courses beyond complete clinical remission.

TRIPLE THERAPY. Use cyclophosphamide 1g/m² IV on day 1 plus vincristine 1·4mg/m² on day 1 and oral or IV methotrexate 15 mg/m² on day 1. Repeat these drugs every 2-3wks for one course beyond complete clinical remission.

CAUTION!
(1) Beware of the acute tumour lysis syndrome, especially if there is a large tumour burden because it may be fatal. During the first 24-48h maintain a high urine output, and alkalinize the urine with sodium bicarbonate (the adult dose is 3g in water every 2h until the urinary pH>7, followed by 5-10g od).
(2) If the mass is large, treat with allopurinol 20mg/kg od.

As always, explain to the parents what has happened and what you are going to do. Tell them the prognosis, and that if the tumour does return, you may be able to treat him, but that follow-up is essential. Try to review monthly for the 1st year for signs of CNS relapse, presenting especially as cranial nerve palsies, drowsiness and headache.

Prognosis with Burkitt's lymphoma
If there is no response to the initial treatment, death is inevitable in a few months, whatever you do.

If the airway, the ureters, or the gut are obstructed, the obstruction may respond to chemotherapy. So add supportive treatment (intubation, nephrostomy, nasogastric drainage) and *don't give up!*

If there is no relapse during a year after triple therapy, there is about a 90% chance of surviving indefinitely. About a third of all children have no relapses for at least 2yrs, and should survive indefinitely. The prognosis with cyclophosphamide alone is less favourable.

If there is a CNS relapse, check the HIV status again and repeat the course of chemotherapy and use weekly doses of intrathecal methotrexate (see below) until the CSF is clear of lymphoma cells.

If there is an 'early relapse' within the first 3 months of treatment, it is usually in the same site. This is common with HIV disease and abdominal tumours, and is probably a regrowth of the original tumour. The results of repeating chemotherapy are likely to be poor. Use doxorubicin 60-75mg/m² IV over 30mins every 3wks.

If there is a 'late relapse' more than 3 months after treatment, the tumour is likely to arise in a previously uninvolved site, and will probably respond rapidly to the same agent(s) used initially. A few patients have multiple successfully treated relapses, at intervals which may be as long as 10yrs.

CAUTION! Don't neglect the opportunity of saving patients in relapse. Follow them up carefully, and treat late relapses (>3 months) aggressively, if necessary several times: vigorous treatment in relapse can nearly double the chances of survival.

Difficulties with Burkitt's lymphoma
If there are cranial nerve palsies (often multiple) or meningeal involvement, the central nervous system is involved. This becomes increasingly common as the number of relapses increases. Add 10mg methotrexate diluted in 10ml saline intrathecally with each course of chemotherapy.

If there is paraplegia, treat as generally described. If you can start treatment in the first 3-4 days, the chances of recovery are good. If you can organize laminectomy, *i.e.* decompressing the spinal cord, do so urgently; the less the delay, the better the prognosis.

(b) Hodgkin's lymphoma

This disease of the reticulo-endothelial system occurs all over the world, and is unusual in having a bimodal age distribution curve, with a peak in teenagers, and another >50yrs.

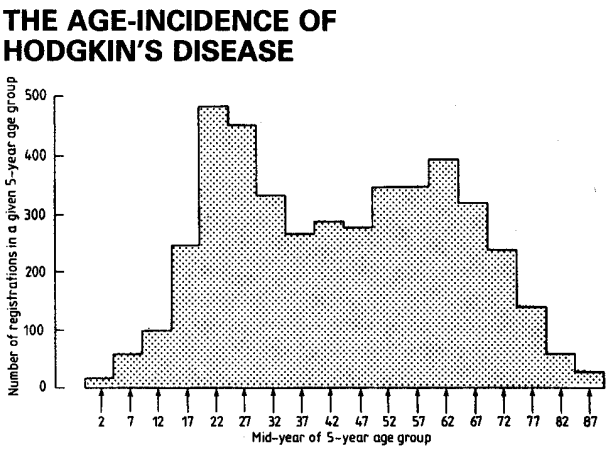

THE AGE-INCIDENCE OF HODGKIN'S DISEASE
Number of registrations in a given 5-year age group
500
400
300
200
100
0
2 7 12 17 22 27 32 37 42 47 52 57 62 67 72 77 82 87
Mid-year of 5-year age group

Fig. 17-2 AGE DISTRIBUTION OF HODGKIN'S LYMPHOMA – bimodal with 2 peaks. *After Mould R. Cancer Statistics. Adam Hilger, 1983 with kind permission.*

Presentation is with:
(1) Enlarged, discrete, painless, rubbery lymph nodes in the neck (60%), mediastinum (15%), abdomen or groin (15%), or axilla (10%).
(2) Weight loss.
(3) Fever which may simulate infection and is classically (but rarely) cyclical (Pel-Ebstein type) for up to 7 days, followed by a remission of up to 7 days.

The various histological types each have their own prognosis:
(1) Predominantly lymphocytic (15% of cases).
(2) Nodular sclerotic (50%).
(3) Mixed cellularity (20%).
(4) Lymphocyte-depleted (15%).
The nodular sclerotic type predominates <30yrs; in older patients the lymphocytic and mixed cellularity types are more common. Characteristic atypical mononuclear (Reed-Sternberg) cells must be visible to make the diagnosis.
Initially, only the lymph nodes are involved. Later, the disease spreads to the spleen, marrow, and liver.
Late complications include: obstruction to the trachea and bronchi, pleural effusion, biliary obstruction causing jaundice, cord compression, lytic and/or sclerotic bone deposits, anaemia, and infections, especially in debilitated patients.

Untreated cases deteriorate, many only slowly, and die in a few months to a few years. Radiotherapy and chemotherapy cure some of them, and cause many long-term remissions. Chemotherapy is so successful in early cases that only Burkitt's lymphoma should have greater priority.

(1) **Diagnosis**
Examine all accessible lymph nodes. Note the nature and size of all enlarged ones. Feel for enlargement of the patient's liver and spleen; assess the weight loss.

(2) **Special tests**
Do a total and differential white count, and count the platelets. Get a chest radiograph: look for widening of the mediastinum and enlargement of the hilar lymph nodes. You must establish the diagnosis by biopsy (cytology alone cannot give you a definitive answer). Measure the blood urea. Check the HIV status.

(3) **Differential diagnosis** includes the common causes of lymph node enlargement (17.1). This may significantly vary from place to place. Significant lymphadenopathy may be from toxoplasmosis, dengue, schistosomiasis, trypanosomiasis (in West Africa), toxocariasis, and filariasis (34.14).
Suggesting Hodgkin's lymphoma: painless, non-tender, rubbery discrete nodes with a uniform light greyish-yellow 'fish-flesh' cross-section.
Suggesting tuberculosis: enlarged nodes which are matted together, and occasionally tender; caseous areas on cross section.

(4) **Staging and response.** Here is the method of staging and the response rate (as measured by the failure of the disease to progress) that you can expect with radiotherapy, or the 'MOPP' cytotoxic regime described below, which are about equally effective.
The 5yr survival rate also depends on the histological grading, and for all stages together on 'MOPP', it is 95% for 'lymphocyte predominant', 75% for 'nodular sclerotic', 55% for 'mixed cellularity', and 35% for 'lymphocyte depleted'.

SURVIVAL		**5yrs**	**10yrs**
Stage I	Single group of nodes	**90%**	**90%**
Stage II	≥2 groups of nodes same side of diaphragm	**80%**	**75%**
Stage III	Nodes both sides of diaphragm	**60%**	**50%**
Stage IV	Disseminated disease	**45%**	**10%**

(5) **Radiotherapy** will probably be impossible, so you will have to rely on chemotherapy. If you can provide radiotherapy, here are the relative indications for it:

Stages I-II. Comparable results to chemotherapy, but radiotherapy has fewer side-effects.
Stage III. Radiotherapy difficult because a wide area has to be irradiated; chemotherapy better.
Stage IV. Radiotherapy impractical, chemotherapy necessary.

(6) **Chemotherapy preparation**
Prepare for chemotherapy (37.4). Sadly this is too costly in most instances; the regime mostly used is a variation of the 'MOPP' combination:

'MOPP' VARIATION: AN ESTABLISHED COMBINED DRUG REGIME FOR HODGKIN'S LYMPHOMA
Chlorambucil 6 mg/m², max dose 10mg, PO days 1-14
(cyclophosphamide 1g/m² may replace chlorambucil).
Vincristine 1·4mg/m², max dose 2mg, IV days 1&8.
Procarbazine 100mg/m², max dose 200mg, PO days 1-14.
Prednisolone 40mg/m², PO days 1-14.

USING THE DRUGS
Add sedation with 25-75mg chlorpromazine orally.
Then use chlorambucil PO and vincristine IV 2h later. Use a freshly made preparation in a free-running IV drip.
CAUTION! A leak will cause a severe local skin or subcutaneous tissue reaction, so infuse it with the greatest possible care.

Repeat the course monthly. Complete 6 courses, and then review monthly. Measure the total and differential white count, the Hb and the platelets weekly. Assess the response on the Karnofsky scale (37.4) and by measuring the nodes and spleen with a tape measure.

If the white count falls <2500/µl, use ½ the dose of chlorambucil and procarbazine.

SINGLE-DRUG REGIMES. If you don't have the drugs for 'MOPP', treat with cyclophosphamide 1·5g/m² IV, repeated every 3-4wks, for 6 doses. Or chlorambucil, or vincristine or procarbazine, in the same doses as in 'MOPP'. These single drugs have about a 40-50% response rate, which is not as good as 'MOPP', but is much better than nothing.

(7) **Difficulties with chemotherapy**
If, as is more than likely, there are toxic effects, anticipate and manage them like this:

Nausea and vomiting from chlorambucil and procarbazine seldom last for >8h. Minimize them by adding chlorpromazine beforehand. They may limit the dose of procarbazine you can use, but tolerance increases with repeated doses.

Leucopenia from chlorambucil and procarbazine; the latter may also cause anaemia and thrombocytopenia, which may present late. Delay the drugs till the counts recover.

Local irritation, ulceration and peripheral neuritis occur if the drug leaks from a vein. They will not be helped by delaying the drugs; they take far too long to recover. Thrombophlebitis is also common. Change the veins used, and use a wide-bore vessel.

Peripheral neuritis, from vincristine and procarbazine, presents as pain, weakness, loss of sensation, and hot palms and soles of the feet. These effects are usually reversible if the drug is stopped, but take time. In the next course, use only ½ the dose. Or, if pain is a problem, stop them at least temporarily. Stop them if there is any objective muscle weakness on dorsiflexion of the foot. Unfortunately, the response rate will then be much lower.

Ileus from vincristine. Delay the next dose until the condition improves, and use ½ the dose on restarting.

Stomatitis and diarrhoea from procarbazine (unusual). Use ½ the dose.

Lethargy, hyperexcitability, and fits (uncommon) from procarbazine. Stop the drug.

N.B. The steroidal side-effects of prednisolone – raised blood pressure, moon face, increased weight, peptic ulceration, the unmasking of diabetes, etc. – are seldom sufficient to stop the drug, or to reduce the dose.

If there is a relapse, the possibilities are:
(1) Radiotherapy.
(2) A second course of 'MOPP' with a response rate of about 50%.
(3) An alternative regime: adriamycin 25mg/m² IV day 1&15, bleomycin 10U/m² IV day 1&15, vinblastine 6mg/m² IV day 1&15, dacarbazine 375mg/m² IV day 1&15 monthly for 6 courses.

(c) Non-Hodgkin's lymphoma

This mixed group of lymphomas is difficult to differentiate histologically.

For the purposes of prognosis, they are conveniently divided into: (a) low grade (small cell lymphocytic and follicular); (b) intermediate or high grade (diffuse, large cell with small cell).

B-cell lymphomas are found in HIV disease.
Non-Hodgkin's lymphoma may present as:
(1) An enlarged group or groups of lymph nodes.
(2) Symptoms caused by enlarged nodes compressing a patient's trachea and/or the bronchi, the biliary tract, the bowel, the urinary system, or the spinal cord.
(3) Invasion of these structures.
(4) Involvement of the central nervous system.
(5) Fever; this is unusual and mild.
(6) Abdominal pain, diarrhoea and weight loss in primary gastrointestinal tract disease.

Diagnosis, special tests and differential diagnosis are as for Hodgkin's disease.

(1) **Staging** is the same as for Hodgkin's lymphoma, but is less important because 90% of patients present in Stages III or IV. The prognosis depends more on the histological type than on the clinical stage.

(2) **Prognosis.** 'Small cell', high-, or intermediate-grade lymphomas are more likely to respond favourably. 'Large cell' tumours are likely to be unfavourable. Nodular involvement promises a better prognosis than diffuse involvement, which is unfortunately most commonly found in children.

Untreated low-grade cases survive 7-8yrs and intermediate- or high-grade cases 2-3yrs, but of course this is much less if there is HIV disease also. All patients with HIV will relapse after chemotherapy unless antiretroviral treatment is also used (5.8). This may then become complicated because of drug interactions and blood count toxicity.

(3) **Chemotherapy** is the mainstay of treatment, and radiotherapy is no better.

Combination treatment: low-grade follicular lymphoma, or lymphoma of intermediate- or high-grade type, large cell or small cell.

Single-agent treatment: low-grade lymphocytic lymphoma with masses of tumour tissue, anaemia, and leucopenia.

No treatment: Asymptomatic low-grade lymphocytic lymphoma, in stages III or IV.

PREPARATION. Establish a baseline (37.4). If necessary, transfuse blood before starting.

'CHOP': A WELL-ESTABLISHED REGIME FOR NON-HODGKIN'S LYMPHOMA
Cyclophosphamide 750mg/m² IV on day 1
Doxorubicin 50mg/m² IV on day 1
Vincristine 1·4mg/m² (max. 2mg) IV on day 1
Prednisolone 50mg/m² orally on day 1-5
(Use these drugs monthly for 6 courses.)

17.7 Salivary gland tumours

About 85% of salivary gland tumours occur in the parotid gland (17-3A,B), 10% in the submandibular gland,

and 5% in the mucous-secreting glands inside the mouth, especially on the palate (31-16C). Similar tumours occasionally arise from the lacrimal gland (28.10, 11).

Malignant tumours need radical excision, but this is only really justified for advanced but mobile tumours of the parotid. Even after this, some recur locally.

Malignant submandibular tumours may need a block dissection of the neck for any hope of cure. However, spread to the lymphatics and bloodstream is usually late.

There are several histological varieties. The relatively benign ones are:
(1) pleomorphic adenomas,
(2) monomorphic adenomas,
(3) adenolymphomas (usually cystic).
The more malignant ones are:
(4) adenocarcinoma (including cylindroma),
(5) muco-epidermoid carcinoma,
(6) pleomorphic adenocarcinoma, and
(7) squamous cell carcinoma.
(8) lymphoma (17.6) can occur within the salivary gland.

The patient presents with a slowly growing mass in one of the salivary glands, which may be inside the mouth.

If there is any sign of a facial palsy, the facial nerve is involved, and the tumour is malignant. Unfortunately, the absence of a facial palsy does not mean that the tumour is benign. Even then, many salivary tumours extend outside their 'capsule' and need excision with a wide margin.

(a) Differential diagnosis includes:
(1) Lymphadenopathy (17.1), especially tuberculosis and actinomycosis
(2) Non-salivary tumours (neuroma, lipoma, lymphangioma) and mandibular or dental tumours
(3) Chronic sialadenitis or sialectasia
(4) Masseteric hypertrophy or tumour
(5) Mastoiditis.

CAUTION! Don't biopsy the growth. This may spread a benign pleomorphic adenoma locally, and you may damage the facial nerve.

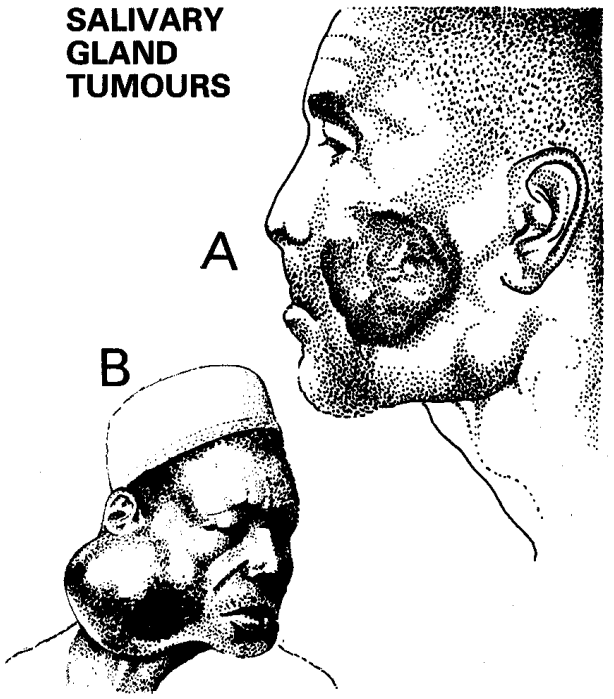


Fig. 17-3 SALIVARY GLAND TUMOURS.
A, inoperable adenoid cystic carcinoma of the left parotid, with extensive ulceration and metastases in the patient's cervical nodes. B, large pleomorphic adenoma, which has grown slowly over 20yrs. *After Adekeye EO, Robertson JM. Salivary gland tumours in Northern Nigeria. Tropical Doctor 1979;9(4):168-74 with kind permission.*

(b) Management
In a parotid tumour, facial palsy is the critical sign because it means the prognosis is poor, even after radical surgery and radiotherapy.

If there is no facial palsy, excise the tumour completely, and *don't merely shell it out*. This makes sure that the commonest lesion (a pleomorphic adenoma) is completely removed and will not recur. The patient needs a conservative parotidectomy, in which the 5 branches of the facial nerve are dissected out, and the part of the gland containing the growth is removed – either its superficial part, its deep part, or both. This operation is difficult but important, because correct surgery will cure a pleomorphic adenoma, if it is early.

For a submandibular tumour, complete excision of the gland is not so difficult.

(c) Intervention
SUBMANDIBULAR SIALADENECTOMY
(GRADE 2.5)

Excision of the submandibular gland is indicated for benign tumours or a retained deep stone. Remember the mandibular branch of the facial nerve lies superficial, and the lingual nerve lies deep, to the deep part of the gland. You should discuss with your patient possible damage to these nerves and whether their sacrifice is justified in trying to remove the tumour. The 2 parts of the gland are separated anteriorly by the *mylohyoid* muscle.

POSITION
Position the patient with the head tilted up and turned away from you, stabilized on a head ring.

INCISION
Cut 5cm below the border of the mandible, parallel to it so that it extends 2cm either side of the gland, through *platysma* down to the gland (17-4A). If the tumour is here, *don't dissect right onto it*, but at a little distance from its edge. Free the superficial part of the gland by dividing the facial artery and vein above and below it.

Make sure your haemostasis is perfect. Get your assistant to retract the superficial tissues (and, with it, the mandibular branch of the facial nerve unless this is involved by tumour) and so completely free the superficial part of the gland.

Then, get your assistant to retract the border of the *mylohyoid* medially and pull on the gland laterally (17-4B), so you can free the deep part of the gland. *Don't hold the gland with clamps*: you may cause spillage of cells which produce a recurrence.

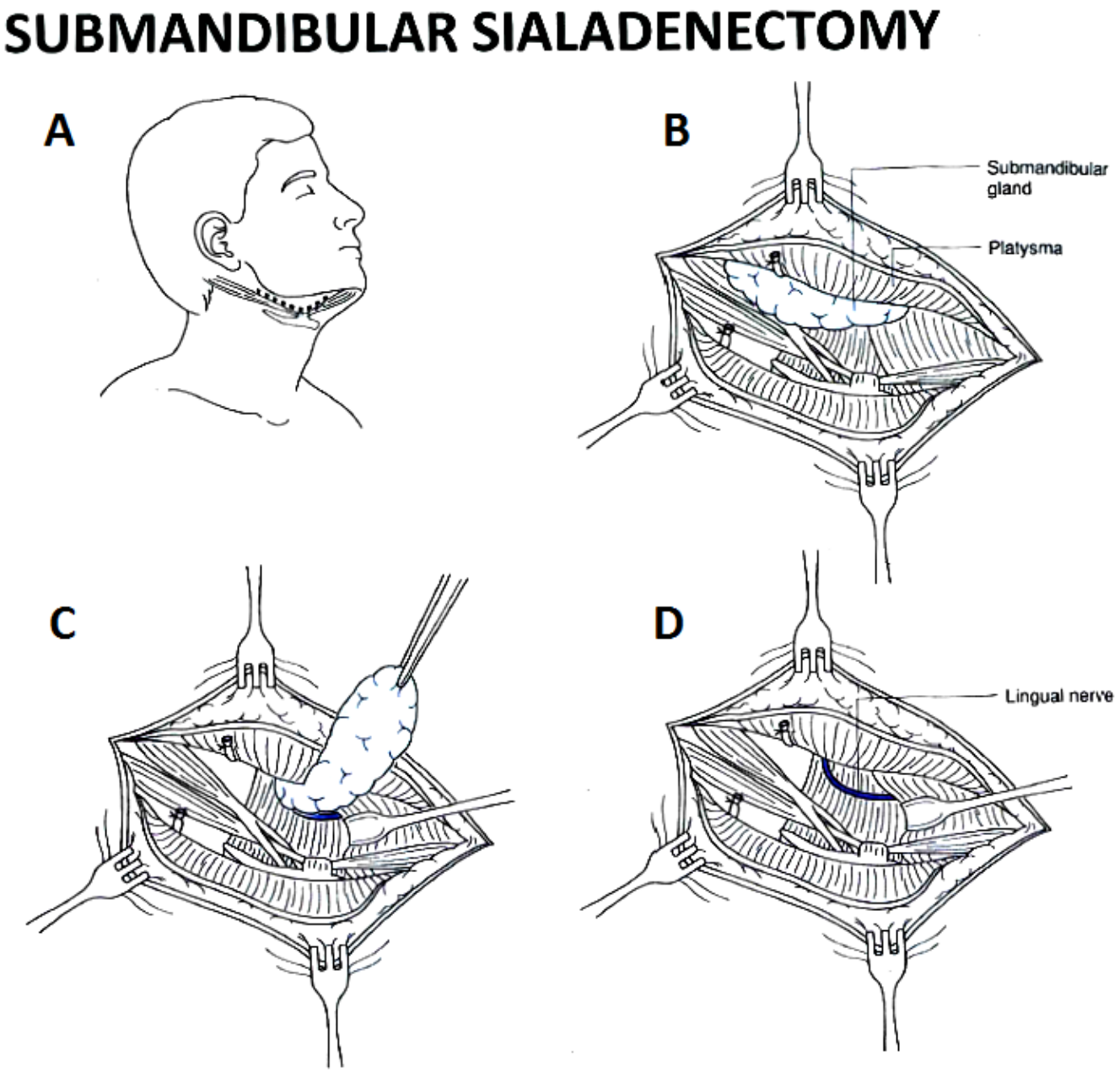


Fig. 17-4 SUBMANDIBULAR SIALADENECTOMY.
A, skin crease incision. B, divide the *platysma* and retract the submandibular ramus upwards. C, lift up the gland to reveal the lingual nerve underneath. D, the view with the submandibular gland excised.
After Greenfield LJ et al, Surgery, Scientific Principles & Practice, Lippincott-Rowen, 2nd ed. 1997 p.648.

You can sacrifice parts of adjacent structures but *take care not to injure the lingual nerve* which is in contact with and behind the deep part of the gland (17-4C). It runs along *hyoglossus*, and crosses the much thicker submandibular duct posteriorly.

You may have to cut some branches of the lingual nerve, but try to preserve the main part of the nerve, especially when you find a stone in the duct here. Then lift out the whole gland *en bloc* and make sure there is good haemostasis once you return the patient's head to the level.

Close the wound with interrupted non-absorbable sutures around a Penrose drain.

17.8 Block dissection of the inguinal lymph nodes (GRADE 3.2)

Block dissection of the nodes of the neck or axilla (24.5) is necessary in certain cases of contained malignancy, but these are difficult operations. However, the situation in the groin is different.

Squamous cell carcinomas of the skin of the leg, and the penis, and melanoma metastasize to the nodes of the groin. Removing these metastases in a block of tissue, containing the horizontal and vertical inguinal nodes, can be very successful, because these carcinomas may be slow-growing.

The femoral vein, artery, and nerves lie close to the nodes that need to be removed, and may be displaced by them. Removing them without damaging these structures is a difficult, delicate, major operation. Afterwards, there is always a lymphatic discharge and so the wound can readily become secondarily infected. Healing may be delayed, the flaps may necrose, and lymphoedema may develop in the legs (5-10%).

If you have to remove the inguinal nodes yourself, study the anatomy thoroughly before you start, and dissect carefully. Blood loss is usually not great, *provided you don't damage major vessels!*

The idea is to remove all the nodes *en bloc*, preferably without even seeing the nodes themselves; an adequate tumour clearance is essential for successful oncological surgery.

Don't try to remove nodes prophylactically, in the hope of removing metastases which you cannot feel. It will not improve the prognosis, and may be complicated by lymphoedema (34.12). Only perform a block dissection therapeutically, when the lymph nodes are palpably enlarged by secondary growth. If infection is likely to be the cause of the enlargement, confirm it by fine needle aspiration (17.2) and wait for it to improve after the amputation, and review regularly. Perform a block dissection if the nodes start enlarging. Make the decision to operate clinically, and *don't let a cytology (or biopsy) report adversely influence you;* a malignant deposit in a node may have been missed, or it may only be in other nodes which weren't biopsied.

INDICATIONS

(1) Clinical involvement of the inguinal nodes, with secondary deposits from squamous cell carcinoma of the penis or leg. If a patient's nodes have not ulcerated, removing them may provide a cure. If they have ulcerated, you may be unable to remove the mass of ulcerated tissue completely. The determining factor is whether or not they have stuck to deeper structures, especially the femoral vessels.

(2) Malignant melanoma; block dissection is often only palliative, but is not always so. The nodes may be large and ulcerate.

Don't wait for this to happen before you operate, because then the nodes may be impossible to remove. The prognosis is much worse than with squamous carcinoma, because there may already be secondary deposits elsewhere.

If you need also to perform an amputation, *e.g.* below the knee for a squamous carcinoma, perform both operations at the same time unless the primary tumour is ulcerated and infected. If you are also going to amputate the penis, make the incisions in continuity, and perform a bilateral dissection.

METHOD

Cross-match 2 units of blood. Position the patient supine with a sandbag under the buttock of the affected side. Make a vertical or 'lazy S' incision (17-5A,B): this allows you to remove skin, and produces the least skin necrosis, but finding your way may be more difficult.

If some skin is involved by tumour, keep away from the diseased area, and cut an ellipse round it, so that you can excise it with the lesion.

Make the central limb 8-10cm long, centred just distal to the mid-inguinal point, where you can feel the femoral pulse. Reflect the superior flap with about 0·5cm of subcutaneous fat, and undermine it c.5cm above your incision and expose a triangular block of tissue laterally, medially, and distally. Make its apex at least 4cm distal to any palpable node. Use a knife or scissors to dissect upwards under it, until it is about 5cm wide.

At the upper extremity of the flap, divide the subcutaneous tissues covering the abdominal muscles in the depth of the wound. Reflect a block of subcutaneous tissue downwards (17-5C), until you reach the inguinal ligament. *Don't cut the cord*. Clamp, divide, and tie the vessels as you go.

Divide the *fascia lata* over the lateral edge of *sartorius* and free its attachment. Try to save the lateral cutaneous nerve of the thigh going through it.

Cut through the subcutaneous tissues at the edges of the triangular mass, down to the deep fascia or muscle (17-5D). As you do so, find and clamp the saphenous vein at the lower end of your dissection. Tie it with 0 silk.

Its surface marking is a line from just medial to the mid-inguinal point to the medial aspect of the medial condyle of the femur.

Avoid the femoral vessels, which lie 2-3cm lateral to the saphenous vein near the distal end of the incision. Dissect down with scissors, looking for the vessels, which are covered by a sheath. The femoral vein lies posteromedial to the femoral artery, and is largely covered by it at this point, and by the strap-like *sartorius* muscle. Tie and divide any smaller vessels you meet.

Dissect the block of tissue proximally from the apex of the wound. As you do so, remove it from the femoral vessels, for about 3cm. Retract it with tissue forceps.

Reflect medial and lateral flaps, in the same way as the superior one, as far out as you can retract them comfortably. Then clear the block of tissue from the underlying muscles. On the lateral side, you will meet the femoral nerve proximally. Continuing to work from distal to proximal, reflect the block of tissue from the femoral vessels medially (17-5E). Tie and divide any small vessels you meet, close to the main ones.

CAUTION!
(1) Pulling on the block of tissue may pull up the femoral vessels, so you may think that the femoral vein is the saphenous vein. *Don't clamp, divide, or damage the femoral vein*, which may become flat and empty as you pull on the tissues.
(2) *Try not to damage the profunda femoris or circumflex vessels* (medial and lateral), which pass deep to the muscles of the thigh.

Continue to dissect proximally. This is the difficult part. Find where the saphenous vein (which may be flat and empty) joins the femoral vein. About 1cm distal to the junction it receives several tributaries: the superficial circumflex iliac, the superficial epigastric, and the superficial external pudendal veins. When you are sure you have found it, use an aneurysm needle to pass two 0 silk ligatures under it, at least 5mm apart (17-5F). Divide it between these ligatures, *away from the femoral vein!*

The block of tissue will now be almost clear, with nothing important attached to it. Dissect it free.

If you can, try to dissect out Cloquet's node carefully in the femoral ring, and remove it. Then divide the *sartorius* muscle just below its origin on the anterior superior iliac spine, and re-position it medially to cover the exposed femoral vessels; this is readily possible. *Don't leave the vessels exposed*, or they may ulcerate and bleed disastrously. Suture the *sartorius* to the fascia of the external oblique just proximal to the inguinal ligament.

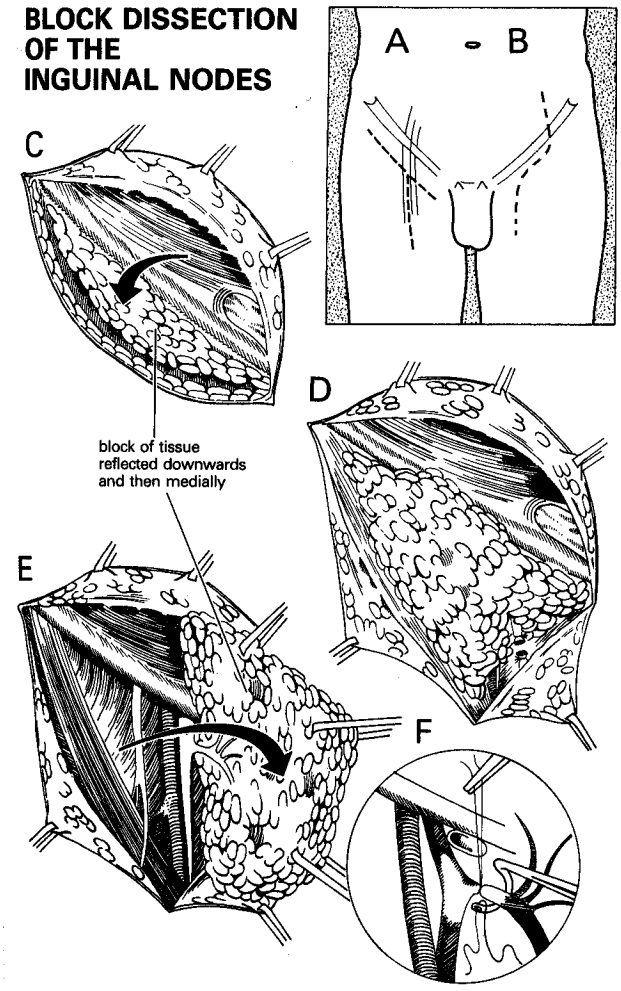


Fig. 17-5 BLOCK DISSECTION OF THE INGUINAL LYMPH NODES. A, vertical or 'T' incision. B, 'lazy S' incision. C, reflect superior and inferior flaps. D, prepare to reflect a triangular block of tissue. E, reflect a block of tissue medially from the femoral vessels. F, tie the saphenous vein.

If you can obtain good skin closure, and the wound is airtight, insert a suction drain (if you have one), with its limbs medially and laterally. If you don't have a suction drain, or the wound is not airtight, insert Penrose drains through 1·5cm incisions medially and laterally.

Beware that you don't close the wound under tension, and compress the femoral vein!

Close the skin flaps with 2/0 interrupted monofilament sutures. Apply a cotton wool pressure dressing for 48-72h. Remove drains when drainage has ceased. Remove alternate sutures on the 12th day, and the others when the wound seems sound.

Remember, if you are operating for carcinoma of the penis, do the same thing on the other side.

DIFFICULTIES WITH BLOCK DISSECTION OF THE INGUINAL NODES

Infection and necrosis of the skin edges are common. Complete healing takes time, but will occur.

If you injure a femoral vessel, usually the vein, press it to control bleeding, get help and prepare the instruments you need (3.1,2). Clamp the vessel above and below with artery forceps covered with suitable pieces of rubber catheter to avoid further injury to the vessels, or better, use bulldog clamps. If possible, close the hole carefully with non-absorbable sutures, then remove the clamps.

If you cannot repair a vein and so control venous bleeding, tie the vein above and below the wound. The leg will swell, but will usually improve in time. It is rare for it to become gangrenous and be lost.

If the tumour is too big or too fixed, *don't attempt heroic surgery which may cause catastrophic haemorrhage* and result in a gangrenous leg; the tumour is anyway too advanced for surgical cure.

If you spill tumour cells from one or more nodes, there will almost certainly be a recurrence of tumour. You can reduce this risk slightly by generously washing the operative field immediately with diluted hydrogen peroxide and betadine.

If closure of the wound is difficult, *don't close it under tension*. If there is suitable muscle in the bare area, apply a split skin graft immediately and suture it in place. Or, take a graft now, store it wrapped in paraffin gauze in sterile saline, and apply it 5 days later. If the femoral vessels are exposed, mobilize the *sartorius*, as described above. Or use a *gracilis* flap.

If lymphoedema develops, advise raising the leg at night, and prop it up when sitting. If possible, apply a graduated compression elastic bandage or, as a poor second-best, a crêpe bandage.

18 Hernias

18.1 General principles

An external abdominal hernia is the protrusion of the contents of the abdomen (any abdominal organ, part of the omentum, or peritoneal fat) through an abnormal opening in the abdominal wall. The swelling varies in size from time to time, but tends to become larger.

If you or the patient can easily return the contents of the hernia to the abdomen, it is reducible, and you can arrange repair at the patient's convenience. A reducible hernia expands on coughing; any bowel in it may gurgle as you reduce it, and if it contains omentum, it feels doughy. A hernia may be congenital (existing at birth) or acquired (through increased intra-abdominal pressure from pregnancy, ascites or massive tumour, heavy lifting, coughing, straining to pass urine, or constipation).

There may be several consequences:
(a) Irreducibility. Coughing or straining may push omentum, or a loop of bowel, through the neck of the sac, after which oedema may prevent spontaneous reduction. This is more likely the *smaller* the hernia defect. Sometimes, you may find the hernia reduces spontaneously with sedation. Occasionally you may be able to effect reduction manually (taxis, 18.6). *This is dangerous if you use force*.

(b) Obstruction. A hernia is one of the commonest causes of intestinal obstruction (12.2, 12.3). Again this is more likely the *smaller* the hernia orifice is. Bowel outside the hernia can rarely also twist and obstruct (12.8, 12.9).

(c) Strangulation. Blood may be able to enter but not leave the organs in a hernia, so that they swell. This is more likely to happen in a hernia with a narrow neck, *i.e.* femoral, or inguinal. If the swelling persists for >6h, the arterial blood supply is cut off and the organs in a hernial sac become ischaemic (strangulated, 18-2A).

If this happens to the omentum or Fallopian tube, the risk is small. But if the bowel becomes gangrenous, peritonitis and septicaemia at worst, or a fistula and cellulitis at best, will follow. If more than a little of the bowel strangulates, it cannot propel its contents onwards normally, so it obstructs. Most strangulated bowel is therefore obstructed also (18.6). Important exceptions are Richter's, (18-2B), Littré's (18.3), Amyand's, and de Garengeot's (18.7) hernias.

N.B. ***Incarceration.*** This is an imprecise term. When a hernia strangulates, it suddenly becomes painful, tense, and tender, and loses its cough impulse. Even so, you will often find it difficult to know if a hernia is merely irreducible and obstructed, or whether it is strangulated, because pain and constipation are present in both.

Pain usually remains colicky until ileus and peritonitis develop, so the change from colicky to continuous pain is a bad sign. Occasionally, a strangulated hernia causes so little pain that a patient does not call your attention to it. Usually, however, the pain, the general condition, and the signs at the hernial site are reliable indicators.

Unfortunately, you have no way clinically of being certain what has been caught in a hernial sac, and neither can you be sure clinically that whatever has been caught has not strangulated. Obstruction is ultimately as dangerous as strangulation, because, if you leave it, strangulation usually follows. So, *be safe, and treat all painful, tense hernias as if they were strangulated*.

If only the omentum strangulates, there is localized abdominal pain, but the attacks of general abdominal pain and vomiting may stop, with subsequent normal bowel action. Gangrene is delayed, but after days or weeks the necrotic omentum may become infected, so that a local abscess or general peritonitis follows.

Common sites of abdominal wall hernia are: inguinal (18.2), femoral (18.7), umbilical (18.10), paraumbilical (18.11), and epigastric (18.12). Rarer sites are lumbar, Spigelian (lateral ventral, through a defect in the *transversus* aponeurosis and *internal oblique* muscles), obturator, perineal or gluteal. Any abdominal wall incision can result in an incisional hernia, but the commonest is the lower midline abdominal incision.

There are some rarer, but important, types of inguinal hernia:

If only part of the wall of the bowel is involved, this is a Richter's hernia (18-2B). This is particularly dangerous because:
(1) the bowel may strangulate without being obstructed, so vomiting may be absent and bowel action normal. Instead, there may be diarrhoea until finally peritonitis develops.
(2) the local signs of strangulation may not be obvious.

If the peritoneal lining of the hernial sac is incomplete, and an abdominal organ (laterally, the caecum on the right, and sigmoid colon on the left, or the bladder medially) forms part of its wall, this is a sliding hernia (18-2C,D).

If two loops of bowel herniate, the central segment between the 2 loops *within* the abdomen may strangulate. This is a Maydl (or W-shaped) hernia.

If the caecum and terminal ileum herniate, because the caecum is more mobile than normal, a loop of ileum may prolapse through a hiatus below the lateral paracolic peritoneum thereby created. This causes strangulation of the proximal bowel *inside* the abdomen. This is Philip's hernia.

PRINCIPLES OF HERNIA REPAIR
(1) Define the sac, separating it from adjacent tissues.
(2) Open the sac (herniotomy).
(3) Examine the sac contents and deal with them as appropriate.
(4) Excise the sac, or close it.
(5) Reduce the hernia.
(6) Repair the defect (herniorrhaphy), restoring the anatomy as best as possible.

N.B. This part is not necessary in children (18.5).

You can perform the herniorrhaphy in several ways but we recommend making a darn closing the gap between the conjoint tendon (which is formed by the fibres of the *internal oblique* and *transversus*) and the inguinal ligament behind the cord.

This is a *tension-free* modification of the Bassini repair. Having done this, you then suture the external oblique aponeurosis in front of the cord. This is an alternative to what is now the standard in the developed world, the Lichtenstein repair, where a mesh is sewn in to strengthen the defect. The other 'definitive' operation, *the Shouldice repair, is difficult to do correctly and is not recommended.* The Lichtenstein method introduces a foreign material which is very hard to remove if it becomes infected. Fixing the mesh may cause a chronic neuroma, which is very painful. So you should be wary of using a mesh: use it only in special circumstances (18-13).

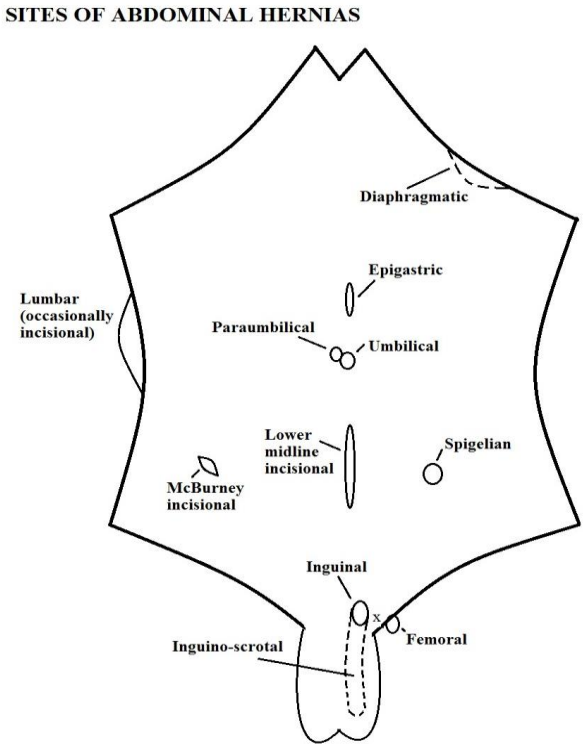


Fig. 18-1 SITES OF ABDOMINAL HERNIAS.
'x' marks the site of the left pubic tubercle.

Direct hernias represent a bulge in the posterior abdominal wall, and so there is rarely a hernia sac to dissect out. Their repair (herniorrhaphy) is usually simpler, unless they are very large.

PROBLEMS OF HERNIA REPAIR
(1) The sac may have no obvious neck (unless it is a Busoga hernia, where there is a narrow defect), so that you don't excise it; instead, you tuck it in (18-10).
(2) The weak area in a direct hernia is ill-defined, and tends to involve all or most of the posterior wall of the inguinal canal. Here a mesh is most useful: you can sterilize mosquito netting to give a perfectly acceptable alternative. You can strengthen this by suturing the external oblique aponeurosis behind the cord: *something you should never do with an indirect hernia*.
(3) A patient with a direct hernia is likely to be older with poor tissues, and perhaps prostatism, a urethral stricture, dyspnoea, a cough, or constipation, all of which will stress the hernia repair.
(4) The bladder may enter the hernia, and is easily injured.
(5) A direct hernia is twice as likely to recur as an indirect one, especially if it is a sliding hernia.

COMMON MISTAKES OF HERNIA REPAIR
(1) To forget to examine the hernial sites of anyone with abdominal pain or vomiting, especially if the hernia lies under an apron of fat.
(2) To forget the possibility of a Richter's hernia (18-2), which may confuse the diagnosis by causing diarrhoea instead of constipation.
(3) To persist in using taxis (18.6) when a hernia should be operated on.
(4) To delay surgery, especially in children.
(5) To repair a hernia, especially a direct one, before dealing with the cough, asthma, constipation or poor urinary flow that have provoked it.

N.B. If you are experienced, and no suprapubic catheter has been used, you may be able to carry out a prostatectomy and hernia repair at the same operation.
(6) Not to transfix the neck of the sac high enough, so as to obliterate it completely; *a simple ligature on its own may slip off.*
(7) To fail to make sure the groin is well cleaned pre-op.

You will have many inguinal hernias to repair, so let them provide you with an unhurried opportunity to increase your anatomical knowledge and your surgical skills. As you'll find you spend much time operating on hernias, it is worth teaching an assistant to learn this procedure.

TREAT ALL PAINFUL HERNIAS
AS IF THEY ARE STRANGULATED

18.2 Inguinal hernia

Common are indirect inguinal hernias in which the abdominal viscera slip down the inguinal canal, from inside the internal (deep) inguinal ring, through the external (superficial) inguinal ring, and sometimes into the scrotum. The hernial sac is closely related to the spermatic cord, and lies in the same fascial planes.

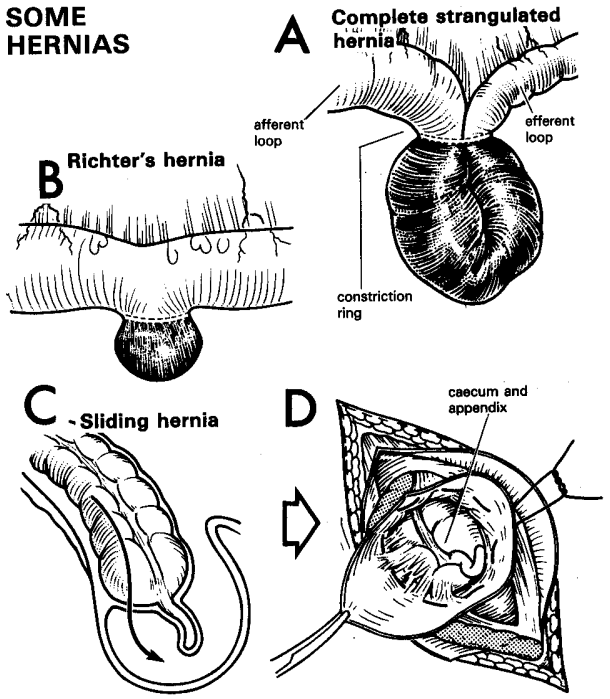


Fig. 18-2 PATHOLOGY OF SOME HERNIAS.
A, strangulated hernia. B, Richter's hernia. C, sliding hernia. D, sliding hernia opened.

A few direct inguinal hernias bulge through a weakness in the posterior wall of the inguinal canal. They don't present through the internal ring, they lack any special relation to the cord, and they don't have the coverings from the cord that an indirect hernia has. Because of the way they arise, *the spermatic cord lies behind an indirect hernia, and in front of a direct one*, but differentiating between the two before operation is not easy and experts often get it wrong. Occasionally, a patient has a hernia of both kinds (18-12).

(a) Indirect inguinal hernias are common in males. Women less often have indirect hernias, and seldom have direct ones. An indirect hernia presents a bulge in the groin, sometimes with a dragging feeling. It often extends into the scrotum. The patient may say that he felt something 'give' in the groin during lifting or coughing, just before the hernia appeared. Often the hernia has been present since childhood (congenital). An operation, which will remove the risk of strangulation, and possibly death, is almost always indicated for an obvious hernia.

Don't advise wearing a truss (a hernia support). This is expensive and difficult to get; it is unlikely to be understood that the hernia must be completely reduced before application, and it is likely to be very uncomfortable in a hot climate. *It also doesn't guarantee keeping the hernia orifice closed.*

(b) Direct inguinal hernias are of two kinds:
(1) Ordinary direct hernias, which seldom strangulate (18-13). They may cause no symptoms, and remain the same size for long periods, and so may not need surgery.

(2) A special variety of direct hernia in which the patient has a narrow defect in the conjoint tendon, or in the *transversalis fascia* (18-10). This is a Busoga (or Gill-Ogilvie) funicular type of hernia; it is not uncommon in certain areas (hence the name, Busoga, in Uganda) and may predominantly affect women. The neck of the sac is small, so bowel *readily strangulates* in this type of direct hernia; it often does so in only part of the circumference of the bowel, producing a Richter's type of hernia (18-2B).

(c) Recurrence is a problem with any inguinal hernia, especially if the patient is old and has weak muscles. Preventing recurrence needs care and skill, but curing a hernia that has recurred needs even more skill. Recurrence is less likely if you:
(1) Repair a hernia early, before it has grown too large.
(2) Tie off the neck of the sac close to the inguinal ring. If you leave the neck or fail to define the sac all round, a hernia is much more likely to recur.
(3) Narrow a dilated internal ring by bringing the edges of the *transversalis fascia* together (18-8A).
(4) Look to see if there is a coexisting direct hernia when there is an indirect hernia, or vice versa.
(5) Put the sutures in the repair through the aponeurosis of the internal oblique, rather than through its muscle.
(6) *Don't pull the darn sutures too tight*.
(7) Use non-absorbable sutures.
(8) Deal with asthma, a cough, urinary flow difficulties, or constipation *before the repair*.
(9) Control bleeding carefully because secondary infection is usually related to haemorrhage, and both will weaken your repair.
(10) Avoid sepsis by thoroughly cleaning and shaving the skin pre-operatively.

ANATOMY (18-3). The internal inguinal ring is a gap in the *transversalis fascia*, about a finger's breadth above the mid-inguinal point, midway between the anterior superior iliac spine and the pubic tubercle. The external (superficial) inguinal ring is an opening in the external oblique aponeurosis just above and lateral to the pubic spine. This aponeurosis forms the anterior wall of the inguinal canal: its posterior wall is formed by the *transversalis fascia*. As the spermatic cord passes down the inguinal canal, the muscle and tendon of the *internal oblique* and *transversus* arch over it, to form the conjoint tendon.

Divide the inguinal canal into thirds: in the lateral ⅓, the *internal oblique* forms its lateral wall; in the central ⅓, it forms its roof; in the medial ⅓ (as part of the conjoint tendon), it forms its floor. A hernia deforms this normal anatomy, but you can always see that this was its original state.

The inferior epigastric vessels leave the femoral artery and vein, and run longitudinally on the medial side of the internal inguinal ring. Direct hernias bulge medial to them, through the posterior wall of the inguinal canal, while indirect ones pass lateral to them through the internal ring. The inguinal ligament is attached to the antero-superior iliac spine laterally, and to the pubic tubercle medially. At its medial end a small curved ligament, called the *lacunar ligament*, joins it to the pubic bone. The lacunar ligament forms the medial boundary of the femoral canal. A few of its fibres continue laterally along the upper border of the pubic bone to form the *pectineal* (Cooper's) *ligament*. You can pass sutures through this when you repair a femoral hernia.

In an infant, the 2 inguinal rings overlie one another; in an adult, they separate, although not always in some people. Inside the inguinal canal you will meet two very constant vessels, but you can easily control bleeding from them: they are the cremasteric artery, and the pubic branch of the inferior epigastric artery.

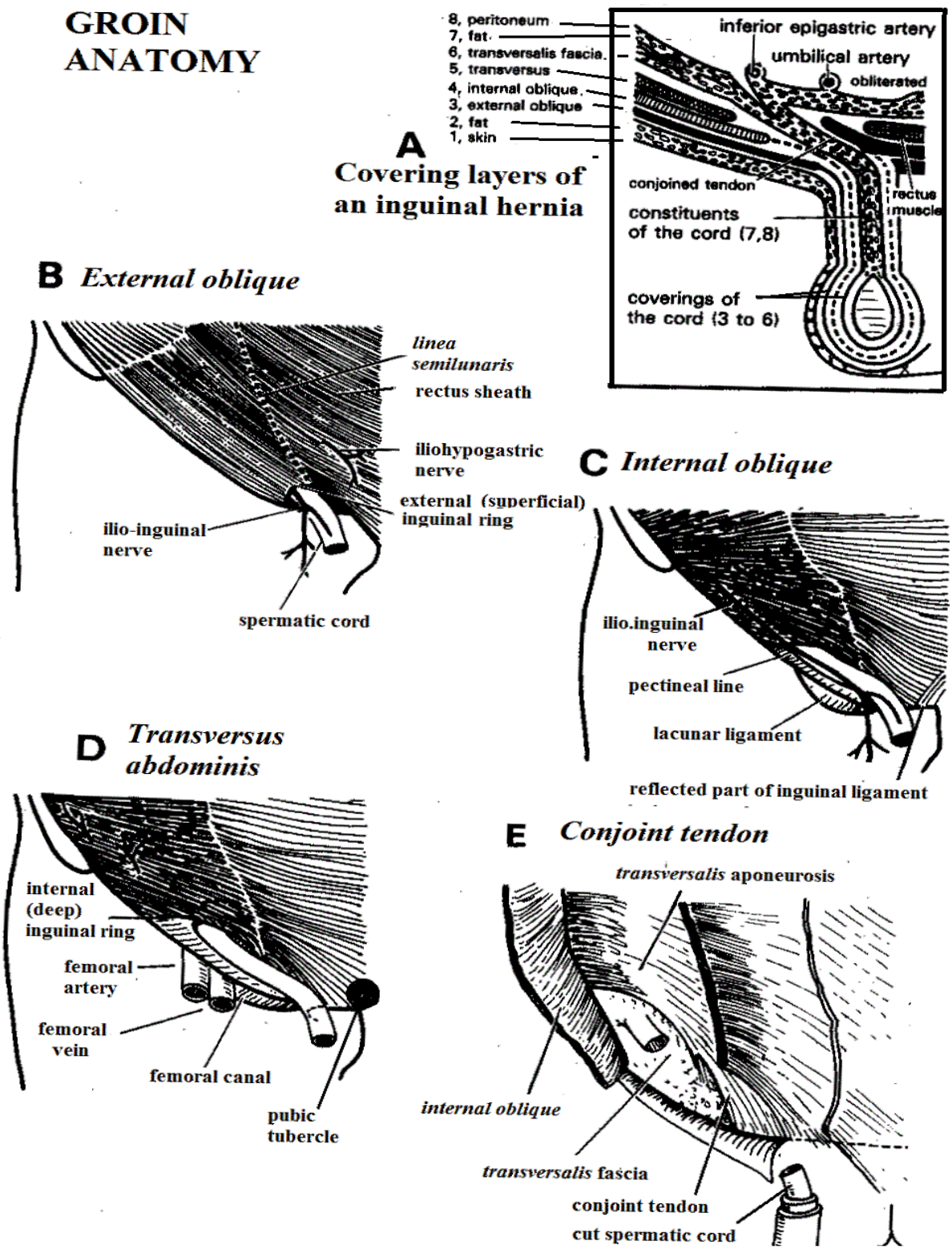


Fig. 18-3 SOME INGUINAL ANATOMY.
A, coverings of the spermatic cord, which also become the coverings of an inguinal hernia. The abdominal wall muscles are: B, *external oblique*; the *linea semilunaris* is the curved lateral tendinous edge of the *rectus abdominis*. C, *internal oblique*. D, *transversus abdominis*, lying deeper to the *internal oblique*. E, conjoint tendon, formed from the aponeurosis of the *internal oblique* and *transversus* as they arch over the spermatic cord; the *transversalis fascia* lies between the inner surface of *transversalis* and the parietal peritoneum.
After Basmajian JV, Grant's Method of Anatomy 9th ed 1975 p.183 Figs 217,219 with kind permission.

TRY TO TREAT ALL HERNIAS LIABLE TO OBSTRUCT WHILE THEY ARE STILL SMALL

(d) Uncomplicated inguinal hernias in adults

DIAGNOSIS

Ask the patient to stand, cough or strain to make the bulge appear. If it is already there, ask him to reduce it. Examine the patient lying down to see if it reduces easily, particularly with an audible gurgle. Does the hernia extend into the scrotum? Are both the testes present? Testicular atrophy is one of the complications of herniorrhaphy, and if one testis is already atrophic, you will have to be particularly careful. Take note of previous scars to see if it is a recurrent hernia.

If there is a history of an inguinal swelling that comes and goes, make a determined effort to demonstrate the hernia clinically.

If, however, it does not appear on standing, straining (*i.e.* a Valsalva manoeuvre) **or coughing**, *don't operate*. Review the situation later, and wait until you have actually seen or felt the hernia. It is worthwhile asking such patients to come for review in 3-6 months.

(e) Differential diagnosis

Suggesting a hydrocoele (27.24): a swelling with no cough impulse. If, with your finger and thumb squeezing on the spermatic cord, you can get above the swelling, no matter how large it is, it cannot be an inguinal hernia, because it cannot have come down through the external inguinal ring.

Suggesting a femoral hernia (18.7): the bulge is more globular, is below the inguinal ligament, and is just medial to the femoral vessels, whereas the inguinal hernia is above and lateral.

Suggesting inguinal lymphadenopathy: the swelling is constant, and below the inguinal ligament. (Femoral lymphadenitis may appear like a strangulated femoral hernia, but there will be no signs of obstruction.)

Suggesting filariasis: a thickened oedematous spermatic cord, with no cough impulse (34.14).

Suggesting torsion of the testis, epididymis, or both (27.25): there is a tender *scrotal* swelling which you can get above.

Suggesting a varicocoele: a soft swelling feeling like a 'bag of worms' in the spermatic cord, which fills from below, unlike the hernia which fills from above.

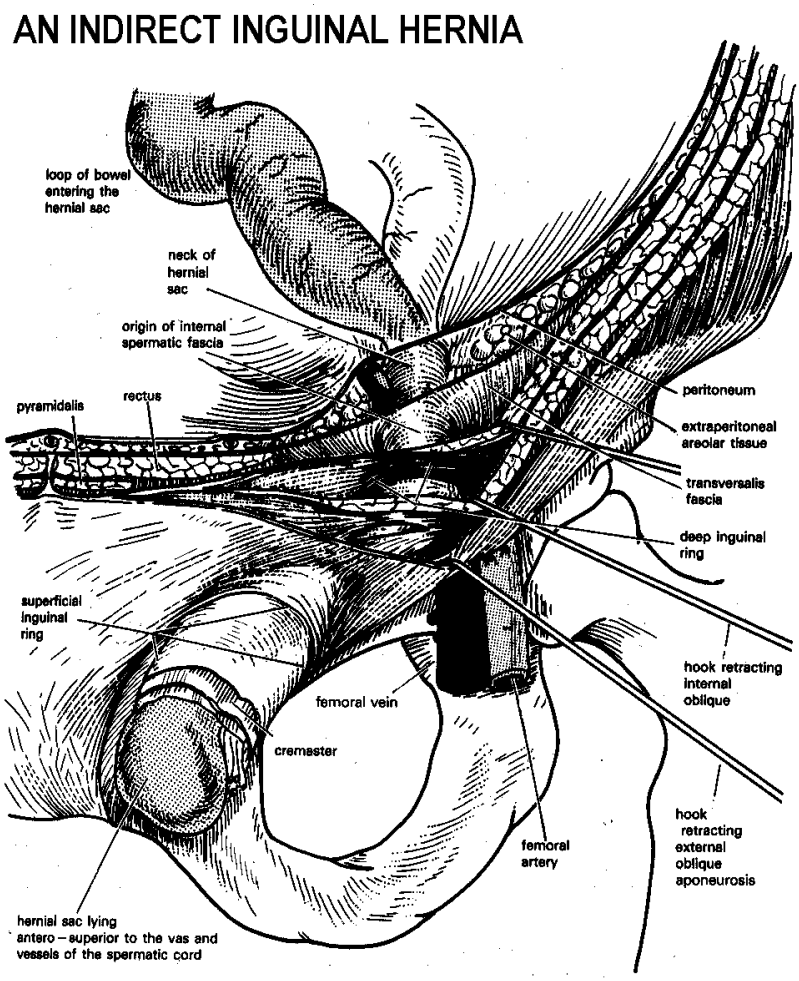


Fig. 18-4 INDIRECT INGUINAL HERNIA, showing the hernial sac passing through the internal and external rings, and lying antero-superiorly to the vessels of the cord. The narrow part is the neck and the distal part is the fundus. The sac takes a covering from each layer of the abdominal wall.

Adapted from a drawing by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

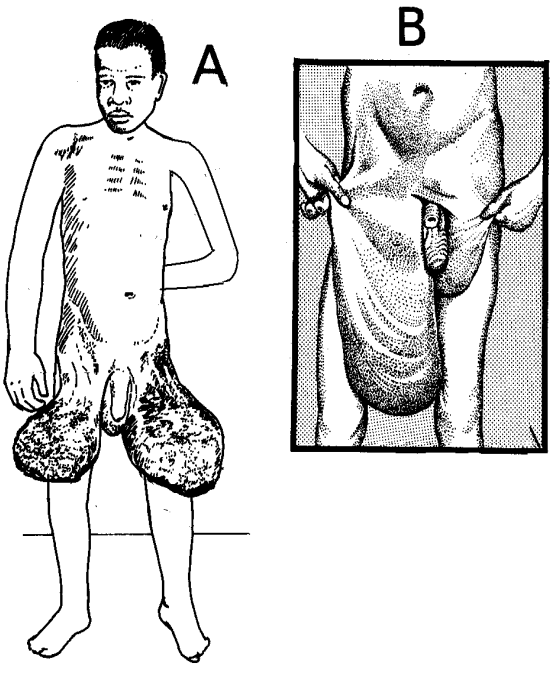


Fig. 18-5 ADENOLYMPHOCOELES or 'hanging groins' can occur in severe *onchocerciasis* or *schistosomiasis*. *Do not confuse them with inguinal hernias*. A, enormous swellings of similar sizes in a Ugandan patient. B, bilateral adenolymphocoeles of unequal size.

Kindly submitted by Dr KT Cherry.

Suggesting an adenolymphocoele: this is a mass of oedematous fibrous tissue which hangs from the groin (usually bilateral) and arises from enlarged inguinal nodes as the result of progressive lymphatic obstruction. Where *onchocerciasis* (34.8) or *schistosomiasis* are endemic, expect to see these. Look for *microfilariae* or schistosoma eggs in skin snips. Treat the disease medically before you operate. Although adenolymphocoeles are easy to remove, the wound heals badly because so many lymph vessels are severed.

(f) Management

INDIRECT INGUINAL HERNIAS IN ADULTS: HERNIORRHAPHY (GRADE 2.4)

An indirect hernia can only be treated effectively by surgery, but if it is small, easily reducible and not painful, and the patient is very old and frail, or has advanced HIV disease or malignancy, the risks may outweigh the advantages. You can use LA, however, so poor general condition is not an absolute barrier to operation.

In a teenager, or young adult with a small indirect hernia and good tissues, **and minimal symptoms**, *don't interfere*. If it does produce pain, all you need do is excise the sac and narrow the internal ring as it is usually a neglected congenital hernia. If it is large, repair the defect with a darn.

For bilateral hernias, discuss whether the patient wants both operated at the same time; recovery will be slower and the risk of urinary retention greater.

PREPARATION

Treat a cough, asthma, constipation or a urinary flow problem first. Persuade a smoker to stop; if the operation is elective, wait 3 months, and review the situation. Make sure the groin is washed and shaved. *Always mark the side to be operated upon beforehand.* Prepare the skin from umbilicus to mid-thigh, including the genitalia. If you are unsure of the anatomy, mark a line from the antero-superior iliac spine and the pubic tubercle with a marking pen: this is the line of the inguinal ligament.

LOCAL ANAESTHESIA FOR INGUINAL HERNIAS

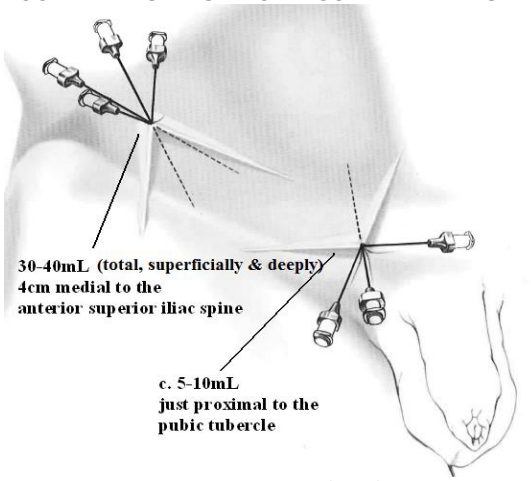


Fig. 18-6 SITES OF INJECTION of LA for inguinal hernia repair.
After Eriksson E (ed). Illustrated Handbook in Local Anaesthesia, Lloyd-Luke 2nd ed 1979 p.53 Fig 41.

ANAESTHESIA

(1) LA infiltration (18-6) is excellent for ordinary small and medium hernias. It will show up the tissue planes beautifully. It is also useful if the patient is old and feeble. *It is not satisfactory if the hernia is strangulated*. *Don't use it in children, or if the patient is tense and anxious*. Remember that pulling on the spermatic cord or peritoneal sac will still be painful!

(2) Epidural or subarachnoid (spinal) anaesthesia is excellent for all sizes of hernia, because relaxation is so good.

(3) Ketamine with relaxants or GA, preferably with relaxants.

INCISION

Make the incision 2cm parallel and above the inguinal ligament, from just lateral to the mid-inguinal point to just medial to the pubic tubercle. For small hernias it can be a little shorter, and for large ones a little longer (18-7A).

Find and tie securely or diathermy the superficial epigastric and superficial external pudendal vessels. If they bleed later, a post-operative haematoma results.

Apply straight haemostats to all bleeding points, and secure haemostasis. Cut through the two layers of the superficial fascia down to the shining fibres of the *external oblique* aponeurosis (18-7B).

Clear the upper skin flap from the underlying aponeurosis by swab dissection, to expose a wide area of aponeurosis above the internal ring. Free the lower flap in a similar way to display the inguinal ligament, and its attachment to the pubic spine. Display the external ring, and identify the line of the inguinal canal. Insert a self-retaining retractor to separate the skin edges.

(1) OPEN THE INGUINAL CANAL.

Make a short split incision in the aponeurosis of the *external oblique* in the length of its fibres over the inguinal canal, and extend it with a half-closed blade of scissors laterally and medially opening up the external ring. Free the upper edge of the *external oblique* aponeurosis, including its extension as the *cremaster* muscle, from the underlying *internal oblique*, as far as the outer border of the rectus sheath.

Clip the upper and lower borders of the *external oblique* aponeurosis with straight haemostats. If you do this, you will not mistake them later for the curved haemostats you have used to control bleeding. Lift each flap of aponeurosis and use gauze or sharp dissection to free it as far as the inguinal ligament inferiorly, which is the lower border of this aponeurosis. You will now see the *internal oblique* muscle, leading medially to the conjoint tendon. Re-apply the self-retraining retractor to separate the edges of the aponeurosis.

Look for the ilio-hypogastric nerve, and, a little below it, for the ilio-inguinal nerve on the surface of the *cremaster*, in front of and slightly below the spermatic cord.

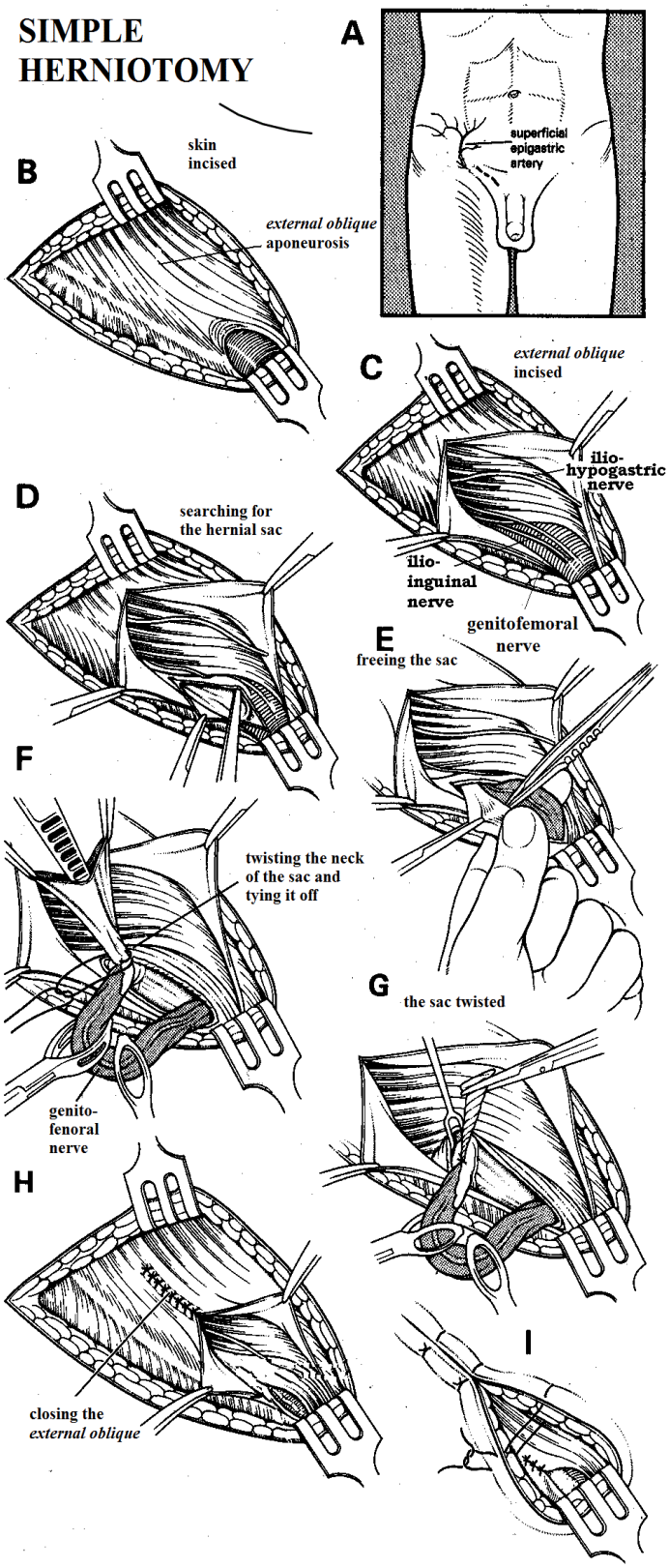


Fig. 18-7 SIMPLE HERNIOTOMY.
A, site of the incision. B, incise the skin and expose the *external oblique* aponeurosis. C, open the *external oblique* aponeurosis, from the external ring laterally, to expose the *internal oblique* with the ilio-hypogastric and ilio-inguinal nerves. D, open the spermatic cord to search for the sac. E, free the sac. F, open the hernia sac. G, tie the sac off and remove the excess. (If a repair is needed, it would be done at this point.) H, close the *external oblique* aponeurosis. I, operation complete.
After Maingot R, Abdominal Operations, HK Lewis 4th ed 1961 p.874, 876, Figs 1,3 with kind permission.

Mobilize the ilio-inguinal nerve, and retract it behind the haemostat on the lower flap. Try not to crush or overstretch either of these nerves, or include them in a suture, because this may cause persistent post-operative pain. Pick up the cord where it crosses the pubic tubercle and gently free it posteriorly, dissect it out enough to put a sling or rubber catheter round it (kinder than the forceps shown in the figure, 18-7F), and retract it. With your left thumb in front and index finger behind, try to stretch the cord and identify the sac. This might be readily visible, but usually needs you to split the fascial layers of the cord to see the curved white edge of the sac. You will see this lying close to and in front of the spermatic cord, which contains the vas and the spermatic vessels.

CAUTION!
Don't try blunt dissection near the ring where landmarks are hard to distinguish, especially if there is much extraperitoneal fat.

At this point, if you identify the vas and vessels, but no sac, examine the posterior wall of the inguinal canal for signs of a direct hernia. Examine also the conjoint tendon, where the Busoga type of direct hernia occurs. Look also for a femoral hernia (18.7).

If you have difficulty outlining a hernial sac, open it and insert the index finger of your left hand. Use this to help you define the rest of the sac for further dissection.

(2) FIND AND FREE THE SAC.
If you are using local or spinal anaesthesia, ask the patient to cough. The sac will swell slightly. It may be easy to find, or difficult if fibrous tissue has formed round it. Catch an edge with forceps, and retract it upwards and outwards (18-7E).

Dissect the sac carefully, hold it with a haemostat, and keep close to its edge. Hold it at extra places as necessary. Usually, sharp dissection with scissors is better than using gauze, unless the tissues are very loose, because there will be less oozing. Free the sac from strands of the *cremaster* at their origin from the *internal oblique*. Separate it from the cord with non-toothed forceps by working transversely to its long axis, using a combination of scissors and gauze-on-finger dissection. If there is extraperitoneal fat round the sac, and it obscures your view, remove it.

CAUTION! Be sure to find and define clearly:
(i) the vas,
(ii) the spermatic artery, and veins (usually 2-3).
N.B. You may accidentally divide all these during dissection. Avoid this by keeping close to the sac.

If the sac descends into the scrotum, open the sac and make sure your finger enters the peritoneal cavity easily through it. Then holding the proximal part of the sac, divide it (but not the cord!) and drop the distal part of the sac back into the scrotum. (Leaving it open prevents formation of a hydrocoele.) Finally continue to dissect out the proximal part.

Dissect the sac free from connective tissue right down to its neck. You may see the inferior epigastric vessels running medial to its neck. Avoid them. If necessary, tie or diathermy any small branches. You will know that you have dissected as far as you should when you find:
(i) the deep epigastric artery and veins on its medial side.
(ii) the constriction that forms its neck.
(iii) a collar of extraperitoneal fat around it.
(iv) its wider junction with the peritoneal cavity,
when you pull it up.

TWO DETAILS OF HERNIA REPAIR

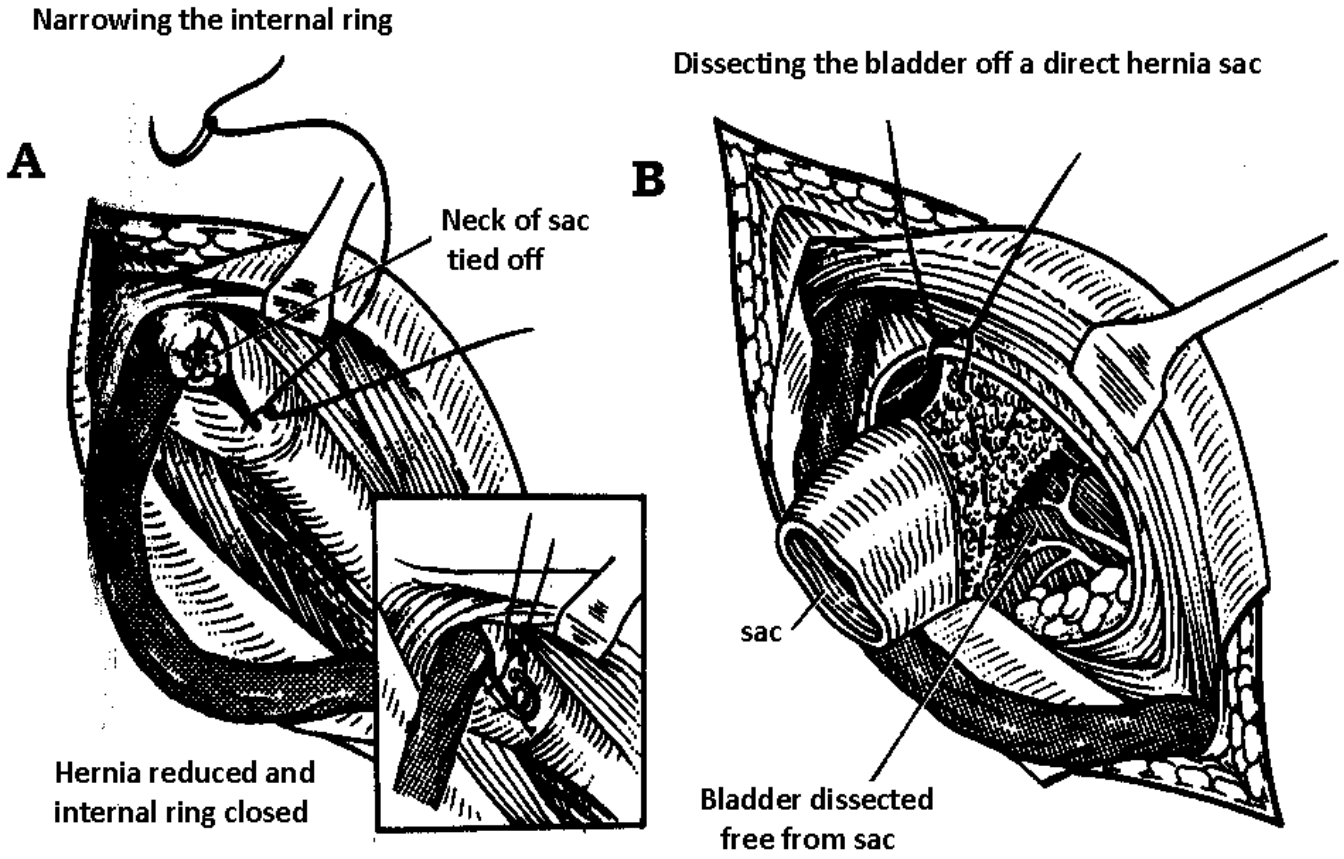


Fig. 18-8 TWO DETAILS OF HERNIA REPAIR.
A, narrowing the internal ring. In an adult a normal internal ring just admits the tip of your little finger. If it is larger than this, narrow it. Start medially and work laterally. B, bladder occasionally bulges forwards extraperitoneally on the inner side of a direct hernia, and *you can easily injure it.*
After Rob C, Smith R. Operative Surgery. Butterworth 2nd ed 1969 Vol 4 Part 1 p.223 Fig 8, p.229 Fig 25 with kind permission.

(3) OPEN THE SAC.
Open its fundus (if you have not already done so) between haemostats, as if you were opening the peritoneal cavity. The sac has a moist shiny inside surface. Examine the neck of the sac (18-7F) and look through it into the peritoneal cavity to make sure the sac is empty, and no bowel or omentum remains inside. *Make sure no adhesions remain of omentum onto the sac.*

If the hernia is irreducible, *beware: you can very easily open bowel as you open the sac!*

If one side of the neck is thick, *there may be bladder or bowel in its wall*. This is more likely in a sliding hernia, either direct or indirect.

(4) CLOSE THE SAC.
Twist its neck until the turns reach the internal ring (18-7G). If there is any bowel or omentum in the sac, this will force it back into the peritoneal cavity. Transfix the neck as far proximally as you can with absorbable suture and encircle the neck of the sac, tying it with a triple throw. Leave the ends of the knot long, and hold them with haemostats.

If the neck of the sac is wide, place haemostats round it from outside, divide it distally, and close it with a continuous suture, as if you were closing the peritoneum of an abdominal wound.
Divide the stump 1cm distal to the ligature. Examine it. When you are sure that the ligature is not going to slip, or ooze, cut its threads. If it is loose, apply another ligature or a continuous suture. When you release the stump, it will quickly disappear from view under the arched fibres of the *internal oblique*.

CAUTION!
(i) *Don't tie the sac distal to the internal ring* as the hernia is more likely to recur.
(ii) *Don't include the vas in your ligature*.
(iii) Leave the distal end open, if you need to divide the sac, or else a hydrocoele may result.
(iv) Avoid damaging small vessels; good haemostasis is essential to prevent a haematoma.

(5) NARROW THE INTERNAL RING.
Feel the size of the internal ring. In an adult, a normal internal ring should not admit your index finger. If it is wider than this, it is dilated. If it is only a little dilated, narrowing it will be enough, as it is in children.

If the internal ring is only moderately dilated, suture it with monofilament nylon, starting medially, and suturing laterally, until the ring fits snugly around the cord *but does not strangle it* (18-8). Tie the inner and outer ends of the suture together to prevent the inner end of the suture line pouching forwards.

CAUTION!
(i) *Don't leave the internal ring too wide*, or the hernia will be likely to recur.
(ii) *Don't constrict the internal ring too much*, or the testis may atrophy.

With experience you may narrow the internal ring as part of your repair, as described below.

In a female, an inguinal hernia is probably caused by a congenital sac which is firmly stuck to the round ligament. This is narrower and less vascular than the spermatic cord. The inguinal canal is smaller and you will hardly see anything to represent the *cremaster*. The hernial sac can extend only to the *labia majora*. Proceed as above, dissecting the sac from the round ligament. Use sharp dissection to free this, the sac and the vessels from the labium, or blunt dissection if the tissues are very loose.

Clamp, tie, and divide the round ligament close to its insertion. Then clean it and the sac as far as the internal ring. Open the sac, inspect its inside, and then probe it to make sure it is empty. Grasp the sac, the round ligament and their vessels. Transfix, and tie them, leaving the ends of the ligature long. Then divide these tissues 1cm beyond the ligature.

Use the long ends of the ligature to anchor the stump to the aponeurosis of the external ring above and lateral to the internal ring.

Obliterate the now empty inguinal canal with a few sutures joining the conjoint tendon and the *transversalis fascia* to the inguinal ligament. This is the so-called standard Bassini repair; *it is satisfactory only if there is no tension on the suture line*. Close the internal inguinal ring completely.

There is nothing to strangulate. If the muscles are weak, draw the upper edge of the external oblique aponeurosis toward the inguinal ligament with a darn: this is a modified Halsted repair.

(6) REPAIR.
Our recommended technique for the male is the Moloney nylon darn repair (18-9), which has been shown to be equivalent in strength and durability to the Lichtenstein mesh repair.

After you have completed the herniotomy, release the straight haemostat that you originally inserted on the lower flap of the *external oblique*, and replace it with the self-retaining retractor so that the cord lies behind the aponeurosis while you repair the posterior wall of the inguinal canal. This will keep the cord out of the way while you proceed with the repair. Alternatively, hold the cord with gentle traction using slings (18-9) or Babcock forceps, or *within* Lane's forceps.

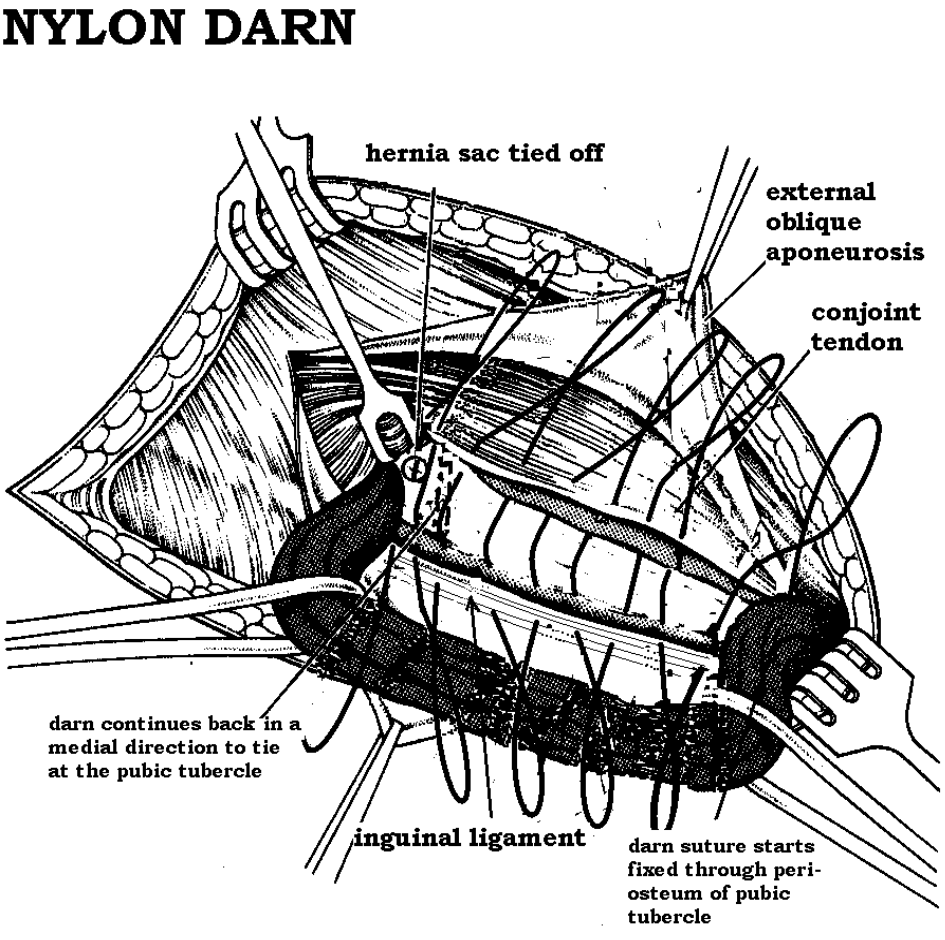

NYLON DARN
hernia sac tied off
external oblique aponeurosis
conjoint tendon
darn continues back in a medial direction to tie at the pubic tubercle
inguinal ligament
darn suture starts fixed through peri-osteum of pubic tubercle

Fig. 18-9 MOLONEY NYLON DARN REPAIR. Use #1 monofilament to make the darn. The first suture goes through the periosteum of the pubic tubercle.
After Maingot R Abdominal Operations, 4th ed 1961 p.879 Fig. 4 drawing 1. HK Lewis, with kind permission; Kirk, RN, Williamson, RCN General Surgical Operations, Churchill Livingstone 2nd ed 1987 p.29 Fig 3.2.

N.B. **The Desarda technique is another tension-free method: here you take an undetached strip of the *external oblique* aponeurosis from its normal anterior position to bolster the posterior wall by suturing it to the inguinal ligament.**

Clean away all the areolar tissue from the upper surface of the inguinal ligament. Retract the fleshy arching internal oblique muscle upwards, and expose the aponeurotic part of the *transversus* and *internal oblique* muscles which form the conjoint tendon. Use this tendinous layer for reconstruction, not the overlying muscle layer, unless the aponeurosis is poorly developed.

Use 1/0 monofilament, or steel, on a round-bodied half-circle needle (*avoid a cutting needle as this is one of the most hazardous operations in terms of pricking yourself!*) to apply a continuous figure-of-8 loop (herring-bone) suture 8mm apart from the arching fibres of the conjoint tendon above, to the inner shelving margin of the inguinal ligament below (18-9). *Don't pull these structures tight: think of the darn as a patch to repair the defect*. Put a narrow retractor at the medial end of the wound, and take the first bite through the periosteum over the pubic tubercle and leave this end long. Proceed from the medial side laterally taking substantial (6-8mm) bites of the aponeurosis.

To avoid splitting the inguinal ligament, take bites which are alternately large and small. Space the sutures evenly, and *don't go too deep*, or you may puncture the underlying femoral vessels. When you reach the internal ring, return medially in the same fashion making a continuous figure-of-8 loop to finish and tie on the pubic tubercle.

CAUTION!

(i) *Beware of the femoral artery and vein*, which lie just behind the inguinal ligament under the mid-inguinal point. Injuring the femoral vessels is the most serious potential complication of hernia surgery.

(ii) *Don't strangulate the cord with your most lateral suture*. Make sure you can still insert the tip of your forceps through the internal ring, alongside the emerging cord.

(iii) Use non-absorbable sutures only. *Don't use silk*; if it becomes infected, it will cause sinuses, and you will later have to pick out every piece.

(g) Ordinary direct hernias

In a direct hernia, the posterior wall of the inguinal canal is weak and flabby and provides little resistance to your fingers as you press. There is usually an obvious bulge medial to the epigastric vessels. The cord almost always lies anterior to it.

If its medial wall feels thick and fleshy, suspect that there is bladder in it and there is a sliding hernia present. There is almost never a danger of strangulation, in contrast to the Busoga hernia.

Don't try to open, tie, or excise the sac of a direct hernia, unless it is of the funicular type. Push it inwards with a sponge dissector, and while you keep it pushed in, make a darn as before.

It may help to bring the layers of the *external oblique* aponeurosis together behind the cord (an anterior transposition of the cord), to strengthen the inguinal region. This is a modified Halsted repair. If it is convenient, overlap the flaps, suture the upper one to the inguinal ligament, and bring the lower one on top of it so as to overlap it about 2cm.

CAUTION! Make quite sure that the unusual double (Saddle or Pantaloon) *hernia, a* COMBINATION OF A DIRECT AND INDIRECT HERNIA (18-12), *is not present.* A few minutes looking for an indirect sac is time well spent!

(h) Busoga (Gill-Ogilvie) direct hernia

This is an unusual, but important, variety of direct hernia, peculiarly found quite frequently in certain areas of Uganda (and elsewhere), predominantly in women. Its importance is that it is a type of direct hernia where the bowel may strangulate, like a Richter's hernia (18-2B, 18-11).

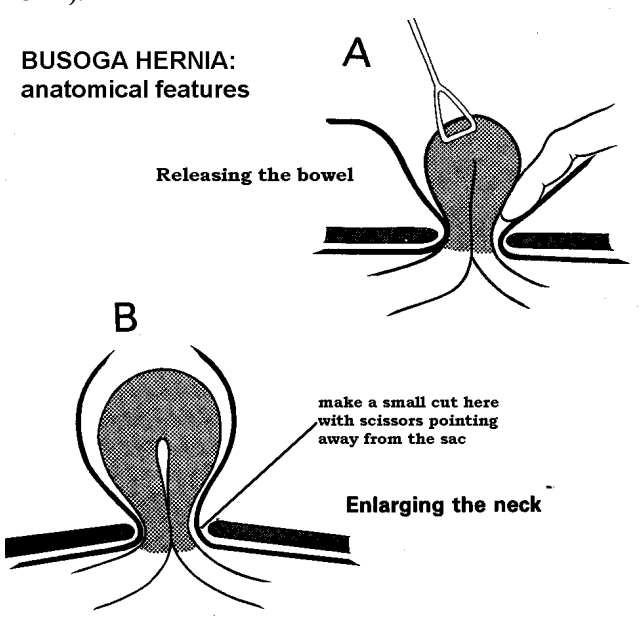


Fig. 18-10 BUSOGA (Gill-Ogilvie) HERNIA: ANATOMICAL FEATURES.
A, release a strangulated Busoga hernia. As soon as the sac is opened, grasp a loop of trapped bowel gently with Babcock forceps. As you do so, gently but firmly stretch the neck of the sac with the tip of your little finger. B, sometimes you have to make a small cut in the fibrous edge of the ring. You can release a femoral hernia in much the same way.
Kindly contributed by Brian Hancock.

You will see a tight bulge of bowel coming through the conjoint tendon medially (18-11) or *transversalis fascia* (18-12B). Hold the bulging bowel lightly with Babcock forceps to prevent it slipping back (18-10A). Cut the edge of the tight ring in the conjoint tendon with a half-open scissors cautiously (18-10B). Dilate it with your finger alongside the sac. Open the sac. Withdraw the bowel and assess it by the criteria of viability (11-6).

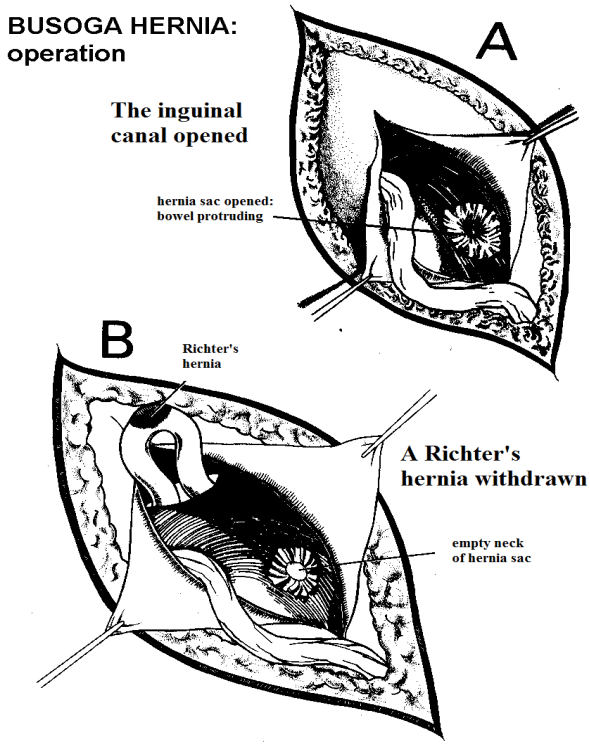


Fig. 18-11 BUSOGA HERNIA OPERATION.
A, open the inguinal canal to show a small defect in the conjoint tendon and a hernia bulging through it; open the sac. B, if the bowel slips back while you are operating, extend the incision in the *external oblique* laterally, then split the *internal oblique* and *transversus*, as in an appendicectomy (14.1). **Withdraw the bowel. In this way you avoid enlarging the neck of the sac and weakening the conjoint tendon.**
Kindly contributed by Brian Hancock.

If the bowel is viable, return it to the abdomen.
If the bowel is dubiously viable, leave it for 10mins covered with a warm, wet swab.
If the bowel is not viable, you will have to decide whether to invaginate or resect it.
Invaginate the necrotic area of bowel by using two layers of 2/0 or 3/0 absorbable to bring its healthy borders together in their transverse axis, so as to push the ischaemic segment safely inside the lumen, where it can safely necrose.
Do this *only* if:
(1) typical Richter-type strangulation which has produced a 'coin-like' area of necrosis with a sharp margin.
(2) *the necrotic area has not yet perforated*.
(3) *it does not extend over >50% of the circumference of the bowel*.
(4) *it does not extend on to the mesenteric border of the bowel*, because invaginating it may interfere with its blood supply.
(5) the bowel at the edge of the necrotic area is healthy and pliable.

If any of these criteria are not fulfilled, resect the necrotic segment of bowel and perform an end-to-end anastomosis (11-7).

If you need to enlarge the defect to get better access to the bowel, extend the incision in the skin a little more laterally. Then split the *internal oblique* and *transversus abdominis* about 5cm above the internal ring, level with the iliac spine, exactly as in the standard approach for appendicectomy. Open the peritoneal cavity, and withdraw the bowel for inspection, invagination, or resection. Alternatively, perform a formal laparotomy through a midline incision. Finally, excise the sac, and close the *transversalis fascia* with a few monofilament sutures.

(i) Sliding hernias (not uncommon)
If you find a boggy thickening in the wall of a hernia sac, suspect that some viscus has slid into it partly behind the peritoneum (18-2C,D). On the right, the caecum and appendix can slide into an inguinal hernia on the lateral side. On the left, the sigmoid colon can do the same (unusual). The bladder can do so on either side medially, more commonly in a direct hernia.

DIRECT AND INDIRECT HERNIA IN THE SAME PATIENT

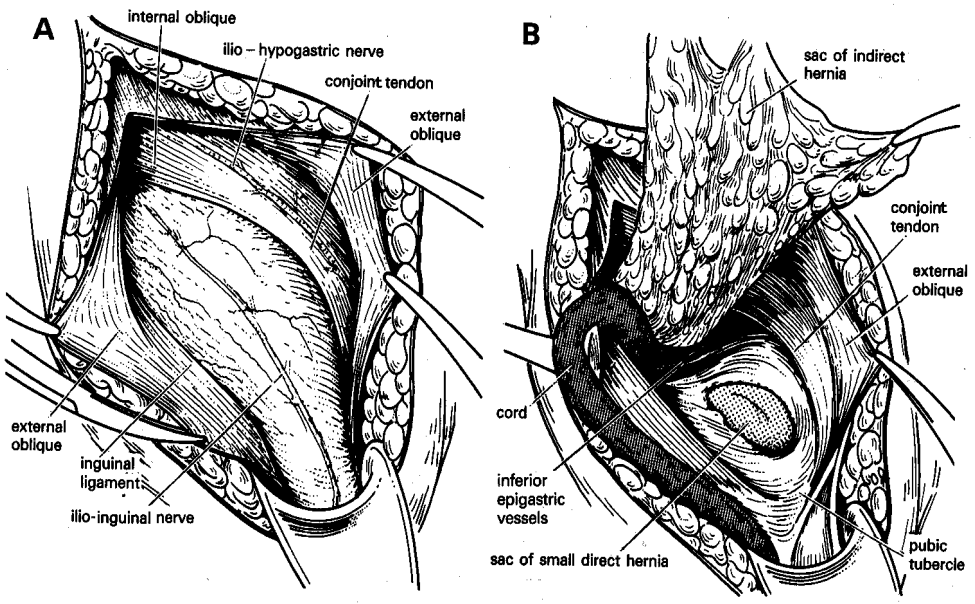


Fig. 18-12 DIRECT AND INDIRECT HERNIA in the same patient (Saddle or Pantaloon hernia). A, split the *external oblique* in the line of its fibres. Expose the cord with the ilio-inguinal nerve on its surface. Free the aponeurosis of the *external oblique*, medially to its fusion with the *internal oblique*, and laterally to the inguinal ligament. B, free the spermatic cord and retract it. Free the sac of a large indirect hernia, with some fat attached to it, from the cord up to the internal inguinal ring. Expose the pubic tubercle. (A small direct hernia protrudes through the *transversalis fascia.*) *N.B. Obesity may make dissection difficult!*
After Maingot R, Abdominal Operations, HK Lewis 4th ed 1961 p.889 Figs 15,16 with kind permission.

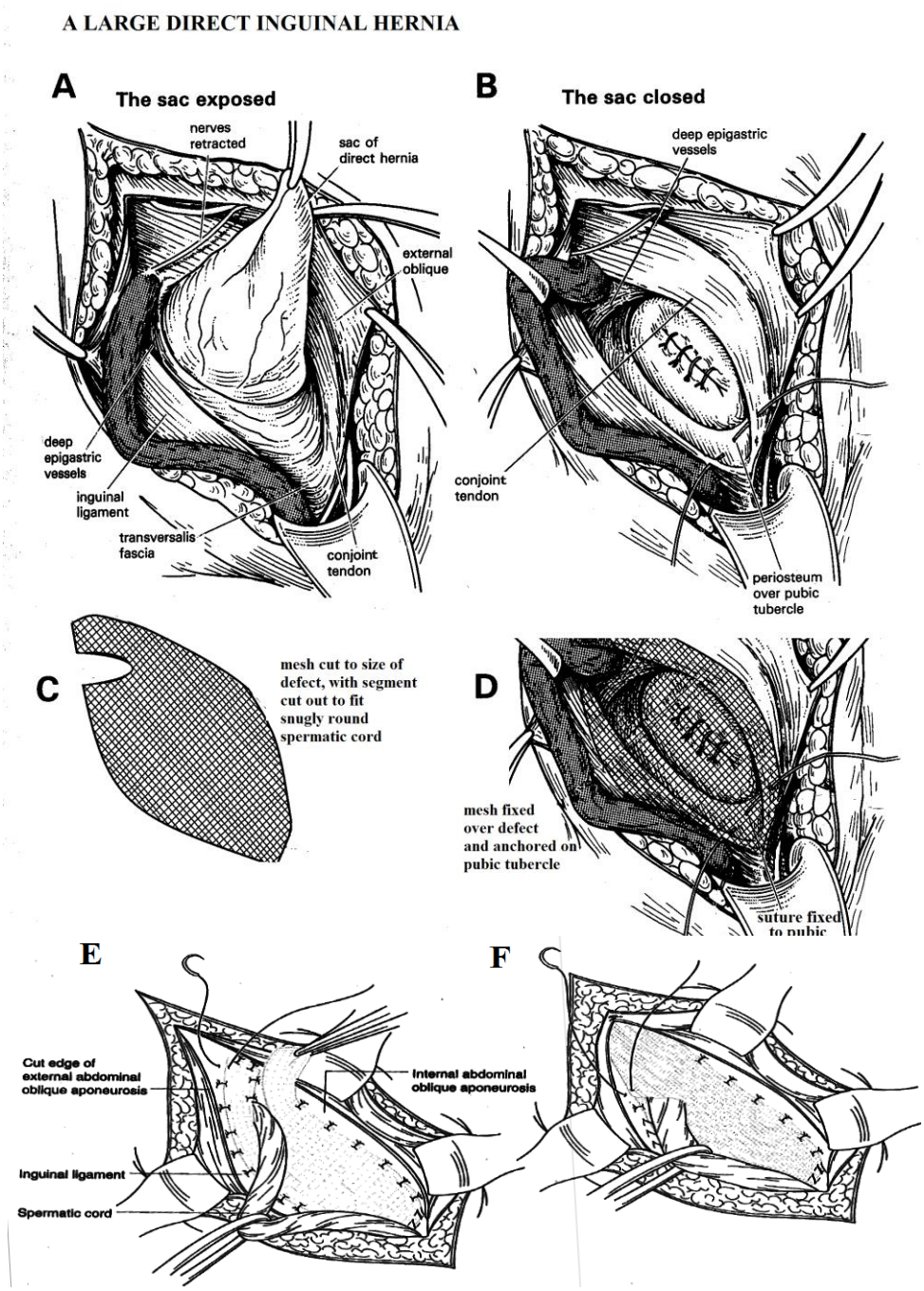


Fig. 18-13 REPAIRING A LARGE DIRECT HERNIA OF THE ORDINARY KIND. (For enormous or giant hernias, see 18.4)
A, large direct hernia protruding through most of the posterior wall of the inguinal canal. B, having reduced the hernia, close the orifice in the *transversalis fascia*. Insert the first suture in the conjoint tendon. C, cut the mesh to fit, with a defect for the spermatic cord. D, suture the mesh in place, anchoring it on the pubic tubercle. E, mesh sutured in place to the conjoint tendon and inguinal ligament. F, window around the spermatic cord closed snugly. *After Maingot R, Abdominal Operations, HK Lewis 4th ed 1961 p.890 Figs 17-20 with kind permission.*

CAUTION! If you cut through a thick part of the wall of the sac, you may enter the viscus.

You may feel something irreducible in the sac which you cannot return to the abdomen. When you open it, you find that the internal margins of the sac are impossible to identify along one side, because there is some viscus in the way. Dealing with a hernia like this can be difficult.

Therefore, *don't attempt to separate the sac from the viscus forming its wall*, and after you have reduced any other hernia contents, close the sac with continuous 0 absorbable sutures. Push the viscus with the stump of the sac into the abdominal cavity. Proceed with the herniorrhaphy.

(j) Very large hernias

If the hernia is extremely large, you will not get a satisfactory repair with the standard nylon darn *(and certainly not with a Bassini repair)*. Insert a mesh: you can make one with ordinary autoclaved polyethylene mosquito netting (18-13C). This has a breaking strength of c.150 Newton prior to autoclaving and c.350 after.

N.B. Don't use insecticide-impregnated mosquito netting! (The permethrin used is neurotoxic and may be carcinogenic.)

Suture the mesh carefully to the inguinal ligament and conjoint tendon (18-13E), leaving an opening you have cut out for the cord to pass through. Close this window with 2-3 non-absorbable sutures around the cord, placing the mesh neatly to make a snug fit.

If the tissues are very weak and stretched, you may be able to 'double-breast' the layers (fold the stretched ligaments on top of themselves) to increase their total strength: this is probably preferable to cutting away excess ligament.

Don't simply suture the mesh to *weak tissues*, because the herniation bulge will just be shifted to below (or above) the mesh.

(k) Recurrent hernias (not uncommon)
If the recurrence is an indirect hernia, usually because of inadequate closure of the internal ring, or a direct hernia, insert a darn as before. Take time to make a neat dissection. If a non-absorbable material has been used previously, and there is only a local defect, close this and leave the sound parts undisturbed.

If the tissues are very weak, put in a mesh as for a large hernia.

If the recurrence is a direct hernia, check if the ligaments are of adequate strength, and attach a mesh to these structures.

CLOSING THE WOUND AFTER ANY INGUINAL HERNIA REPAIR. Now that you have narrowed the internal ring, and done a repair, you can replace the cord. Put it back in the inguinal canal, and make sure the testis rests well down in the scrotum. Use continuous absorbable suture to repair the *external oblique* in front of the cord (unless there is a direct hernia when you can use it to strengthen the repair behind the cord), starting from the lateral side and working medially. When you reach the external ring, reduce it to a size that will transmit the cord comfortably.

Repair the well-defined layer of superficial fascia with 2/0 continuous absorbable, and the skin with 2/0 subcuticular absorbable. Insert 5-10ml of long-acting local anaesthetic into the wound.

CAUTION! Post-operative bleeding is particularly likely to occur in the inguinoscrotal region. So control all bleeding vessels carefully. Any hernia repair can be spoilt by a haematoma, especially if it becomes infected. If haemostasis is not perfect, insert a Penrose drain.

POST-OPERATIVELY, mobilize the patient, start eating and walking the same day. Check if he is passing urine well. If he is bronchitic, treat him with chest physiotherapy. If he smokes, persuade him to stop. If he is a manual worker, he should avoid lifting weights >5kg or straining for 3 months, and if possible, give up heavy work. Treat him with a laxative if he has a tendency to constipation. You should advise him to avoid sexual intercourse for 3wks. There is no restriction on driving a car after 1wk unless the groin wound remains painful.

18.3 Difficulties with inguinal hernia

(a) Pre-operative difficulties with inguinal hernias

If there is a hydrocoele and a hernia, deal with the hernia as usual. Open the hydrocoele and leave its distal end open, evert the testis and replace it outside the *tunica vaginalis* (27.24).

If there is also an undescended testis, perform an orchidopexy (27.27) or orchidectomy if it is atrophic.

(b) Operative difficulties with inguinal hernias

If the testicle is twisted, perform an orchidopexy after untwisting it (27.25), or an orchidectomy if it is non-viable (27.26).

If you cannot find the sac, and you are operating under LA, ask the patient to cough. If that does not demonstrate the hernia, lift the cord and dissect it out carefully, using scissors to spread it proximally. Examine it carefully between your finger and thumb. Look for something like the finger of a glove, but made of tissue like amnion. That's the sac! If tissues are very scarred because of a previous hernia repair or frequent temporary irreducibility, dissect out the spermatic vessels and vas: the sac must be in the remaining tissues which you can safely tie off. If you still cannot find it, look for a direct hernia, or a femoral hernia (18.7, 18.8). If you still cannot find a sac, just narrow the internal ring. If the hernia recurs, next time, consider operating under spinal anaesthesia or GA.

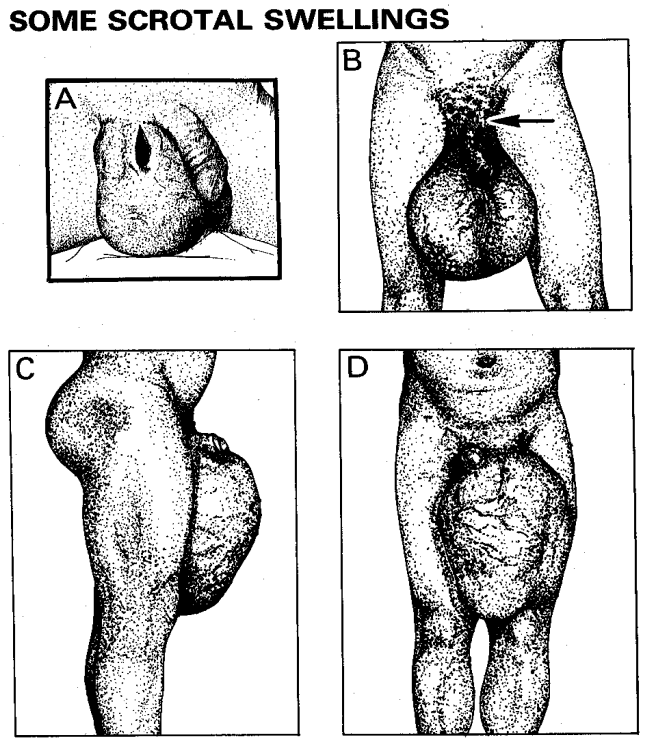


Fig. 18-14 SOME SCROTAL SWELLINGS.
A, if a strangulated hernia presents so late that the scrotum is oedematous, you may be justified in puncturing the mass to form a faecal fistula, as a temporary measure, as has been done here. B, bilateral giant hydrocoeles. If, with a finger and thumb, you can get above a scrotal swelling, as you can here, as shown by the arrow, it cannot be a hernia. *N.B. Transillumination does not work well with dark-skinned patients, and needs are a dark room and strong light source!* C,D, giant indirect inguinoscrotal hernia. Repair will be more secure and recurrence less likely if the patient will allow you to divide the cord.
A-C, after Wosornu L. External hernia – inguinal hernia. Tropical Doctor 1973;3(4):165-170. D, after Bowesman C, Surgery and Pathology in the Tropics, Livingstone, 1960 with kind permission.

If the sac tears at its neck, so that there is no longer anything to twist and tie off, try to free the peritoneum

from the abdomen to close the defect that exists. *If you don't do this, a recurrent hernia is inevitable.*

If the sac is large and reaches the scrotum, dissecting it out distally will be difficult. Dissect it out proximally as usual, and clamp and transfix its proximal end. Divide it and leave its distal end open. If you close its distal end, a hydrocoele may form. You may occasionally have to divide the cord (18.4) in an elderly man to obtain a secure repair. If the defect is very large, use a mesh to repair it as above.

If you have mistakenly passed your needle through a major vessel, continue and tie it tight. If bleeding continues and you think you have gone through the FEMORAL VEIN, remove the needle and press the bleeding area for 5mins until the puncture seals itself. If it does not, you have a major problem. Open the inguinal area about 2cm distal to the inguinal ligament and try to clamp the vein on both sides of the hole. If bleeding is controlled, try to close the hole with fine 4/0 or 5/0 monofilament set 1mm apart; otherwise use an aneurysm needle to encircle it with '0' silk, and tie it off. The leg will be oedematous post-operatively, but this is usually only temporary. *Don't tie the saphenous or profunda femoris veins*, because these will not control bleeding from the femoral vein.

If you think that you have injured the bladder, repair its mucosa, and its muscle with absorbable suture. Tuck it back and continue with the repair. Drain the wound and leave in an indwelling catheter for 2wks.

If you find an inflamed appendix in the hernial sac (Amyand's hernia), excise the appendix (14.1). Close the wound by delayed primary closure (11.8), because of the high risk of infection, and use prophylactic antibiotics as usual.

If the hernial sac contains pus or blood, which has drained from the peritoneal cavity, perform a laparotomy (11.2).

If a piece of bowel has a white ring on it, a (Garré) stricture may develop later at the site of the ring if you return it to the abdomen. So, if you can, resect this segment of bowel.

If a loop of bowel escapes into the peritoneal cavity, and you are not sure if it is viable or not, make a midline incision and examine it. This will be much safer than leaving it.

If you find a Meckel's diverticulum in the hernia sac (Littré's hernia), amputate the diverticulum and close the bowel with absorbable suture.

If the ovary and Fallopian tube appear in the sac (rare), untwist them. If they are viable, return them. If they are gangrenous, tie their pedicle and excise them.

(c) Post-operative difficulties with inguinal hernias

If a haematoma forms, you probably failed to tie the superficial vessels adequately, or used blunt dissection forcefully. Next time, prevent this by delicate technique and carefully controlling bleeding at every stage. Release blood from the haematoma by removing skin sutures, and ease the wound open with sinus forceps.

If the scrotum swells (common), you can reassure the patient that the swelling will probably only be transient, provided you have not tied off the lower end of the sac of an inguinoscrotal hernia. Swelling often follows the repair of such a hernia, and may be due to venous obstruction.

If the testicle swells (not uncommon after a difficult hernia repair), this is usually due to thrombosis of the spermatic veins. This usually settles and leaves a normal testis. Alternatively, it arises from infection relayed through the vas or bloodstream. It is then worth using an antibiotic.

If the testis atrophies, you have probably interrupted the circulation in the spermatic artery by handling it roughly, or strangulated it with sutures at the internal ring. *You will not be able to revive the testis*, so remember the problem when operating the next time.

If urinary retention develops, insert a urinary catheter; remove the catheter when the patient is eating, walking, and pain-free.

If deep infection in the inguinal canal persists, it may not resolve until the non-absorbable suture is removed: swallow your pride, re-open the wound and take out the darn, trying not to disrupt the natural fibrosis around it, and leave the wound open to granulate.

If you have used a mesh and infection persists, you may have to remove this to allow the infection to settle. This will prove to be a messy and difficult operation, so have some blood ready and take your time. The result will be a severe weakness in the inguinal area, which will need another mesh to repair it much later!

If a faecal fistula results, you have injured the bowel. This is a serious problem but if there is no obstruction, wait; it will probably close (11.15). Otherwise open the abdomen (11.2).

If symptoms of intestinal obstruction persist or peritonitis develops after you have reduced a hernia, you may have reduced it 'en masse': the hernial sac has slipped back into the abdomen with its constriction ring, so that the hernia is not properly reduced. This is unusual, and occurs with a hernia previously already strangulating bowel (18.6).

Perform a laparotomy. Isolate the loop of bowel trapped in the constriction ring with packs, and resect the affected segment of bowel (11-7). Repair the internal ring with 2 monofilament sutures from inside the abdomen.

If groin pain radiating to the scrotum persists months after the operation, you may have caught the ilio-inguinal, ilio-hypogastric or branches of the genitofemoral nerves (18-7C) in the repair.

Typical neuropathic pain is stabbing, shooting, 'electric', or described as 'pins and needles'; tapping the course of the nerve exacerbates the pain, and a specific LA block of the nerve implicated removes the pain temporarily. If these criteria exist, re-open the wound, try to find the culprit nerve and free it, burying it within muscle (or excise it if it is hopelessly caught in scar tissue).

CAREFUL TECHNIQUE WILL REDUCE THE RISK OF RECURRENCE

18.4 Giant inguinal hernia

It used to be said that there were two kinds of inguinal hernias in the tropics: those above the knee and those below it! This section deals mainly with those below it, which may have been present for as long as 50yrs! They are now, however, not so common, but giant hernias even above the knee still pose a challenge. They may contain large intestine, stomach or so much of the abdominal content that reduction back into the abdominal cavity may cause excessive pressure on the diaphragm and subsequent respiratory distress.

Differentiate a giant hernia from a hydrocoele (18-14) *or an adenolymphocoele* (18-5).

If a patient has a very large indirect inguinal hernia, or a recurrent direct one, the posterior wall of the inguinal canal will be very weak and its anatomy deformed. It will be difficult to repair, and much more likely to recur. Repair will be more secure if you can divide and transfix the spermatic cord just below the internal inguinal ring, so that you can close it and reinforce the posterior wall of the inguinal canal more securely.

You must increase the abdominal volume pre-operatively to accommodate all the contents of the hernia: this you can do by progressively injecting air into the peritoneal cavity.

PREPARATION
Counsel the patient about probable loss of the testis on that side, and obtain consent for ligation of the spermatic cord. You can expand the abdomen over 2 weeks by introducing 500ml air every alternate day progressively into the peritoneal cavity through the *linea semilunaris* (18-3) using a long cannula, a two-way tap, and a 50ml syringe. This will allow easier return of abdominal contents. Wait 2wks more before surgery.

METHOD
Start perioperative antibiotic prophylaxis. Use GA. Proceed as for a simple hernia repair (18.2); the posterior wall of the inguinal canal will be weak, so a mesh repair (18-13D) will be necessary. If you have permission, remove the testis, and tie and divide the cord at the internal inguinal ring. If you are obliged to retain the testis, but you can divide the cord, tie, transfix, and divide it as near the internal ring as you can. Leave its distal part untouched, so that you don't disturb the collateral vessels. Be gentle, or you will damage them, and the testis will atrophy.

You will probably need to excise some of the stretched scrotum; with the testicle pulled upwards, mark a suitable site on the scrotal wall and amputate its distal part. There will be considerable bleeding, so go slowly, securing haemostasis as you go along, and then close the scrotal wound in 2 layers with a continuous absorbable suture. Leave a drain in place.

One way of reducing the risk of scrotal haematoma is to use tape to secure the empty floppy scrotal sac for 48h to the anterior abdominal wall with 2 pieces of gauze between.

18.5 Inguinal hernia and congenital hydrocoele in infants and children

A baby's *processus vaginalis* (18-15) is usually open at birth, and closes <2yrs. If it is not completely obliterated, it can leave a number of abnormalities (18-15). Note that a congenital hydrocoele is simply an indirect hernia containing fluid. *Never try to aspirate such a hydrocoele!* When you see such, the hernia may be present, or it may have reduced itself, so you have to depend on the mother's history that there is a lump which comes and goes, and gets larger when the child cries. If you want to see it, find some way to make him cry or laugh!

Inguinal hernias in a child are always indirect. Unlike umbilical hernias, they don't become smaller spontaneously with age. Inguinal hernias seldom strangulate in childhood because the neck of the canal is fairly wide and the canal is so short, but they often become obstructed, especially in the 1st year. However, in 30% of premature babies these hernias *will* strangulate and these babies are most easily missed. Herniotomy is one of the most common operations in children, but it is not always easy. The sac is thin, delicate, and difficult to find, and you can easily injure a baby's vas.

At birth the inguinal canal is short, and if the hernia is large, the external ring may lie directly over the internal ring, which is convenient, because it allows you to dissect out the sac without opening the *external oblique* aponeurosis. In young children, simply open the sac (herniotomy), and tie it off. *Herniorrhaphy is not needed.*

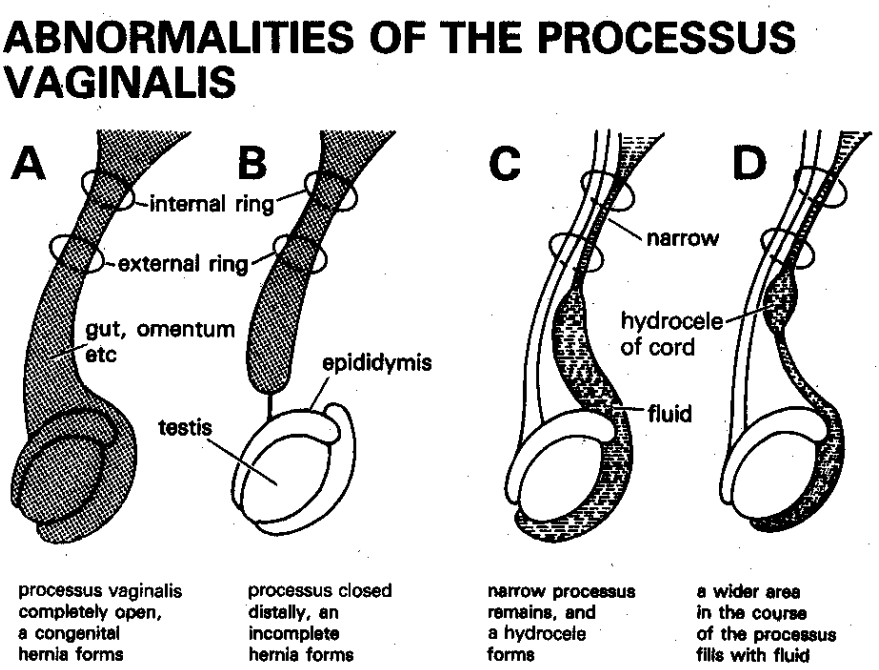


Fig. 18-15 ABNORMALITIES OF THE *PROCESSUS VAGINALIS*. A, when it remains completely open, a complete inguinal hernia forms. B, if it closes distally, and leaves the *tunica vaginalis* covering the testis, an incomplete inguinal hernia forms. C, when the *processus vaginalis* becomes narrow, but does not disappear, fluid passes down it from the peritoneal cavity and forms a hydrocoele around the testis. D, if there is a wider area in the course of the *processus vaginalis*, it may form a hydrocoele of the cord. Operate on a congenital hernia and on a congenital hydrocoele: tie and divide the *processus vaginalis*.
After McNeill Love WJ. Bailey & Love's Principles & Practice of Surgery, HK Lewis 15th ed 1975.

Wait to operate until a baby is 6 or preferably 9 months old, when anaesthesia and surgery will be easier. *Never apply a truss.*
The operation involves dissecting out the sac carefully (making sure that you don't damage the spermatic vessels or the vas), transfixing its neck at the external ring, and excising it.
There is no need to open the inguinal canal of a young child. You may occasionally need to narrow the internal ring of an older child.

You may find a sliding hernia of the bladder or colon, or of a girl's adnexae. Hernias are often bilateral in children: examine the other side carefully therefore to avoid the child having to return later for another operation. Always also look for maldescended or absent testes (27.27).

CAUTION! If you find bilateral inguinal hernias in girls, look for signs of ambiguous genitalia or intersex state.

(a) Manipulation for child inguinal hernias

If a child's hernia is irreducible, sedate him, and put him into gallows (overhead skin) traction if he is <3yrs. There is a 50% chance that it will reduce spontaneously, or with a little *gentle* manipulation.

Don't induce pain, or use any force! If this succeeds, operate as soon as it is convenient. If it fails, operate without delay.

(b) Operative intervention

(1) HERNIOTOMY in young children (GRADE 2.3)

ANAESTHESIA. Ketamine or GA. *Don't use LA.*

INCISION
Make a 3cm incision in the *skin crease* above the inguinal ligament, more medially and superiorly than you would for an adult. Cut through the subcutaneous tissues, and pick up and tie the small superficial epigastric and external pudendal vessels with haemostats. Separate fat and superficial tissues by the 'push and spread' technique (4-8). *Don't split the external oblique aponeurosis*: this is unnecessary.

To find the hernial sac, which should be anterior to the cord, push with your little finger in the scrotum through the external ring. Gently separate tissues off the cord which appears as a distinctly blue structure and thereby free it. You should then be able to get behind it with a finger and so hold it between the thumb and index finger of your left hand. Dissect the sac very carefully away with gauze or scissors from the spermatic vessels and vas, which is a thin white strand. *Beware: it is all too easily damaged because it is so fine!*

Free the sac to the internal ring taking care not to tear it. Unless it is obviously empty, or just has hydrocoele fluid in it, open it between haemostats, as if you were opening the peritoneum for an abdominal operation, and reduce the contents unless they need resection.
CAUTION!
(i) Free the sac completely before you open it. In this way you are less likely to split it.
(ii) A girl's Fallopian tube and ovary may slide into the sac: *don't remove them as part of it*.
(iii) If you find the appendix in the sac, *don't remove it*.
(iv) A boy's vas is very small, *don't mistake it for a piece of fibrous tissue*.

Otherwise, divide the sac and transfix its neck with 3/0 absorbable suture, and tie it off.
CAUTION!
(i) Make sure you are cutting the sac only. You can easily cut the vas, because it is adherent to the posterior surface of the sac.
(ii) *Don't split the sac lining.* If you do so accidentally, apply fine haemostats to the parts of the sac that are free and try to separate the sac off its underlying structures; this is difficult as it tends to tear further.

If you have pulled the testis out of the scrotum, be sure to return it properly, or else it may adhere in the groin; there will be some scrotal swelling afterwards, but reassure the parent this is *not a recurrence*.

(2) INGUINAL HERNIAS IN OLDER CHILDREN

In a child >14yrs, assess the size of the internal ring. The internal ring of an older child is no longer under the external ring. It will have started to migrate laterally. Try to put your finger though it into the peritoneal cavity. If it is big enough to let you insert your index finger (the internal ring is >1½cm wide), it probably needs herniorrhaphy.

Retract the cord laterally. Put the tip of your finger through the hole in which the stump of the sac has retracted. Feel the margins of the hole, put a haemostat on its medial margin, and lift it forwards. Bring the fine upper and lower edges together with 3/0 absorbable sutures, so as to wrap the *transversalis fascia* snug round the cord. You should still be able to pass the tip of your forceps through the ring alongside the cord. You may have to open the inguinal canal for about 5cm to get access to the internal ring: cut upwards and laterally from the external ring in the direction of the fibres of the *external oblique* aponeurosis as in an adult hernia operation.

(c) Difficulties with child inguinal hernias

If there is a hernia and a hydrocoele that are separate, proceed as above, and open the *tunica* distally by pushing up on the hydrocoele: the patent *processus* is probably just too thin to allow the hydrocoele to drain (18-15C). Leave the *tunica* open distally to do so.

If the sac splits up to and perhaps through the inguinal ring, this is inconvenient. *Be especially careful*, as you search for something to sew together, *that you don't tie the vas.* If the hernia recurs, you will probably have to close the defect internally *via* a laparotomy.

If the hernia recurs, re-operation can be difficult: see above.

If there is a maldescended testis together with the hernia, separating the sac may be more difficult. Isolate the vas and its artery, the testicular artery, and pampiniform plexus to mobilize the testis to get it down to the scrotum (27.27).

If there is a strangulated hernia as a neonate, the sac will be very friable and will not take sutures; be content to close the internal ring by approximating the internal and external oblique muscles.

If the testis atrophies later, you have probably interfered with its blood supply. This is one of the commonest complications. The parents, who may have difficulty accepting that one testis can function as efficiently as two, will not be pleased. The other testis should however be normal, so reassure them.

18.6 Irreducible and strangulated inguinal hernia

You can relieve a strangulated inguinal hernia and resect bowel through the ordinary incision for an inguinal hernia. Unlike with a femoral hernia, there is usually no need to open the abdomen through a separate incision to get better access.

(a) Differential diagnosis

Suggesting torsion or inflammation of an inguinal testis: absence of the testis from the scrotum. A retained testis is often associated with an interstitial hernia (into the anterior abdominal wall).

Suggesting inflamed inguinal nodes: the swelling is more diffuse, there is sometimes redness and oedema of the overlying tissues. Vomiting and abdominal pain are minimal or absent.

(b) Taxis (manual reduction) for irreducible inguinal hernias

INDICATIONS

An inguinal hernia which has only been irreducible for a short time, *and is not very tender to touch*.

CONTRAINDICATIONS

Any hernia which is tender to touch.

METHOD

Use morphine and put the patient in a steep Trendelenburg position. Wait for at least ½-1h. Often, a hernia reduces spontaneously. If it does not, use gentle manipulation, but *never* force. For children <3yrs, use gallows traction (18.5).

CAUTION!

Don't apply pressure which may rupture bowel, or risk reducing the hernia contents 'en masse'. Watch carefully for signs that any non-viable tissue has been reduced. This is unlikely to have happened, and if it has, the tissue is more likely to be omentum than bowel. If you are in any doubt, restrict oral intake, insert a nasogastric tube, administer IV fluids and observe for abdominal tenderness: if this develops, perform a laparotomy.

(c) Operation for irreducible or strangulated inguinal hernias

PREPARATION

Use perioperative gentamicin and metronidazole. Get consent for a laparotomy and possible bowel resection. Prepare all the skin of the abdomen and genitalia. Insert a nasogastric tube to drain the stomach well. Use analgesic sedation and GA.

BISTOURY, guarded. This is a curved probe with a cutting edge on its concave surface near the tip. It is the safest instrument for enlarging a constricting hernia ring.

INCISION. Proceed as before (18.2) to open the inguinal canal.

(1) OPEN THE SAC.
You will see a tense mass emerging from the internal ring and passing towards the scrotum. If oedema and congestion make identifying the overlying structures difficult, use blunt-tipped scissors and the 'push and spread technique' (4-9B) to incise the first 2 layers: the external spermatic fascia and the *cremaster* muscle. If they dissect off easily, good; if they don't, leave them, except for a small area near the fundus. Incise this between a pair of fine haemostats, just as you would if you were opening the peritoneum for a laparotomy.

CAUTION! Surround the operation site with large swabs to prevent the soiling of the wound by the septic contents of the hernial sac, which is likely to contain virulent aerobic and anaerobic organisms.

Pick up layer by layer in forceps, and carefully incise each layer till you reach the peritoneum when fluid will run out, and you will see bowel or omentum. Apply several haemostats to the peritoneal margins to prevent them retracting into the abdomen. Attach a Babcock forceps to the bowel or omentum to prevent them slipping inside the abdomen.

(2) RELEASE THE CONSTRICTION RING.
Feel for the constriction with your finger. If you can insert an instrument through it and nick its lateral margin, do so. If not, retract its upper edge with a retractor, and cut down on it from outside. Alternatively, push your little finger into the ring. While your assistant holds the contents of the sac out of the way, push a large haemostat into the ring lateral to the neck and spread it open. Divide the lateral side of the ring with scissors or a bistoury.

Gently deliver the contents of the sac. If it extends to the scrotum, it may be easier to deliver the testis also. The bowel or omentum may be blue, purple, or black.

CAUTION!
(i) *Don't damage the spermatic cord as you open the sac*.
(ii) *Don't incise the medial side of the internal ring*, or you may cut the inferior epigastric artery. *Don't cut the bowel!*

Examine the contents of the hernia. If the bowel has been trapped, withdraw a few centimetres of the afferent and efferent loops. Assess its viability (11-6).

If viable bowel or omentum is present, replace them into the abdomen by gently manipulating the bowel through the widened neck of the hernia. Be patient and manipulate small segments at a time. Make sure the patient is well-relaxed.

If bowel is non-viable, **or dubiously viable**, resect it. Gently pull out adequate lengths of bowel on either side of the gangrenous portion. If this is difficult, widen the neck of the sac further; if there is still not enough length, perform a laparotomy *via* a midline incision.
When the bowel anastomosis is complete, return it into the abdomen with extreme care, *so as not to disrupt the anastomosis*.

If omentum is strangulated, pass long haemostats across the healthy part, cut off the gangrenous part distal to them, transfix the healthy omentum with a needle, and then tie it off. You may need more than one haemostat and transfixion suture.

CAUTION! Be sure to control all bleeding before you return anything to the peritoneal cavity.

(3) CLOSE THE SAC as before (18.2).
(4) NARROW THE INTERNAL RING, because you have deliberately widened it!
(5) REPAIR the defect with a darn unless there has been gross sepsis, in which case it might be best to advise a formal repair later.
(6) *DON'T CLOSE THE WOUND if there has been gross sepsis*: leave it open and use delayed primary closure (11.8). If there was obvious perforation, drain the canal through the scrotum.

(d) Difficulties with irreducible or strangulated inguinal hernias

If presentation is very late with oedema, cellulitis or abscess formation on the abdominal wall or scrotum, overlying the gangrenous contents of a strangulated inguinal hernia, a faecal fistula is about to form. Expect this beyond the 4th day of strangulation. It can form:
(1) in the inguinal region, where the prognosis is better, especially if the hernia is of the Richter type (18-2B) and the bowel obstruction is incomplete.
(2) in the scrotum, where the prognosis is worse.

In this situation, start IV gentamicin and metronidazole, and infuse 2L IV Ringer's lactate. Start nasogastric suction.

Open the groin, and identify the strangulated loop of bowel; doubly ligate both ends tightly with #2 silk as close as you can to the hernia neck. Pack off the area of the internal inguinal ring, remove the gangrenous bowel between the ligatures.

Curette the fistula track, taking care not to damage any local structures. Wash the groin wound thoroughly and leave it open applying betadine dressings bd. Exteriorize the bowel ends as ileostomies. *Don't attempt any hernia repair at this stage.* Continue resuscitation till you are sure the patient is passing good volumes of urine, and then plan bowel anastomosis as below.

If there is an established small bowel fistula, following a strangulated hernia weeks or months ago, *don't attempt local repair*. Make sure the patient is well re-hydrated and his potassium deficit is corrected. Then perform a laparotomy: make a midline incision and apply non-crushing clamps on each loop (proximal and distal) of bowel where they enter the hernia orifice internally. Keep the clean laparotomy wound clear of the groin and explore this as above if you haven't already done so.

Now re-scrub and go back to the laparotomy. Withdraw the viable part of bowel out of the abdomen.

Make an end-to-end anastomosis (11-7). Close the hernia defect internally with a #1 nylon purse-string suture. Wash out the abdomen thoroughly and close it.

If, in a Busoga hernia (18-10,11)**, you cannot bring bowel through the narrow opening in the conjoint tendon**, extend the incision in the *external oblique* a little more laterally, and then split the *internal oblique* and *transversus* muscles about 5cm above the internal ring level with the iliac spine, as in the muscle splitting approach for an appendicectomy (14-1C). Open the peritoneal cavity, withdraw the bowel, and if necessary, invaginate or resect it. This approach is useful in a strangulated Busoga hernia and avoids enlarging the opening in the conjoint tendon and weakening it.

If, in a Busoga hernia (18-10,11)**, you find the sac necrotic, but no bowel in it**, there is probably no need to open the abdomen and examine the bowel. If it has slipped back, it is unlikely to be seriously non-viable. Debride dead tissues. Post-operatively, continue careful observation for signs of peritoneal irritation and general deterioration.

If you cannot return the bowel to the abdominal cavity, tilt the table head downwards, make sure the patient is well-relaxed with a nasogastric tube *in situ*, put a retractor under the anterior lip of the wound to raise it. Then with extreme care, return the bowel to the abdomen, a little at a time, starting at one end and gently squeezing it between your finger and thumb. If this is absolutely futile, try the La Roque procedure: make a 2nd muscle splitting incision in the *external oblique* and enter the peritoneum laterally, and then pull the bowel down gently from inside.

If you find a gangrenous testis on opening the sac (especially in children), perform an orchidectomy (27.26) and make sure the other testis is present in the scrotum. If not, perform an orchidopexy (27.27) on that side.

18.7 Femoral hernia

A femoral hernia is more likely to strangulate than an inguinal, but is much less common. It is rare where people, especially children, walk barefoot because the resulting enlarged femoral nodes close the defect. Whereas inguinal hernias are almost entirely a male disease, the sex incidence of femoral hernias is more nearly equal, with femoral hernias only marginally more common in men than in women in most communities.

A patient with a femoral hernia complains of a painful, tense, slightly tender, spherical mass below the inguinal ligament, 2cm infero-lateral to the pubic tubercle. *Usually, you cannot reduce it.* If you can, you may be able to pass your finger upwards through the dilated femoral canal.

There is usually no cough impulse. Sometimes, a femoral hernia turns upwards, and may come to lie over the inguinal ligament, where you can mistake it for an inguinal hernia, or it can turn outwards or downwards.

Repair is not difficult, and recurrence is rare. So operate; *a truss cannot control a femoral hernia*. The low approach to a femoral hernia is described here, and is satisfactory unless you need to resect bowel.

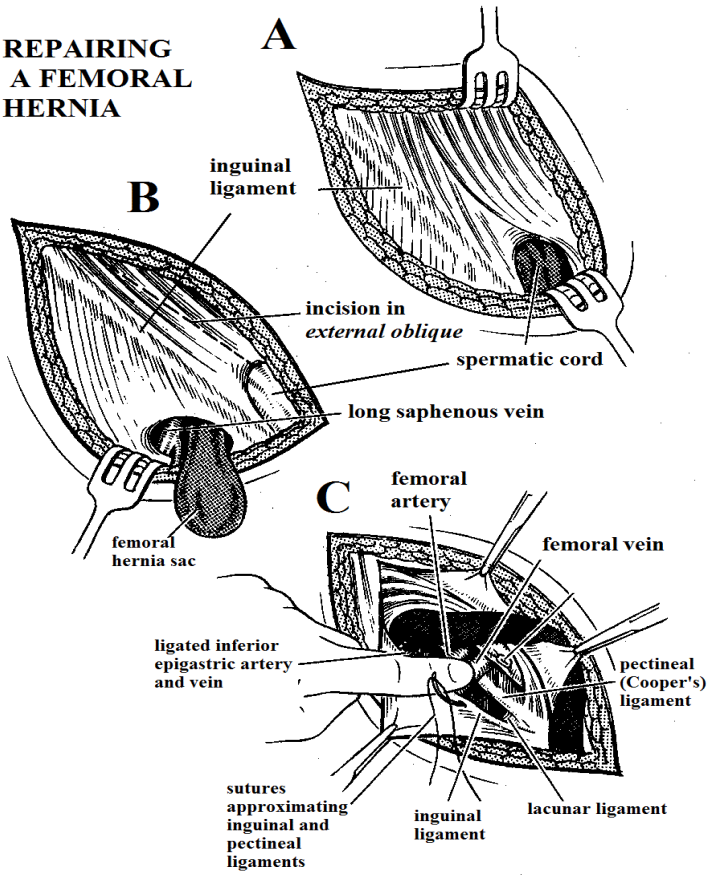


Fig. 18-16 REPAIRING A FEMORAL HERNIA.
A, expose the external inguinal ring. B, retract the lower flap and mobilize the sac. C, suture the inguinal ligament to the pectineal (Cooper's) **ligament.**

ANATOMY. A femoral hernia comes through the femoral canal. This is about 2cm long and is filled with fat and a lymph node (Cloquet's). *Anteriorly*, it is bounded by the inguinal ligament, and *posteriorly* by the pectineal (Cooper's) ligament, which is a thickened part of the pectineal fascia, and overlies the pectineal ridge of the pubic bone. *Laterally* lies the femoral vein, and *medially* lies the sharp edge of the lacunar ligament.

A femoral hernia extends forwards through the *fossa ovalis* where the long saphenous vein joins the femoral vein.

Other, rarer femoral hernias can emerge within the femoral sheath but anterior to vein and artery (Velpeau's hernia), lateral to the femoral vessels (Hasselbach's hernia), or posterior to the femoral vessels (Serafini's hernia). Narath's hernia is posterior to the vessels and only visible when the hip is congenitally dislocated. In de Garengeot's hernia, the appendix is within a femoral hernia.

Other rare hernias in this area come through the lacunar ligament (Laugier's hernia), the pectineal fascia (Cloquet or Callisen's hernia), and the saphenous opening (Béclard's hernia).

(a) Differential diagnosis

Suggesting enlarged lymph nodes: look for a septic focus on the leg, the lower abdomen or the buttock or evidence of tuberculosis elsewhere. An enlarged deep inguinal lymph node may be almost impossible to distinguish from a femoral hernia, except for signs of intestinal obstruction.

Suggesting a varix of the long saphenous vein: a soft, easily compressible swelling (unless it is thrombosed), which fills up again when you release the pressure.

Suggesting a psoas abscess: a much larger fluctuant swelling associated with spinal TB or HIV disease.

(b) Operation for non-strangulated femoral hernia (GRADE 2.3)

ANAESTHESIA
(1) LA, especially if the general condition is poor. Use the same method as for an inguinal hernia. Infiltrate a wide subcutaneous area, and infiltrate the neck of the sac as you dissect deeper.
(2) Subarachnoid or epidural anaesthesia.
(3) GA with relaxants.

INCISION
Make a 6cm incision directly over the hernia below the groin crease. Deepen the wound through the subcutaneous tissue to expose the sac (18-16A). Tie the tributaries of the long saphenous vein. Use blunt dissection to mobilize the sac free from the tissues around it (18-16B). Trace it to its neck, where it disappears into the femoral canal.

Carefully incise the fundus of the sac. Cut through fat until you find the much smaller peritoneal sac. Expect to cut through many layers. Inspect its contents. This will usually be omentum, except in longstanding hernias. Reduce the contents completely, and divide any adhesions.

When the sac and its contents are cleanly exposed, and you are quite sure that you have completely reduced its contents, twist it.

Transfix its neck proximally with thread as high up as you can, and excise the protruding sac, leaving a generous neck distal to the transfixing suture. The stump will disappear up into the femoral canal. Then insert a few monofilament sutures, so as to approximate the inguinal ligament to the thickened part of the pectineal fascia, on the floor of the femoral canal. This is the pectineal (Cooper's) ligament (18-16C). Protect the femoral vein laterally with your finger, while you are inserting these sutures. Close the wound in layers.

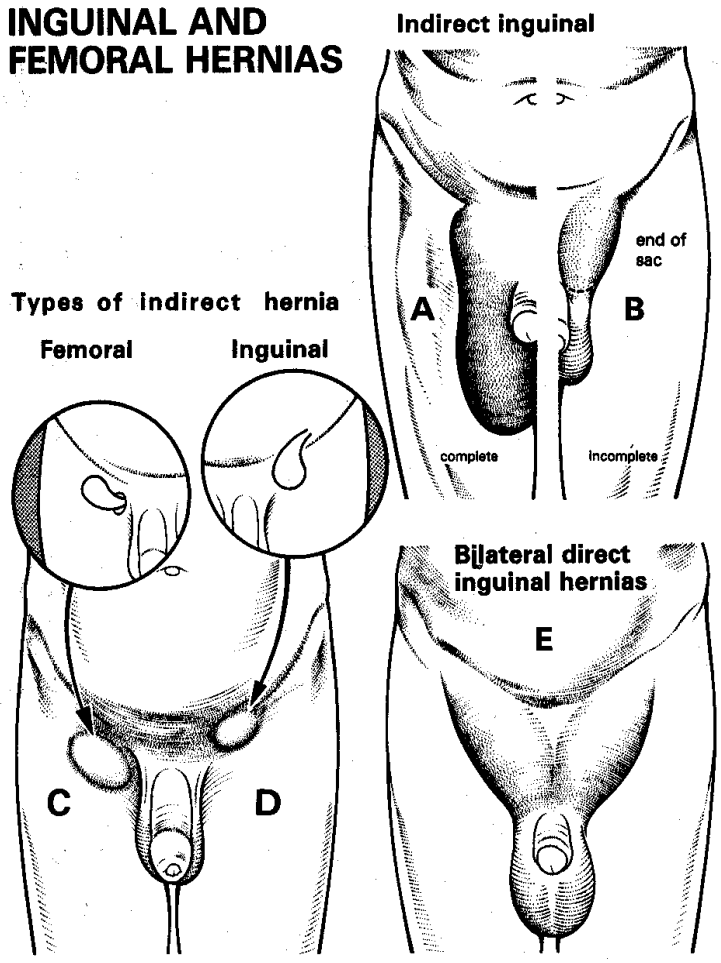


Fig. 18-17 INGUINAL AND FEMORAL HERNIAS.
A, complete indirect inguinal hernia reaching right down into the patient's scrotum. B, incomplete indirect hernia in which the *processus vaginalis* ends just above the testis. C, femoral hernia, showing how the sac extends upwards. D, small indirect inguinal hernia showing the sac extending downwards. E, bilateral direct inguinal hernias.
After McNeill Love WJ. Bailey & Love's Principles & Practice of Surgery, HK Lewis 15th ed 1975

(c) Difficulties with a femoral hernia

If you cannot get good bites of the pectineal ligament as it lies on the pectineal fascia, get a short curved needle, or a fish-hook needle, and set it in a needle holder in such a way that it points back at you. Insert this into the femoral canal, and try to hook the ligament on your way out.

If you injure the femoral vein, press on the bleeding point, arrange suction and obtain vascular clamps (18.3).

If you cannot return the contents of the sac easily, pass your finger gently outside it and dilate the femoral ring. If this fails, stretch the ring by putting a haemostat into it and opening it in an inferio-superior direction. Or, carefully enlarge the superomedial side of the femoral canal, but be careful of an abnormal obturator artery (18-18A).

If you find an inflamed or gangrenous appendix in the hernial sac (de Garengeot's hernia), excise the appendix (14.1). Close the wound by delayed primary closure (11.8), because of the high risk of infection.

If there is arterial bleeding as you enlarge the femoral canal, you have injured an abnormal obturator artery, which arises in about 25% as a pubic branch of the inferior epigastric artery.

This abnormal obturator artery may occasionally pass over the internal aspect of the femoral canal, or run in the edge of the lacunar ligament, where you can easily cut it (18-18A).

If so, open up the inguinal (*NOT femoral)* canal, open up its posterior wall between the inguinal ligament inferiorly and the conjoint tendon superiorly. This will expose the peritoneum. Push this up and you will find the abnormal obturator artery crossing the internal aspect of the femoral canal. Grasp it with a haemostat and tie it.

If you suspect strangulation, extract the bowel carefully from the femoral canal and examine it. If, after covering it with warm packs, it does not recover, it needs resecting (18.8).

18.8 Strangulated femoral hernia

A strangulated femoral hernia is more often misdiagnosed than a strangulated inguinal hernia:
(1) It may be small, and lost in the thick fat of the groin.
(2) Only the circumference of the bowel may be caught (Richter's hernia), so that you can hardly feel anything in the thigh.
(3) When it is large, it may have a rounded fundus and a narrow neck, which allows the fundal part to move painlessly, so you may think there is no strangulation.

This makes it very important to explore any doubtful lump in the femoral region when a patient has abdominal symptoms and especially intestinal obstruction, especially if femoral hernias are not uncommon in your area.

There are 2 approaches to a strangulated femoral hernia, with some debate as to which is better:
(1) the standard approach, which requires two incisions, one over the hernia and another in the lower abdomen, and
(2) the Lotheissen approach through a single incision in the posterior wall of the inguinal canal: this is more difficult.

In the standard approach, cut down just above the patient's inguinal ligament and aim to:
(a) Expose and isolate the sac.
(b) Open and inspect its contents.
(c) If the bowel is not viable, open the abdomen through a lower midline incision. Expose and, if necessary, enlarge the femoral ring from above.
(d) Amputate the bowel in between pairs of clamps.
(e) Remove non-viable bowel from the hernia sac.
(f) Excise the sac and leave the wound open.

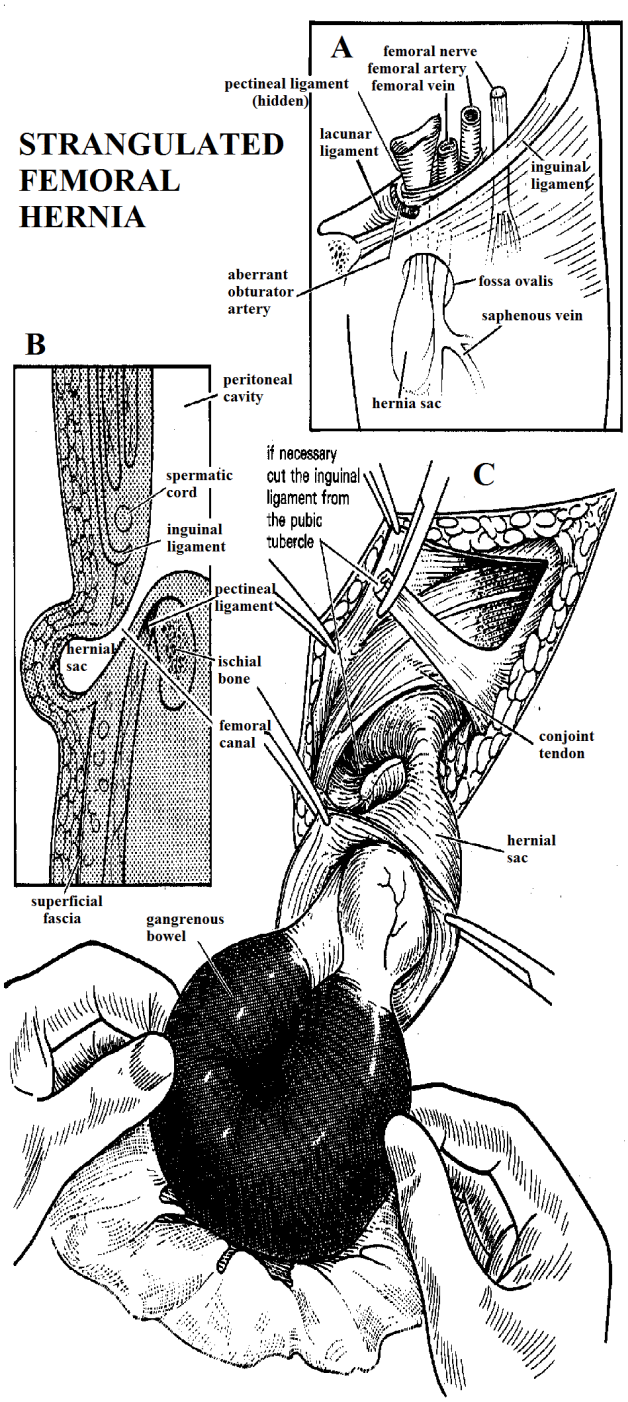


Fig. 18-18 STRANGULATED FEMORAL HERNIA.
A, anatomy of the femoral canal. *N.B.* The femoral vein lies laterally and the lacunar ligament (reflected part of the inguinal ligament) lies medially to the sac. An abnormal obturator artery may run in the edge of this ligament. B, side view of the femoral canal showing how a femoral hernia forms. C, opening a strangulated femoral hernia proximally. (Most femoral hernias are smaller than this. It is rarely necessary to divide the inguinal ligament.)
C, adapted from a drawing by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

IF THERE ARE ABDOMINAL SYMPTOMS, EXPLORE ANY TENDER FEMORAL LUMP

(a) Operation for strangulated femoral hernia
(GRADE 3.3)

ANAESTHESIA
(1) Ketamine and LA: infiltrate the field widely. Inject more solution into the deeper tissues as you get to them.
(2) Spinal or epidural anaesthesia if the patient is fairly fit.
(3) GA and tracheal intubation with relaxants.

PREPARATION
Pass a catheter and empty the bladder. Make sure the patient is well-hydrated. Use perioperative IV gentamicin and metronidazole.

INCISION (STANDARD APPROACH)
Make a transverse incision in the skin crease over the hernia itself. Divide the covering layers, including the deep fascia, and dissect them off the sac. Sweep your finger round the hernia to mobilize it, and define its neck. Clean it by dissection with your finger, and a swab and not-too-sharp-nosed scissors.

OPEN THE SAC by inserting retractors and packing off the sac while you carefully cut down on it. Like an onion, it will have more layers than you expect. As soon as you are inside it, there will be a warning spurt of turbid blood-stained fluid. The bowel is often gangrenous.

RELEASE THE STRANGULATION by holding the bowel in a swab between the finger and thumb of one hand. Meanwhile, try to widen the femoral canal by inserting the very tip of your finger into the hernia, just outside the sac itself. With your finger inside the femoral canal, move it around the neck of the sac and try to free it.

CAUTION! Don't let go of the bowel at this point, because if you lose it, you have to perform a laparotomy to retrieve it.

Now draw the bowel down out of the sac a bit more. If it does not quite come, repeat the dilating manoeuvre, but this time with your finger inside the sac, between it and the bowel.

If you still cannot deliver the bowel into the wound, clear the neck of fatty tissue. Enlarge the ring on its medial side by dividing the lacunar ligament, and the fibrous tissue in front of the ring.
Protect the contents of the sac while you divide the ligament by passing a grooved director up the medial side of its neck. Then carefully cut down on the director with one or two nicks of a scalpel. *Watch out for an abnormal obturator artery.*
With the bowel drawn down into the sac, wrap it in a warm wet swab, to see if it is viable (11-6).

If the bowel is viable, let it slip back into the abdominal cavity, and repair the hernia from the groin (18.7).

If the bowel is not viable, perform a lower midline (or Pfannenstiel) incision, and resect and anastomose the bowel from inside by laparotomy (18.6).

If protruding omentum looks as if it might not be viable, transfix it, tie it, and excise it.

If an area of necrosis only involves part of the wall of the bowel (Richter's hernia, 18-2B**)**, bury it with invaginating seromuscular absorbable sutures. You need not resect it, *provided it follows the criteria for safe invagination* (18.2).

If you are in any doubt about the viability of the bowel, (including a Richter's hernia), excise the damaged portion and perform an end-to-end anastomosis (11-7).

CAUTION!
(1) Always open the sac and inspect its contents before you return them to the abdomen. They may be gangrenous. If there is a Richter's hernia, *be especially careful not to let it escape back into the abdomen*.
(2) *Take great care not to contaminate the peritoneal cavity*.

REPAIR. If you have opened the abdomen, it may be difficult and dangerous to reduce the hernia contents if they are incarcerated (and especially if they are strangulated). You might need to divide the bowel loops inside, and then remove the remainder outside (18.6).
CAUTION! Take care to free the sac of surrounding tissues before you excise it, or you may pass sutures into a protrusion of the bladder or colon.
Close the femoral canal by passing three interrupted monofilament sutures between the inguinal ligament and the pectineal ligament (18-16C). *Don't go too far laterally with these sutures*, or you may constrict the femoral vein.

(b) Difficulties with strangulated femoral hernias
(see also 18.3,7)

If you cannot dilate up the femoral canal enough to mobilize the strangulated bowel, approach it from above via a laparotomy. Use blunt dissection to expose the neck of the sac medial to the femoral vessels.

If this is not successful, cut the lacunar ligament, which is the medial boundary of the femoral ring, under direct vision. *Be careful*; you may meet an abnormal obturator artery (18-18A)!

If you still cannot dilate up the femoral canal enough, divide the inguinal ligament: this is very rarely necessary. At the end of the operation, suture its free end against the pectineal line, so as to obliterate the femoral canal. The danger with this is that, if the wound becomes infected, a hernia may form later which will be difficult to repair. Whatever you do, remember that the femoral vein lies on the lateral side of the femoral canal!

If you find a strangulated femoral hernia expecting an inguinal hernia, proceed as in the Lotheissen approach:

(c) The alternative Lotheissen approach through the posterior wall of the inguinal canal

Strangulated bowel and omentum may be more easily dealt with by this method than by the 'standard approach'.

Make an incision 1-2cm above the inguinal ligament, as for a strangulated inguinal hernia (18.2, 18.6). Sweep away the superficial fatty tissue from the external oblique in the lower wound flap, until you come to the bulging femoral hernia below the inguinal ligament. Deal with the hernia sac as above.

Open up the inguinal canal as for an inguinal hernia. Hold the cord out of the way, and incise its posterior wall (the conjoint tendon and *transversalis fascia* medially and the *transversalis fascia* only laterally). Make a 2½cm incision 5mm above and parallel to the inguinal ligament. Tie and divide the inferior epigastric artery and vein, which lie deep to the inguinal ligament in the medial border of the internal inguinal ring; then extend the incision laterally to 4cm. Apply haemostats to its upper and lower edges to hold them apart.

Look for the neck of the hernia from above by gauze dissection. You will find a tongue of peritoneum disappearing into the femoral canal. Working from above and below, and using the methods described above, reduce the hernia and the sac. Be careful to clear the sac from the bladder medially. Deal with strangulated bowel or omentum as above.

Transfix, tie, and excise the sac. Use interrupted monofilament to close the femoral canal, by passing sutures between the inguinal ligament and the pectineal ligament. Protect the femoral vein laterally with your finger while you place these sutures. Close the posterior wall of the *external oblique* aponeurosis as for an inguinal hernia.

18.9 Hernia of the umbilicus and anterior abdominal wall

There are several hernias in this region, and you must not confuse them:
(1) The common true umbilical hernias of children, which rarely need surgery.
(2) The much rarer paraumbilical hernias of adults through or beside the umbilicus, which usually need surgery.
(3) Small and usually harmless epigastric hernias of the *linea alba* between the xiphoid and the umbilicus, which often don't need surgery, but which are easy to repair.
(4) Hernias which follow incisions, particularly Caesarean sections, other laparotomies, appendicectomy or kidney operations.
(5) Rare lumbar or Spigelian hernias, which are direct hernias in the flank or 3-4cm above the inguinal ligament through the *linea semilunaris* (18-3).

If there is a large midline bulge in the upper abdomen, which feels muscular, this is a wide separation (divarication) of the *recti* muscles: you should not operate on this. Try to get the patient a corset for support.

Remember that, in both adults as well as children, an umbilical hernia will bulge with coughing or crying, and other causes of abdominal distension. The hernia itself is *not* the cause of the problem, and you should resist attempts of a patient or parent to get you to operate.

18.10 Umbilical hernia in children

In many areas of the world, a child commonly has a defect in the *linea alba* at the umbilicus through which a hernia forms (18-19A). These hernias rarely obstruct or strangulate, usually heal themselves without treatment, and seldom need repair. In areas where they are common, and accepted as being merely a variant of the normal, there will be little demand for surgery. Accept this and *don't operate without good reason*.

If you do have to operate, repair is usually straightforward. The child's umbilical scar is weak and the neck of the sac wide; it has one compartment, and is covered by skin, to which it may be closely adherent. It may contain small bowel, omentum, or large bowel, and rarely strangulates. *Strapping such a hernia in a child is useless.*

A large defect at birth (omphalocoele or exomphalos) requires a different approach (33.4).

(a) Management
If a child is born with a small hernia, reassure his mother that it will become a little larger up to 3-5yrs; 90% will close spontaneously by 3yrs and 95% by 5yrs unless the defect is >2cm diameter.

If the parents blame a hernia for recurrent bouts of periumbilical pain, make sure that this is not due to hookworms or sickle-cell crises, or even a cough! The hernia will always bulge when the child cries, but it is usually not the cause!

An irreducible hernia will however often reduce under sedation or ketamine with *gentle* taxis (18.6).

(b) Operative treatment
UMBILICAL HERNIA REPAIR (GRADE 2.3) for an *uncomplicated* hernia is indicated only if a child has reached 6yrs, the hernia is more than 5cm across at its neck (rare), or he has previously had an incarcerated hernia reduced. Otherwise it is indicated for the irreducible or rare strangulated hernia, which may have inspissated thick hard faeces or undigested seeds caught in the herniated loop of bowel.

INCISION
Preserve the umbilicus; you may have to excise it only if the hernia is large. Make a curved transverse incision, above or below the umbilicus (18-19A).

Dissect down to the anterior rectus sheath and around the umbilicus, so as to reflect a lower or an upper flap to include it. If dissecting the fundus of the sac free from the umbilicus is difficult, leave it. Find and define the sac back to the *linea alba* (18-19B).

Reduce the contents of the sac, if it is not already empty, and open it between haemostats, as usual when entering the peritoneal cavity (11-2). Enlarge the opening with small lateral incisions. Close the sac with a purse-string suture, or, transfix, tie, and excise it (18-19C).

Drop the stump back into the abdomen. Overlap the edges of the rectus with interrupted sutures (18-19D). Close the skin, and apply a firm dressing (18-19E).

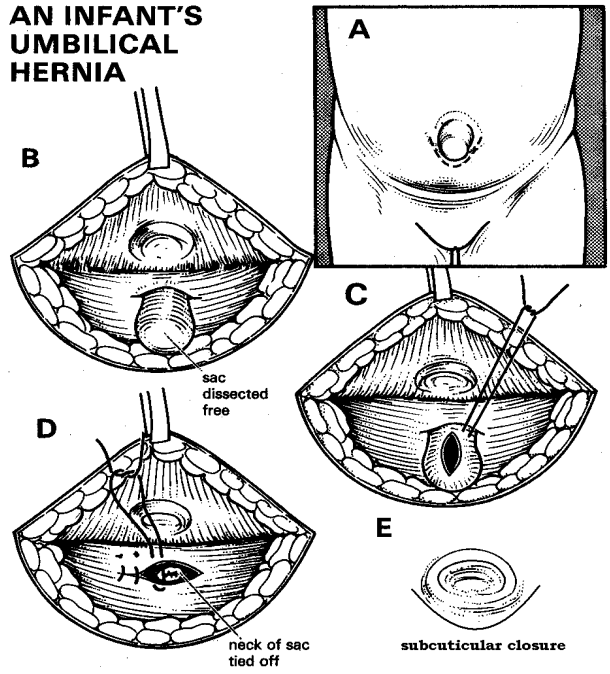


Fig. 18-19 INFANTILE TYPE OF UMBILICAL HERNIA.
A, make a curved incision just below the umbilicus. B, reflect a skin flap which includes it. C, open the sac at its neck, and close it with a purse-string suture. D, close the opening in the *linea alba* with a few interrupted sutures. E, operation complete.
After Rob C, Smith R Operative Surgery: Part I: Abdomen, Rectum and Anus, Butterworth 2nd ed 1969 p.208 Figs 1-4. with kind permission.

(c) Difficulties with umbilical hernias
If there is a discharge at the umbilicus, check for an inspissated keratin plug deep in the umbilicus: lift it out and clean the skin. Ask if it has been present since birth and look for vitello-intestinal or urachal remnants.

KAKAZI (14yrs), who had just received a letter admitting her to a secondary school, presented at a remote rural hospital with an obstructed, infected, ulcerated, gangrenous umbilical hernia the size of a small fist. She was vomiting and the abdomen was distended. There were no sterile drums, and no diesel with which to run the generator and operate the electrical sterilizer. There was no petrol for the ambulance, so she could not be referred. Equipment was sterilized on a charcoal stove. She was given the hospital's last bottle of intravenous fluid and anaesthetized with ether. There was no hernial sac to isolate, because infection had destroyed it. Gangrenous small bowel was resected and anastomosed, the abdomen was closed, she recovered completely, and did not lose her place at the school. LESSON Never give up! *Thanks to Dr Bosco Rwakimari.*

18.11 Paraumbilical hernia in adults

Try to distinguish in adults whether a hernia in the umbilical region occurs above or below the umbilicus, through a weak place in the *linea alba*, rather than directly through the umbilicus itself. You may occasionally see a true umbilical hernia so huge that it can accommodate a pregnant uterus! This may arise from a healed exomphalos (33.4) where the defect was never repaired in childhood.

The patient is usually an obese multiparous woman, with a large multilocular hernia in the upper part of the umbilicus. Its margins are firm, so that obstruction and strangulation, particularly Richter-type strangulations of the large bowel (18-2), are common.

Repair of a small paraumbilical hernia is quite easy; but repair of a large hernia is difficult, because:
(1) The viscera in the sac stick to its wall, and in freeing them you may damage bowel.
(2) There are usually several loculi, divided by fibrous septa.
(3) The sac often extends to the skin.
(4) You need to raise flaps, under which blood and exudate can collect and become infected post-operatively. Minimize this risk by closing the dead spaces under any flaps you make, as best you can.

(a) Differential diagnosis
A hard lump at the umbilicus may not be an irreducible hernia.
If it bleeds at menstruation, it is endometriosis.
If it is stony hard, it is a Sister Joseph's nodule indicating widespread intra-abdominal malignancy.
If it is purplish with a tendency to ulcerate and bleed, it is Kaposi sarcoma.

(b) Paraumbilical hernia repair in adults (GRADE 2.4)
(1) PREPARATION. If the patient is very fat, encourage him to lose weight before you operate. Obesity makes surgery difficult and recurrence much more likely. Clean the umbilicus carefully to remove all debris that might contaminate the wound. Warn the patient that, if the hernia is large, you may have to remove the umbilicus.

(2) SMALL PARAUMBILICAL HERNIAS
These are hernias in which the lump (not the ring) is <5cm in diameter. Preserve the umbilicus if you can.

INCISION
Put a plastic-covered pillow under the knees to help relax the abdomen. Make an elliptical incision 1cm above or below the umbilicus. Above, make the incision concave downwards; below, make it concave upwards. Extend it so that it goes 2cm beyond the lump on either side.

Deepen the incision down to the *linea alba* and the rectus sheath on either side of the hernia. Reflect a flap above if your incision was below the umbilicus, so that you can see all round the hernia.

Control bleeding carefully with diathermy. Define the margins of the neck of the hernia. This is seldom as neat as in other hernias. You may not be able to grasp the fundus if it is firmly attached to the umbilicus, so define the neck on all sides, and ignore the fundus initially.

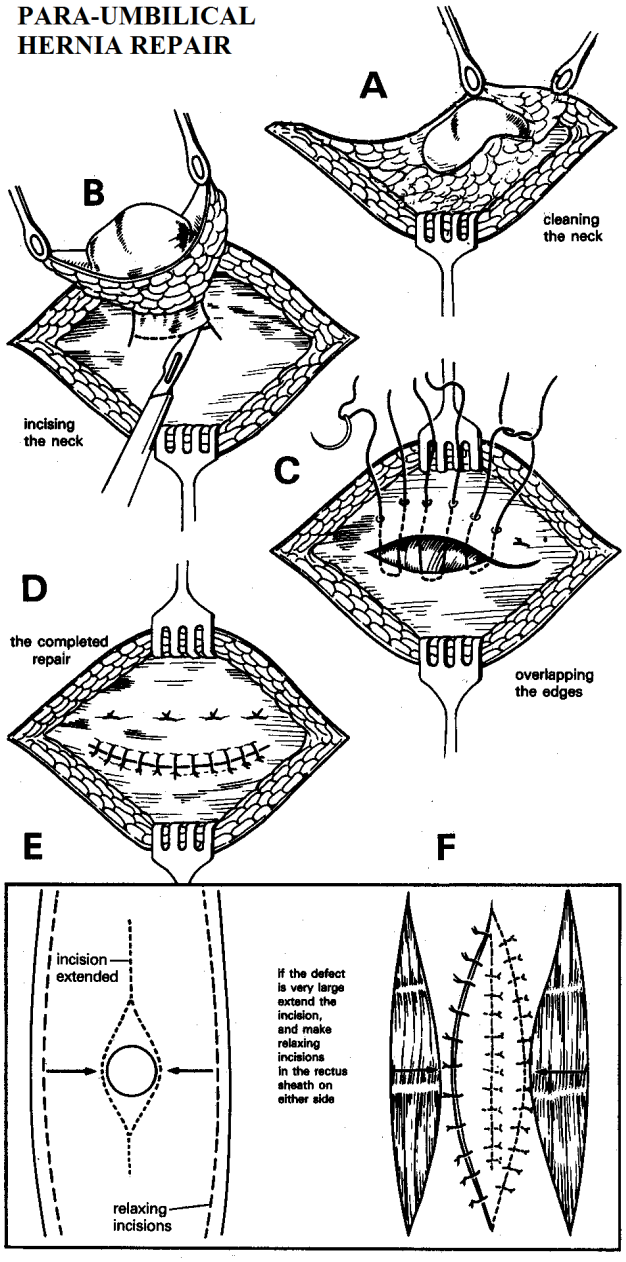


Fig. 18-20 MAYO'S OPERATION FOR PARAUMBILICAL HERNIA. A, undermine the upper flap and clear the tissues towards the neck of the sac. B, cut round the neck, and reduce the sac. C, insert mattress sutures, so as to pull one flap under the other. D, complete repair. E,F, if the defect is very large, you may have to extend it longitudinally, make relaxing incisions in the rectus sheath on either side, and then overlap the aponeurosis at its edges.

If the hernia is small and truly umbilical, try to dissect under the skin around the umbilicus. You can then pass a sling around it and pull it forward. Open the sac close to its neck, because this will be free of adhesions. Do this between haemostats, as if you were opening the peritoneum (11-2).

Continue to open the sac with blunt-tipped scissors, working from the neck towards the fundus. As soon as you have made a sufficient opening, put your finger into it and feel for adhesions. Cut round the circumference of the sac with scissors (18-20B). Carefully examine the contents of the sac.

If a loop of bowel has stuck to the sac, pass your finger up beside it. Find a part of the sac wall which is free of adhesions, and open this up as best you can: it is better to leave a piece of sac adherent to the bowel than to injure it.

If the omentum has stuck to the sac, clamp, transfix, tie, and divide small sections of it at a time, and then return it to the abdomen.

Turn the sac inside out, so that you can see its contents and peel them away. Remove adherent omentum along with the sac, and separate adhesions between loops of bowel. Finally, cut the umbilical skin off the fundus of the hernia.

Enlarge the opening in the abdominal wall laterally, without trying to separate the peritoneum as a separate layer. The rectus muscles will probably be so widely separated that you will not need to open their sheaths. If necessary, incise the anterior rectus sheath at the ends of these incisions, but *don't injure the rectus muscle*.

You will probably be unable to separate the peritoneum as a separate layer, so suture it with the *linea alba*, which is likely to be broad. Overlap the upper and lower edges of the defect. Clear the under-surface of the superficial flap free of as much fat as you can. Then insert several mattress sutures of #1 monofilament (18-20C). When these have drawn one flap under the other, insert some simple interrupted sutures (18-20D) to 'double-breast' the repair. Ensure haemostasis. Close the skin incision, and apply a pressure bandage over the wound.

(3) LARGE PARAUMBILICAL HERNIAS

Under GA, proceed as above, making a longitudinal elliptical incision to include and excise the umbilicus. Dissect down to the fascia above and below the umbilicus on either side. Open the sac at its neck. Expect it to have several loculi, and be prepared to find firmly adherent transverse colon. Evert the sac, and carefully free the viscera from the loculated pockets of the sac.

You may be able to overlap the edges of the sac longitudinally. Make a long midline incision and lateral relaxing incisions in the rectus sheath (18-20E,F). Overlap the sheaths and suture them with #1 monofilament. If this is inadequate, use a mesh (18.13).

Suture the superficial fascia to the anterior rectus sheath. If possible, insert suction drains. Apply pressure dressings, and hold them in place with an abdominal binder, or plenty of adhesive strapping.

(c) Difficulties with paraumbilical hernias
If a paraumbilical hernia discharges faeculent fluid, a loop of large bowel has strangulated and perforated, and caused a faecal fistula. General peritonitis is usually prevented by the tight fit of the neck of the sac, which seals off the rest of the peritoneal cavity.

The differential diagnosis includes other causes of a discharging umbilicus, carcinoma of the transverse colon or stomach, and a persistent vitelline duct. A patent urachus will discharge urine.

You can deal with this electively: perform a laparotomy taking care not to soil your operative field. Resect the colon with the hernia *en masse* and close the abdominal wall, but leave the skin open.

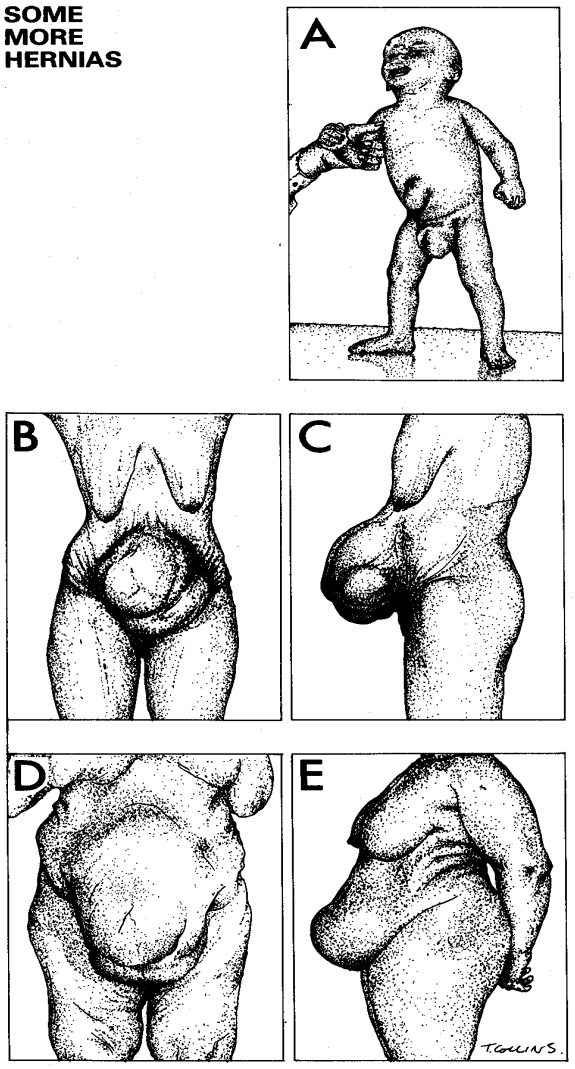


Fig. 18-21 SOME MORE HERNIAS.
A, umbilical and inguinal hernia in boy of 3yrs. The inguinal hernia will need repairing, but the umbilical one will not. B,C, paraumbilical hernia containing most of the patient's bowel and omentum. Note the healed ulcer on the fundus. The mass is knobbly because of visible peristalsis. D, incisional hernia following midline laparotomy. E, incisional hernia following Caesarean section.
After Worsornu, External hernia – ventral hernia and summary. Tropical Doctor 1975;5(2):56-61, with kind permission.

18.12 Epigastric hernia

A patient with an epigastric hernia complains of attacks of pain and a lump, or occasionally more than one lump, which may be surprisingly painful. You will find a small, soft, rubbery, globular, and sometimes lobulated lump, somewhere along the *linea alba*, between the xiphoid process and the umbilicus. Extraperitoneal fat has bulged through a small (≤10mm) cleanly punched-out hole. It may be so close to the umbilicus as to resemble an umbilical hernia. Because the fat in it is tightly wedged, it has no cough impulse, and you cannot reduce it. You can easily mistake it for a lipoma, although it is more firmly fixed. The key to the diagnosis is its position. *Many such patients have been treated for a long time with antacids because they have never been examined!* Repair is usually straightforward. These hernias rarely strangulate.

If the whole length of the upper abdominal midline bulges on sitting up, this is a divarication of the *recti* (18.9), and rarely needs surgical correction (which is anyway not easy).

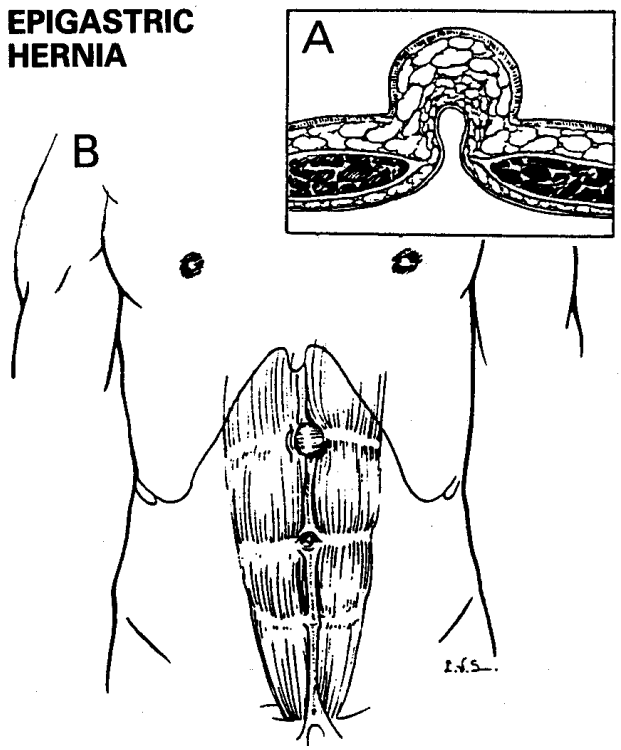


Fig. 18-22 EPIGASTRIC HERNIA. Note that this has occurred through the *linea alba* above the umbilicus.
After Dunphy JE, Way LW. Current Surgical Diagnosis and Treatment. Lange Medical 3rd ed 1979 Fig. 36-7 with kind permission.

EPIGASTRIC HERNIA REPAIR (GRADE 2.2)
If you find an epigastric hernia incidentally and it is small and painless, leave it. If the lump is more then 3-4cm, and is causing symptoms, repair it.

ANAESTHESIA. Infiltrate with 0·5% lidocaine with adrenaline.

INCISION. Make a small vertical incision and dissect out the mass.

Clean and mobilize the sac. It will probably dissect off easily. The contents are usually fat or omentum, rarely transverse colon, and extremely rarely small bowel. Reduce the mass by massaging it back inside the abdomen with your fingers. Repair the defect with a monofilament suture.

If you cannot reduce the hernia, enlarge the defect in the *linea alba* by extending the incision. Remember there may be 2 separate hernia defects.

If the hernia is tender (very rare), open the hernia sac and examine its contents, and act accordingly.

18.13 Incisional hernia

These range from a small bulge at the site of a stab drain, to the huge multiloculated swelling that follows the breakdown of a major incision, usually a lower midline laparotomy. Incisional hernias are more likely in the following circumstances: poor suturing, particularly with catgut (11.8), missed repair of the posterior rectus sheath, infection either in the wound or from generalized intra-peritoneal sepsis, a chronic cough, constipation, ascites, HIV disease, advanced malignancy or malnutrition.

Incisional hernias are, however, avoidable! They are, by definition, lumps or bulges under the scar of a previous abdominal incision. If they grow very large, the bowel may only be covered by peritoneum and skin, which may be paper-thin and adherent to the bowel itself. If they are longstanding, the rectus muscles may have separated widely, so that the abdominal contents flop outside the belly. The commonest lower midline incisional hernias are not too difficult to repair but often recur if the repair is not done carefully.

Although recurrence is common, strangulation is not, so *don't operate on these hernias unless you have to*, especially if the hernia is large, and below the umbilicus. Obesity makes repair even more difficult.

(a) Examination

With the patient supine, put your hand through the weakened area in the abdominal wall to feel the size and shape of the hernia. It may be elliptical, or irregular, and there may be more than one defect.

Ask him to raise his head and shoulders off the couch without using his arms. This will fill the sac and show you its true size. If so, you should be able to repair the hernia without too much difficulty.

(b) Management

Unless you are experienced, *don't operate on difficult recurrent, large, incisional hernias*, especially with HIV or other systemic disease present *(including obesity)*; advise wearing a corset if the hernia is completely reducible and symptomatic.

If you are experienced, you can sew in a mesh (best over the posterior rectus sheath layer (the underlay method)): you can use sterilized mosquito netting. Take precautions to prevent infection: if this ensues, you'll have to remove the mesh. You must make sure that the mesh is sutured carefully to the sheath at least 2·5cm beyond the edge of the defect. *Don't put the mesh directly over the bowel* (inlay method), because it may erode into the bowel wall and produce terrible fistulae.

PREPARATION. Encourage an obese patient to lose weight. If there is infected intertrigo, prepare the skin with special care some days beforehand.

ANAESTHESIA. You will need good relaxation, so use subarachnoid (spinal) anaesthesia, or GA with relaxants. While the abdomen is relaxed under anaesthesia, feel the margins of the defect carefully.

LOWER MIDLINE INCISIONAL HERNIA REPAIR (GRADE 2.5)

INCISION. Make an elliptical incision in the long axis of the hernia, wide enough to include a ⅓-½ of the bulging skin, and extending 4cm beyond the defect at each end. Design the ellipse so as to remove the original scar and to produce a new one, without redundant skin or a tense suture line.

Define the margins of the defect. Use sharp dissection to free the peritoneum, and the anterior rectus sheath, from the fleshy fibres of the rectus muscles, which are sandwiched between them. The posterior rectus sheath, which has often been missed out in the previous closure, may have retracted quite a long way laterally.

Control bleeding, which may be troublesome, and try to repair the lower abdominal wall, layer by layer. Make flaps at either side, so that the skin and subcutaneous tissue (if there is any) are undermined for at least 4cm to allow for tension-free closure.

Try to find a plane of cleavage between the peritoneum and the skin, without button-holing either of them. Undermining will be easier if you insert tissue forceps at the skin edge, and ask your assistant to exert traction on them, while you dissect the skin from the underlying sac.

If freeing the ellipse of skin from the underlying hernial sac is difficult, because the hernia is subcutaneous in the centre of the sac, leave the ellipse attached to the sac.

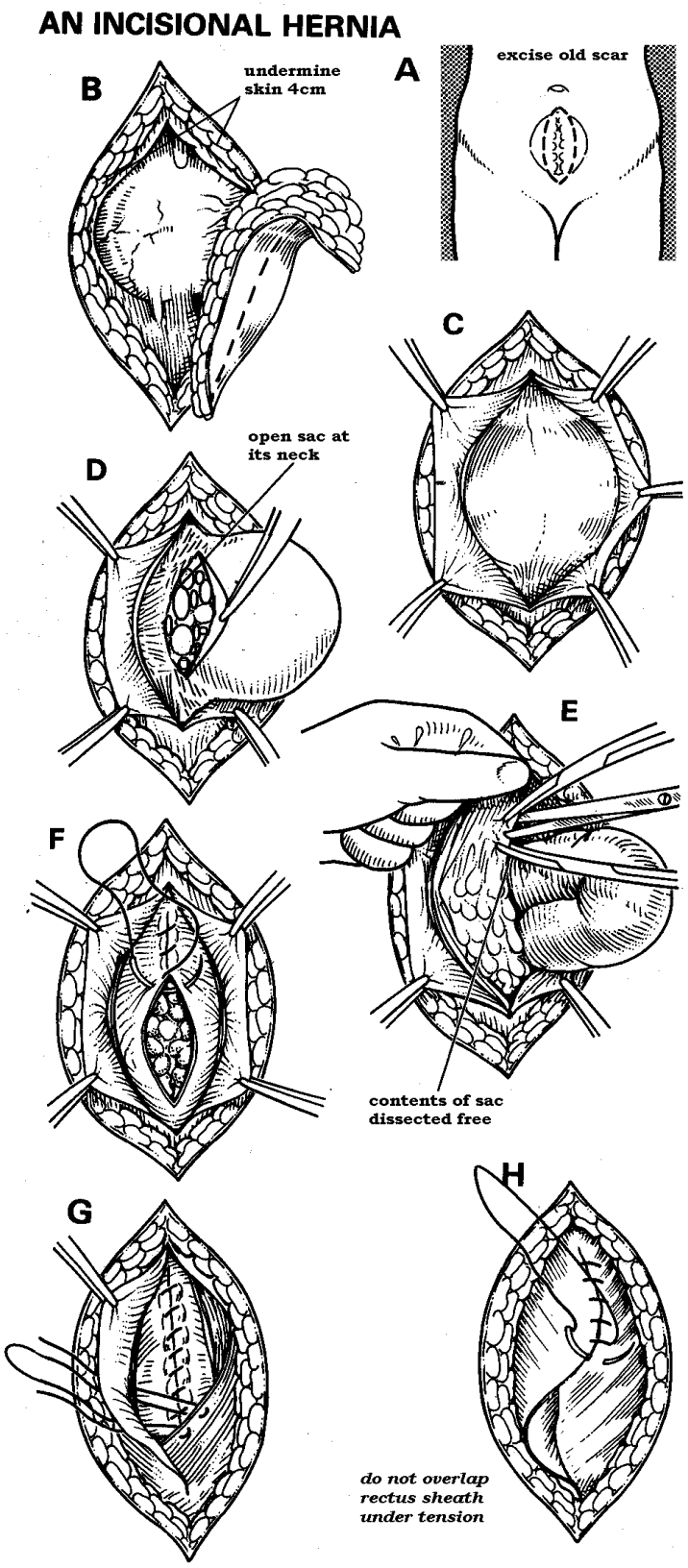


Fig. 18-23 INCISIONAL HERNIA.
A, remove skin ellipse with the old scar. B, undermine skin edge 4cm. C, dissect flaps of stretched aponeurotic tissue from the sac. D, open hernia sac at its neck. E, free adherent contents of the sac. F, close peritoneum and posterior rectus sheath. G,H, overlap anterior rectus aponeurosis. *If there is tension, insert a mesh.*
After Rob C, Smith R. Operative Surgery: Part I: Abdomen, Rectum and Anus, Butterworth 2nd ed 1969 p.212 Figs 1-9 with kind permission.

Proceed to raise flaps of scar tissue as described below, and excise the ellipse and part of the sac together. Take care to control all bleeding points carefully.

Raise flaps of aponeurotic scar tissue from the covering of the sac on either side (18-23C).

The neck of the sac will probably be diffuse, and not easy to define. Open it between haemostats, as for a laparotomy (18-23D), and incise the peritoneum far enough to see if there are adherent loops of bowel. Free these adhesions and the omentum (18-23E), and return the bowel and omentum to the abdomen.

If you cannot easily free the bowel and omentum from the fundus of the sac, leave them attached to it; free it from the skin, if you have not already done so, and fold the sac inwards into the abdomen. Check the viability of the bowel (11.3, 11-6), and resect any non-viable part.

Excise the redundant part of the sac (*i.e.* the peritoneum), and close this layer, taking good bites of the retracted posterior rectus sheath with continuous monofilament (18-23F).

Dissect and trim the scarred flaps of aponeurosis, to expose the edges of the rectus muscles on either side. If these have been stretched and thinned out, trim these flaps away to leave a broad strip on one side, and a narrow strip on the other. Overlap these strips so as to bring the rectus muscles to the midline (18-23G).

Otherwise, if they don't come together easily, *don't overlap them*: you must avoid tension in your repair. You can use continuous or interrupted #1 monofilament sutures. Take close small bites making sure you get a suture length-to-wound length ratio of 4:1, and *don't tie the sutures so tightly* that they strangle the tissues. In this way, a double-thickness layer of fibro-fascia will replace the *linea alba*. Insert a few absorbable sutures between the superficial and the deep fascia, in order to obliterate any potential spaces where blood might collect.

Insert a multiple perforated tube through a stab wound, let it lie under the flap, and attach it to a low-grade suction apparatus. Suture the skin edges, apply a firm pressure dressing, and *don't disturb it until the sutures are to be removed*. A many-tailed bandage (an oblong piece of cloth with cut strip tied or overlapped and pinned around the abdomen) will provide physical and psychological support.

CAUTION! If a cough develops post-operatively, it is likely to disrupt the repair. Teach supporting of the wound by pressing the hands on the sides of the abdomen. Use a corset.

(c) Difficulties with incisional hernias

If you cannot overlap the aponeurotic layers, make parallel relieving incisions 10cm laterally into the sheath only, but beware this is likely to be bloody, so have diathermy ready.

If the defect is so large so that you cannot bridge the defect, you will need to suture in a mesh to close the defect. You can use sterilized insecticide-free mosquito netting; if you don't have any mesh, *don't try to make a repair under tension*.
Put the mesh in under the rectus muscle, but over the posterior rectus sheath (**underlay method**); you need to dissect the rectus sheath quite far out laterally, inferiorly and superiorly to secure the mesh, and this really needs diathermy. Use prophylactic cloxacillin intra-operatively. Close the mobilized anterior rectus sheath, and advise wearing a corset. *Don't lay mesh directly on bowel.*

N.B. You can put the mesh over the rectus muscle, under the anterior rectus sheath, though this may be very deficient and tempt you to use the mesh as a sort of fill-in graft for the defect (**onlay method**). This is simpler but much less effective, and if the wound becomes infected, you may end up having to remove the mesh to control the sepsis!

If there is a recurrent incisional hernia, repair is likely to be very difficult indeed. If the patient is comfortable in a corset, treat her non-operatively.

If the pregnant uterus bulges through an incisional hernia, consider doing the repair immediately after delivery, and tying the tubes.

If there is a persistent wound infection after a mesh repair, remove the mesh. Sometimes this gets 'swallowed' by intestine, resulting in a fistula: you will then have to resect the affected portion of bowel as well as removing the mesh: this is difficult surgery!

(d) Peristomal hernias

Stomas inevitably leave a weak area in the abdominal wall, through which bowel may herniate either alongside the stoma itself, or into the layers of the abdominal wall. If the weakness in the abdominal wall is just a bulge, owing to weakness of the muscles themselves, *don't attempt any repair* (11.6). If there is a true bowel herniation, you will probably need to re-site the stoma and repair the old defect. Make sure you select the new stoma site carefully pre-operatively (11.5).

Perform a formal laparotomy; start by mobilizing the stoma as for its closure (11-16) till it is free, then deal with the herniated bowel, and finally re-fashion a new colostomy at the new site, making sure you have mobilized the bowel enough to reach its new position. Then carefully close the old stoma defect layer by layer.

You may be able to approach the defect extra-peritoneally and repair it successfully by dissecting around the stoma: this is difficult and, to be effective, probably needs insertion of a mesh.

(e) Other incisional hernias

Any abdominal incision repair may give way and result in herniation, but this is very rare indeed in Pfannenstiel or Kocher's (subcostal) incisions; an appendicectomy incision (gridiron or Lanz) (14.1) may leave a small defect through which bowel may herniate. This needs repair because strangulation can occur suddenly.

The lumbar incisional hernia found after operations on the kidney through the flank, however, rarely needs repair.

19 The surgery of conception

19.1 Maternal mortality

Over half a million women died in 2005 of pregnancy, 99% being in low- and middle-income countries. The chances of this happening depend on how often a woman becomes pregnant, and how dangerous each pregnancy is. This can be measured by the maternal mortality ratio (MMR), which is the number of maternal deaths during a given time period expressed per 100,000 live births during the same time period. This is not the same as the number of deaths per pregnancy because this depends on the total fertility rate (TFR).

A maternal death is: '*The death of a woman while pregnant or within 42 days of termination of pregnancy, irrespective of the duration and site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes.*' International Classification of Diseases (ICD-10)

In 2013 the global MMR was 289. In sub-Saharan Africa (population 950 million) 180,000 women died of a pregnancy, in Southern Asia 60,000 and in Western Europe and North America (population 800 million) only 1,000.

The MMR includes direct causes (ectopic gestation, uterine rupture, septic induced termination, bleeding after delivery) and indirect causes (malaria, HIV, TB, severe anaemia) but excludes accidental causes (*e.g.* car crash while shopping, but includes an ambulance involved in a crash during referral for complications in pregnancy). It excludes incidental causes (*e.g.* poisoning or suicide); although these deaths could be related to the pregnancies, they are kept out of the definition.

To facilitate the identification of maternal deaths in circumstances in which cause-of-death attribution is inadequate, ICD-10 introduced an additional category, **pregnancy-related death**, which is defined as the death of a woman while pregnant or within 42 days of termination of pregnancy, irrespective of the cause of death.

If official death registration forms could have one extra question – "Was the deceased pregnant or within 6wks of a pregnancy when she died?" – then the collection of this data would be much easier in many countries.

Another useful statistic is the **lifetime risk of maternal death**: this is the probability of maternal death during a woman's reproductive life, usually expressed in terms of odds: these were, in 2013:

Afghanistan and Sierra Leone, $^1/_6$
Sub-Saharan Africa, $^1/_{16}$
Northern Europe, $^1/_{25000}$

(a) The causes of maternal mortality (MM) in sub-Saharan Africa, which broadly reflects the situation elsewhere in LMICs, are:

(1) Haemorrhage	35%
(2) Sepsis/infections	10%
(3) Hypertensive disorders	10%
(4) HIV/AIDS	6%
(5) Termination (mostly induced)	4%
(6) Obstructed labour	4%
(7) Anaemia	4%
(8) Embolism	2%
(9) Other indirect causes	17%
(10) Other direct causes	5%
(Unclassified	*3%)*

N.B. Regarding (4): An HIV+ve woman has a 4-5 times higher chance of dying in pregnancy than an HIV-ve woman. The extra MMR related to HIV was 1,300. HIV infection in pregnancy increases the risk of obstetric complications. HIV-related illness such as anaemia or tuberculosis might be aggravated by pregnancy. The quality of care received by women who are known HIV+ve might also be worse than that received by other women. There is no good evidence that HIV disease progresses faster because of pregnancy. Biological changes are probably responsible for the fact that women are twice as easily infected by HIV when pregnant. Existing programmes to prevent mother-to-child transmission need also to inform HIV-ve women about the increased risks of acquiring HIV if they fall pregnant.

Regarding (5): This might be an underestimation. The WHO systematic review estimates an MMR from induced termination as 37 in sub-Saharan Africa, 12 in South Asia, and 23 in Latin America. However, much higher estimates have been reported globally and in individual studies, especially in countries where termination is illegal.

Regarding (10): This includes ectopic gestation, for example.

(b) Where and when do women die

Maternal deaths happen in <50% cases at home. This is likely to be higher in Islamic countries but figures are hard to come by. Most die in a health institution where they might have arrived moribund. Much dying also happens *en route*. Most die on the day of delivery or in the next few days. However, the theoretical lowering of MM by living in town can be easily nullified by a higher incidence of unsafe termination and of HIV. An academic hospital will not be very effective in this respect if most of the work is done by unsupervised junior doctors; a district hospital may often then be a safer place to deliver! As indicated previously (1.5), delays play a very important role in the failure of women being able to seek medical attention.

The first potential delay is the recognition by women and their relatives of the need for medical help. The second delay relates to physical, cultural, and financial constraints as well as the security situation. The third delay relates to the time needed to organize and pay for the actual medical care.

(c) Room for improvement

In Latin America and North Africa the MMR figures have improved impressively. There seems to be little progress in sub-Saharan Africa. Income is an important factor between and within countries but does not explain all the differences. For example, people in Sri Lanka, Vietnam, Ecuador, and Ghana have similar purchasing power. Their estimates of MMR are respectively 29, 49, 87, and 380. Countries such as the Netherlands, Sweden and Denmark had MMRs of 300-350 a century ago, mainly because of the presence of many well-trained midwives. The USA caught up (in 1935 the MMR was around 600) only when there were enough doctors to provide maternity care.

The MMR varies widely as seen above. It used to be high all over the world. History shows how often kings lost their queens in childbirth. The natural MMR, which means no medical interference whatsoever, is around 1,600. In some communities in Africa and Afghanistan it is still 1,000 or more, which means that a mother has around a 1:100 chance of dying from pregnancy-related causes/per pregnancy, or a 1-in-14 chance in her lifetime, if she has 7 pregnancies. The result is that a woman living in a remote area of Afghanistan has a 1,000 times greater lifetime chance of dying of a pregnancy than a woman in Scandinavia. The MMR in high-income countries makes almost no impact on the global MMR. One of the problems apart from war is that in certain countries, authorities discourage (higher) schooling of women, as was the case in Europe 100 years ago, and male doctors are forbidden to attend to pregnancies, as in Europe 400 years ago.

The deaths of mothers are more difficult to prevent than those of their children, because a good basically equipped hospital with well-trained staff is needed. On the other hand, reducing infant and under-five mortality by means of vaccinations, provision of clean water and improved nutrition is less of a technical problem. The provision of a new district hospital, including its staffing and transport access, often results in good outcomes from high-risk pregnancies of women staying in the shelters nearby. However, the low-risk pregnancies, which still may have unexpected complications, continue to do badly because these women arrive in hospital too late. This is related usually to traditional customs and beliefs. Organizing a 'bed & breakfast' industry for all women in the last 6wks of their pregnancy near every good district hospital might prevent much misery.

Something else which should be easy is providing for the unmet need for contraceptives. You only need staff, dedication and the right contraceptives (for which donors are easy to find). Half of the pregnancies in the world are unintentional. To prevent these would halve the number of women who die of pregnancy. On a global scale this has already happened: in 1950 the TFR, the average number of children a woman bears, was 5; recently it has become 2·5. This is of course not represented by the MMR because this refers to how dangerous each pregnancy is. Nonetheless, in absolute numbers, it makes a big difference. Furthermore, the MMR is likely to go down because high-risk pregnancies will decrease when contraceptives are widely available. There will be more health workers per delivery and the economic benefits are likely to increase the budget for the Ministries of Health and Education. Educated women are far less likely to die of a pregnancy.

Women who die often have not had antenatal care (ANC); they are 'unbooked' and they are often seen late when there are problems. ANC detects some problems in time (anaemia, syphilis/HIV/malaria infection, breech presentation, high blood pressure, severe growth retardation) and makes it possible to discuss a plan: "Are you going to stay with your cousin near the hospital? What will you do if your membranes rupture too early? What transport options exist at various times of the month/week/day? How can we prevent the next potentially dangerous pregnancy? Do you wish for and are you ready for sterilization?"

Saving pregnant women is *not* basically a question of having beautiful machines, laboratories and computers. In North Western Europe before the Second World War (1939-1945), the perinatal and maternal mortality rates were better than they are in many large African hospitals now. At that time the Caesarean section rate was <1%. There were no antibiotics, modern anaesthetics, safe blood transfusions, vacuum extractors, reliable IV oxytocin, anti-D and anti-tetanus injections, prostaglandins, contraceptives other than condoms, cardiotocographs, ultrasound, or Doppler machines, or modern suture materials. There was little evidence-based knowledge. Polio, rachitis (severe pelvic deformity due to vitamin D deficiency) and (then untreatable) syphilis were common. The difference was that the facilities were provided and there were enough staff. They received reasonable salaries and pensions. The exodus of staff for more lucrative jobs was rare and they were revered for identifying with the suffering of their own people. So encourage such staff, and do what you can to improve their working conditions!

Never in history has a doctor, prepared to work in a rural district hospital, had so much opportunity to save lives. Saving their lives requires also improved education for women and services at 3 levels: in the community, in clinics and health centres, and in basically equipped and staffed district hospitals. Especially, it needs plenty of well-trained midwives. The midwives alone made the first large dent in the MMR in Europe. Furthermore, non-doctors are easier to retain. A study from Mozambique showed that over 7yrs 88% of the medical assistants were still working in the rural areas but *NONE* of the doctors!

Changing maternal mortality needs political will. To raise this, you will need to know your local/regional MMR. To get an idea, ask adults what happened to their adult sisters and whether or not they died in childbirth. Their mortality experience will be a measure of that of the community as a whole.

You can obtain more or less reliable figures plus much more statistical data like **total fertility rate**, **contraceptive usage**, **infant mortality rate**, **family planning** and **death rates** from the UN Demographic Yearbook, DHS (http://www.measuredhs.com) or from your Central Statistical Office.

A simple way in LMICs to monitor the very basic quality of obstetric care and associated transport facilities is to record the vesico-vaginal fistula (VVF) rate. This is easier to prevent and record than MM. Women with VVFs are very likely to present at some stage at a health facility. All VVFs with all details of the identity of the woman involved and date and place of delivery, transport bottlenecks and so on should be sent once a year to the Ministry of Health. At that level duplicate recording of women can be sorted out and an annual VVF report made. This might be a very sensitive indicator of the year-on-year difference in quality of obstetrical care. VVFs caused by obstructed labour (not those made at Caesarean section) happen only when women have been in labour for >2 days. It could also show very well, for example, that an increase in the price of fuel results in more VVFs.

19.2 Obstetric aims and priorities

Between 5-10% of the babies of LMIC mothers die in the perinatal period (from 28 weeks of pregnancy to 7 days after delivery), frequently related to the development of the placenta. Preventable causes include malaria, syphilis, and obstructed labour. Most perinatal deaths are of normally formed, normal weight babies who die avoidably from trauma, asphyxia, or infection. Statistics show that perinatal mortality decreases with the number of weeks of pregnancy. At around 36wks this trend reverses because mechanical factors, especially cephalopelvic disproportion (CPD), are now playing an increasing role while these deaths are, in theory, all preventable.

Many neonatal deaths also occur in babies whose low birthweight is due to their being born too soon (prematurity), or to not having grown normally before birth (intrauterine growth retardation: IUGR). If a mother dies, the baby is also likely to, even if it is born alive in a good condition. The baby will often die without the breast milk and the special attention which is nearly unique to the biological mother. The deaths of both mothers and babies are largely due to the material, political, and socio-economic conditions and traditions under which they live. Here we are concerned with obstetric causes of death.

Despite the challenges of pregnancy and childbirth, an important task in many communities is to reduce the frequency with which pregnancy occurs. Most governments are in theory convinced that proper development cannot be combined with women having many children. The problem is often that governments have many tasks and not enough resources. Facilitating free access to **family planning**, however, is a task governments often don't see as a priority because there may be stiff opposition from various quarters. Reducing unwanted pregnancies will decrease the need to keep on building new schools, hospitals and universities, create jobs and to import food.

Populations especially in Africa and the Far East, and to a lesser extent elsewhere, are growing so fast that they are causing acute pressure on land, on food, on wood (to cook the food), on jobs, on education, and on the health and other social services. Increasing population brings pollution and land degradation. In some areas this population pressure is already finding its expression in desertification and starvation, in abject poverty, in people risking their lives trying to flee to Europe and the US, and in civil disorder. The slaughter in Rwanda, but also the Second World War, stimulated by the German call for *Lebensraum* (space for living), were clearly related to deemed population pressure. The population in Rwanda has since increased again to the previous level. Your own community may not have reached this point yet, but is it already exerting such pressure on its environment that 'ecological collapse' is not far away? If it occurs, your community may become 'ecological refugees', if indeed there is anywhere to flee to. If birth rates don't fall, death rates may rise to their old values (of 100 years ago) or higher, with a much larger population in a much-impoverished environment.

This is described as the **demographic trap**. Europe solved its population pressure problems in the past by colonization. The result is that half the people of European stock live in the 'New Worlds', where they drove away or killed the indigenous peoples. Africa and the Middle East don't seem to have that option. China, South Korea, Thailand, Singapore, and Sri Lanka have shown that limiting population increase can allow for enormous economic boost, resulting in a rise in the standard of living. This rise is not, however, without problems because of risks of gender number inequality, where boys are valued more than girls, who are therefore eliminated.

The chance for all the people in the world to have a reasonable standard of living becomes more remote if the world population keeps on increasing at the rate it has done recently (1 billion people extra every 15yrs) despite HIV disease. If everybody has 2 children only, there might just be enough food globally if it is equally distributed. If everybody has 4 children, such equal distribution will not help. The rich must adjust their standard of living and reduce their waste, and the poor should have fewer children. But without reduction in child mortality, women will still want many children as an insurance for the future. Many politicians also believe that a large population is important because it makes a country powerful. But Sweden (7 million) has probably more influence than Bangladesh (145 million). If China had not had a population policy, it would have had 350 million more people and not be such an economic powerhouse as it is today.

Because so much obstetrics must be delegated, the instruction and supervision of those to whom you delegate it are critical. Some mothers will be delivered in hospital, and some by midwives in health centres. Most of them will probably be delivered at home, attended either by their families, or by traditional birth attendants (TBAs), such as the *dais* of India. A study from Burkina Faso showed that active encouragement from the local chiefs increased the deliveries in health institutions from 25% to 56%.

Another way to reduce the maternal and perinatal mortality in your district may be to start with the TBAs, to concern yourself with what they do, and to instruct them where you can. If a specialist group of TBAs are at work in your area, each of whom delivers several mothers every year, try to run training courses for them. For example, as the main cause by far for maternal mortality outside hospital is post-partum haemorrhage (PPH), you might start a programme in which TBAs are provided misoprostol x3 tablets (600µg) to dispense orally directly after the baby is born (if not a twin).

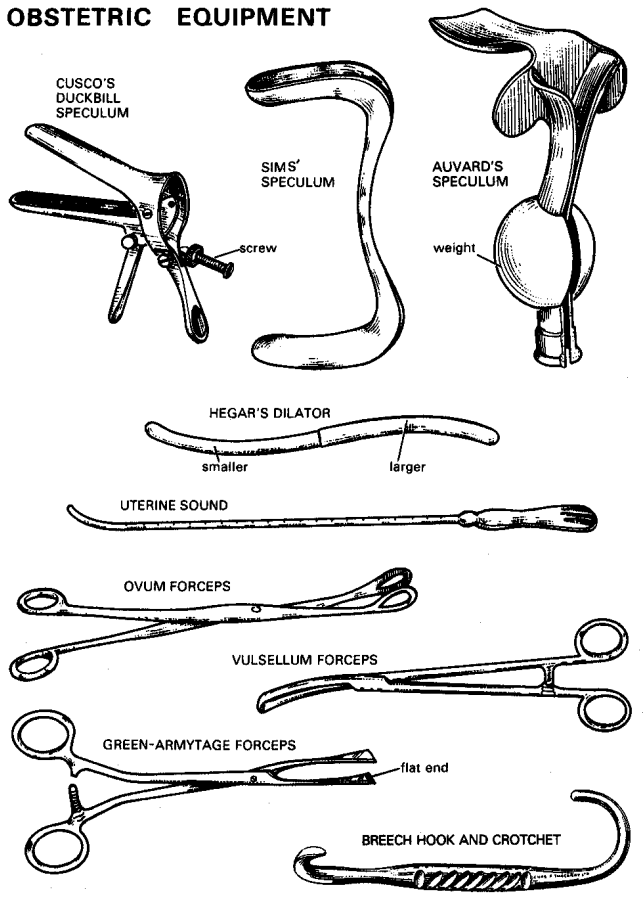


Fig. 19-1 SOME OF THE EQUIPMENT you will need.

STETHOSCOPE, foetal, plastic. These don't bend so easily as aluminium stethoscopes.
DOPPLER FOETAL HEART DETECTOR, 'Sonicaid' pattern or equivalent. This is comparatively inexpensive (about $250) and very useful.
PORTABLE ULTRASOUND. This is so useful that you should really try to get one.
SPECULUM, vaginal, Sims', double-ended, medium size, 27-30mm.
SPECULUM, vaginal, Cusco's, duckbill, small and large, stainless steel. These specula open like the beak of a duck, and in doing so enable you to examine the cervix.
SPECULUM, vaginal, weighted, Auvard's, chromium-plated. The weight on this speculum presses it downwards, and so keeps the vagina open.
FORCEPS, uterine vulsellum, curved, 1-2 teeth and 3-4 teeth, box joint, 230 mm. Use these to grasp the cervix when you curette it.
FORCEPS, post partum ring (sponge) are better to grasp the cervix.
DILATORS, cervix, double-ended, Hegar's set of 12 sizes, 1/2mm to 23/24mm. Use these to dilate the cervix before curetting it.
MANIPULATOR, uterine. Use this to bring the uterine fundus up against the abdominal wall when you perform a mini-laparotomy.
SOUND, uterine, malleable, metric, graduated shaft. Use this to measure the depth of the uterus before inserting an intra-uterine device (IUD) or a small suction curette in post-menopausal bleeding (PMB). A sound is a dangerous instrument in a recently pregnant uterus, because you can easily perforate it.
RETRACTORS, Langenbeck (large). Useful as well for a mini-laparotomy.
FORCEPS, Babcock (small). Use these to grasp a Fallopian tube while performing a tubal ligation or operating for an ectopic gestation.
FORCEPS, ovum, curved, screw joint, McClintock, 250mm. Use these to remove the products of conception from an incomplete miscarriage, after you have dilated the cervix. If you don't have them, use sponge-holding forceps.
CURETTE, uterine, double-ended, blunt and sharp, 8mm and 5mm. The great danger with a curette is that you may push it through the wall of the uterus, especially a recently pregnant uterus. Let a curette lie gently in your fingers, so that you can feel the wall of the uterus: don't grasp it firmly.
CURETTE, suction, stainless steel, reusable, sizes 8 and 10 Hegar. Use this for evacuating a hydatidiform mole (it causes much less bleeding than dilatation and non-suction curettage), and for terminating a pregnancy which has lasted <12wks.
KARMAN'S CANNULA, plastic, size 4-12mm. These are better than a curette. Use small sizes for dysfunctional uterine bleeding (DUB, 23.3) or PMB, and larger sizes for retained pieces of placenta of 22-42wks, or a very large molar pregnancy. The in-between sizes are very useful for removing retained products of conception from the uterus, best with the help of an electrical suction machine.
CURETTE AND SYRINGE for manual vacuum aspiration (MVA), sterile, plastic, disposable (but boilable though not autoclavable). They are also very useful for non-septic incomplete miscarriages seen at the outpatient department if there is no suction machine there.
CANNULA, cervical, Leech Wilkinson or Miller. This is for doing a hysterosalpingogram (HSG, 38.1).
SCISSORS, episiotomy, Vant. These have straight blades and round points.
VACUUM EXTRACTOR (VE), Bird's modification of Malmstrom's, complete with 3 suction cups 40, 50, 60mm, one posterior cup, traction handle, vacuum hand pump, chain, spare vacuum bottle and spare baskets. Bird's modification is better than the original Malmstrom extractor, and is quicker and easier to assemble. The anterior and posterior cups are not absolutely necessary. An expensive disposable KIWI will give you a luxurious feeling but the old-fashioned well-maintained Bird's is good enough. You need several sets of cups and tubes because you should be able to do 2-6 vaginal examinations in a weekend. Good simple maintenance of this basic equipment can make the difference between life and death for mothers and babies.
FORCEPS, outlet, Wrigley. Outlet forceps are the only safe ones for anyone but an experienced obstetrician. Even then, becoming a master with the vacuum extractor is easier and better.
FORCEPS, haemostatic, straight, Green-Armytage, 203 mm. Use these for clamping the (digitally) cut edges of the uterus during a Caesarean section (the faster you become, the less you need them), and for repairing a ruptured uterus.
STURDY FOLEY'S CATHETER with balloon content >25ml. Use this to deliver a dead breech presentation (21.8). Introduce the catheter *via* the foetal rectum, inflate the balloon and then traction is very easy.
PERFORATOR, Simpson's. This is the standard instrument for opening the skull when doing a destructive operation. If these are not available, Kochers forceps (large, straight or curved) will also do, or even large artery forceps.
RETRACTOR, Doyen's. Use this for Caesarean section; it has a curved lip which fits over the lower end of the wound and keeps the bladder out of the way of the operation.
RETRACTOR, Kirschner. This gives an excellent exposure for laparotomy, with a good view for operating in the pelvis.
SAW, decapitation, Blond-Heidler, complete with ring, thimble and blades. Use this for decapitating a dead baby when labour is obstructed by a transverse lie. It is a piece of wire with teeth on it, hooks at each end to fit handles, and pieces of tubing to prevent it from cutting his mother. It also has a thimble you can push round his neck to fix the saw.
SCISSORS, embryotomy, Queen Charlotte's pattern. Use these scissors, which were specifically designed for destructive operations, if you don't have a saw.

19.3 Infertility

Infertility causes much distress, particularly in those districts of Africa where true incidence is unknown and the subject is surrounded by much taboo. Some 30% of women aged 25-49yrs suffer from secondary infertility (failure to conceive after the first pregnancy). You may decide in your district hospital that you have other priorities, and that infertility investigation and treatment is so unrewarding that you are not going to try. If you do decide to assist, make it part of your family planning activities and promote an integrated 'fertility service', which is concerned with both too much and too little fertility.

N.B. Couples with HIV infection are more likely to suffer from infertility. Reasonably, this, like other STIs, needs treatment first.

During a normal menstrual cycle, between a couple staying together where the woman is ≤32yrs, there should be a 25% chance of pregnancy when sex is ideally timed. This percentage drops as the woman becomes older. After 1yr of normal sexual intercourse on random days, a couple should conceive. However, this data is based on statistical probability and may not apply in the individual case. Nonetheless, in normal circumstances, it is reasonable to start investigations after 1yr. *You must start with the husband! Always enquire about previous pregnancies!*

Take a sexual and menstrual history. If the woman has regular cycles, she is almost certainly (95%) ovulating. Failure to ovulate is typically associated with irregular cycles (normal cycles are 25-35 days long) or amenorrhoea. An irregular cycle will, however, in 75% of cases, still be associated with ovulation. If you are not sure, you can ask her to keep a temperature chart. She may be sufficiently educated and motivated to do this, particularly if she is a member of the hospital staff or a teacher. A sustained 0·4ºC temperature rise for 14 days before the start of menstruation is good evidence that she is ovulating; if the rise continues, she might very well be pregnant. Absence of this rise, especially if her periods are irregular or scanty, is reasonable evidence that she is not ovulating. This test will of course not work if she has a pyrexial illness including malaria, TB or HIV.

(a) Management

(1) Perform a post-coital examination: examine the mucus from inside the cervix.

N.B. With clomiphene treatment, especially in high doses, later than day 7 of the cycle, this test is not reliable.

(2) Perform a uterine curettage during the second half of the cycle, or better, the 1st day of the period, and send the scrapings for histology.

(3) Examine the ovaries laparoscopically (19.5) to see if they have a corpus luteum with a scar (stigma), showing that ovulation has occurred. At the same time, test the patency of the tubes, by injecting blue dye though the cervix, and seeing if it appears in the peritoneal cavity.

(4) With vaginal ultrasound, easily follow a follicle in growth and disappearance.

(5) Measure progesterone: a high level 1wk after the presumed ovulation more or less proves it has occurred.

(6) Check urine luteinizing hormone (LH) levels daily using commercially available test kits at the time of an expected ovulation. An LH peak predicts an ovulation within 1 (35%) - 2 (60%) days.

You can classify anovulation into 4 groups:

Group; Frequency (%)	**FSH level**	**Oestrogens**	**Abnormality**	**Therapy (Pregnancy chance %)**
1.Hypogonadotropic 10%	Low	Low	Hypothalamic -pituitary	Pulsatile gonadotropin release hormone (80%) or gonadotropins
2.Normogonadotropic 75%	Nor-mal	Normal	Hypothalamic -pituitary-ovarian	Clomiphene (50%), weight optimization (20%), gonadotropins
3.Hypergonadotropic 10%	High	Low	Ovary	None
4.Hyperprolactinaemic 5%	Low	Low	Pituitary	Bromocriptine/ cabergoline (65%)

N.B. You will probably not be able to treat women in groups 1 and 3, but it is nevertheless a good idea to advise the contraceptive pill (for growth/maintenance of the breasts, to prevent osteoporosis and to have monthly periods if they so wish). *Remember the side-effects of the contraceptive, though!*

Clomiphene is only indicated for anovulatory infertility. Warn of the increased incidence of multiple pregnancies. The dose is 50mg od from the 3rd-7th day of the menstrual cycle, or at any time if cycles have stopped, to a maximum of 12 courses. Monitor ovulation with a temperature chart. If she does not ovulate, increase the dose by 50mg amounts each month, to a maximum of 200mg od for 5 days.

N.B. CAUTION with clomiphene:

(1) *Only use clomiphene to HIV-ve women with patent Fallopian tubes and fertile husbands. Don't use it randomly on all infertile patients*.

(2) It is contraindicated in hepatic disease, ovarian cysts, pregnancy, and abnormal uterine bleeding.

(3) Side-effects include visual disturbances, hot flushes, nausea, vomiting, depression, insomnia, breast tenderness, weight gain, rashes, dizziness and hair loss. It may make the ovaries tender and temporary cystic, and can simulate an acute abdomen.

Ideally only if your laboratory can determine prolactin levels should you treat with bromocriptine.

The combination of lactorrhoea, amenorrhoea and infertility might, when prolactin testing is unavailable, prompt you to try bromocriptine 2·5mg od.

Gonadotropins have no place in district gynaecology.

(b) Blocked Fallopian tubes from adhesions and that follow PID or perhaps a septic miscarriage, tuberculosis (especially if HIV+ve) or rarely schistosomiasis, cause c.60% of infertility.

Anti-TB medication is unlikely to make a woman with endometritis fertile; she has c.8% chance of conception, but only 2% chance of a live child. She also runs an increased chance of an ectopic gestation.

Sometimes, you can make the diagnoses by histology from D&C scrapings on the 1st day of her period, or by seeing miliary TB at laparoscopy or laparotomy.

Repairing tubes that are blocked is an expert's task, and even then the success rate is low, so you may decide that there is little point in investigating these patients if they cannot be reliably referred. But sometimes it is very easy to open Fallopian tubes which are only closed at the distal end (like phimosis) using blunt small artery forceps. If *fimbriae* are then visible and the tube can be kept open by a few very small atraumatic nylon sutures, the prognosis is not that bad. *You must then warn about the symptoms and signs of an ectopic gestation*.

If you suspect blocked tubes, you can:
(1) Perform a hysterosalpingogram (HSG, 38.1l), which will tell you where a block starts and sometimes about a fibroid in the uterine cavity. This investigation is time-consuming and expensive, though.
(2) Insufflate the tubes, which is cheaper, but gives less reliable information. Also, the instrument can leak, and you can make mistakes.
(3) Perform a laparoscopy, which again is expensive and time-consuming.

(c) Fibroids (23.7) can be the cause of infertility especially if situated in the uterine cavity or when compressing the tubes in the cornua. *More often fibroids are the result of infertility*. Typically though, a woman with infertility and fibroids is also found to have blocked tubes or an infertile husband. Sometimes large or many fibroids cause repeated miscarriages and should be (partly) removed.

You might succeed by removing the largest, most distorting and other easily accessible pediculated fibroids, but you might also cause many adhesions which will subsequently interfere with fertility. Careful operating, keeping tissues moist, no rough swabbing, and closing the pelvic peritoneum will reduce such adhesions. Try to remove several fibroids through the same incision, and avoid wounds on the posterior surface of the uterus.

(d) In men:
Examine the seminal fluid within 2h of production. It is normal if: it has a volume of 2-6ml, it is liquid >30mins, it has 60% of motile sperms, and it has ≥20million sperms/ml, <15% being abnormal.

If there is a low sperm count, suggest abstinence from sexual intercourse until the 12th-14th day of the woman's cycle. Tight nylon underwear or other exposure to heat (*e.g.* sitting often on the engine of a truck, working in a bakery) and heavy smoking may be causes. Suggest loose cotton boxer shorts.

If there are pus cells in the ejaculate, treat the infection and of course his partner's. It can take 3 months after successful treatment before the sperm count improves.

Some men refuse to cooperate. Often the woman is not responsible, but she may well be sent away by the man if a pregnancy does not appear. The history is very important. If a woman is now with her 3rd partner and has had no children at all, it is unlikely that the man's semen is the problem, especially if he has children with other partners. Beware, though, of vague stories of men who say they have a child in a town 400km away! Sometimes these stories are backed up by his family to spare the man embarrassment.

The post-coital test will help if the husband refuses to cooperate or if your laboratory cannot do proper semen analysis. It also gives extra information about the quality around the ovulation of the cervical mucus (sticky, clear, elastic up to 6-8cm) and will also confirm that semen has been deposited in the right place!

Timing is very important. Only 1-3 days around the time of the ovulation (if present) do sperms enter the cervix and swim to the tubes; they may survive in the cervix for quite some time (2-3 days). It is satisfactory to examine the cervical mucus the morning after evening sexual intercourse.

Expose the cervix, use a syringe (without needle) to suck some mucus from *inside* the cervical canal, and put it on a microscope glass (not easy as it will tend to adhere to the syringe but the cover glass might help trapping the mucus). Examine under the microscope with 10x10 magnification. Progressively mobile sperms will more or less exclude a male factor, hostile cervical mucus (a rare cause for infertility), and a faulty sexual technique. If the test is -ve, repeat it, try, if necessary, on consecutive days to catch the ovulation.

N.B. In some cultures it is accepted that a woman becomes impregnated secretly by a male relative of the husband, if he is azoospermic. *Note the risks of tramsmission of HIV and other diseases by this practice.*

(e) In women:
(1) UTERINE CURETTAGE (GRADE 1.3)
Don't do this if periods are regular or she is taking the contraceptive pill!
Perform the test very early when her periods start. Preferably use a microcurette, as an outpatient. Otherwise, perform a D&C (23.4). Send half the curettings for histology, indicating the request is for ovulation, and the other half for TB investigation.

(2) TUBAL INSUFFLATION (GRADE 1.4)
INDICATION. Although this procedure is theoretically simple, false results are not uncommon. If insufflation is the only method of investigation you have, this suggests that expert tubal surgery is unlikely to be available, which should make you question the value of insufflation.

METHOD
Place the patient in the lithotomy position. Insert the insufflator into the cervix and fill the vaginal canal with fluid, so that the end of the cannula and the cervix is submerged, and you can see if there is a leak. Introduce some carbon dioxide, and listen over the lower abdomen with a stethoscope for the sound of it bubbling out of the tubes. Measure the rise in P_{CO_2} before free flow occurs.

If the tubes are patent, pressure will peak, and flow occur <40mm Hg. If they are blocked, it may rise as high as 160mm Hg. You can also feel the difference with the plunger of the syringe. (If you are using air, use a maximum of 250ml, and don't allow a pressure >100mm Hg because of the risk of air embolism.) You might consider using normal saline; if you can inject 20ml easily, at least one tube is open or you might have opened up one tube.

N.B. Ultrasound can detect passage of liquid out of the tubes.

(3) LAPAROSCOPY AND DYE INJECTION (GRADE 2.3)
Under GA, insert a Miller cannula into the cervix. Insert a laparoscope abdominally, as for tubal ligation (19.4), and tilt the head down until you see a good view of the pelvis. If you cannot see clearly, insert a blunt-ended hook in the midline suprapubically, and use this to manipulate the tubes. Alternatively, manipulate the vulsellum forceps (hold it vertical and then push it down) on the cervix to anteflex the uterus and make the tubes visible. Inject 10-20ml of methylene blue dye diluted 1:10 in sterile water *via* the cervix, and look for dye spilling from the ends of the tubes. If blue is not available, water-based *(non-soapy)* betadine will also work but is more difficult to see.

Normal tubes: the *fimbriae* look healthy and the dye spills through easily. It may spill on one side only (least resistance), but if both tubes look healthy, they are probably both patent.
Cornual block: No dye enters the tubes. As your assistant injects the dye, the junction of the tube insertion into the uterus blanches or colours slightly.
Fimbrial block: The tubes are often distended; their *fimbriae* are clubbed and sealed over the ostia, and may be adherent to the ovaries. As you inject the dye, the thin walls of the tubes allow you to see it entering them. Usually, no dye spills out. Sometimes the fimbrial block is partial, so that only a little spills.

19.4 Tubal ligation (TL)

This should be one of the most common operations you do and many women and couples will be very grateful for it. Choose it after careful consideration of the alternatives (a copper IUD is good for 8yrs and nearly as effective, a hormone IUD is good for 5yrs and at least as effective but expensive), so your operation must be as safe and as painless as it can be. Try not to keep a mother waiting long for surgery, or she may become pregnant meanwhile!

Large numbers of mothers would like their tubes tied, and if you take the trouble to encourage them, many will be willing to accept it. But however many ligations you do, you will probably be only able to satisfy a small fraction of the community's need.

You can:
(1) Tie a woman's tubes at the same time that you perform a Caesarean section (21.9).
(2) Perform a 'mini-laparotomy', which is a laparotomy through a very small incision.
(3) Occlude the tubes through a laparoscope.

Tying the tubes immediately after delivery (and to some extent after a miscarriage) has several advantages:
(1) They are easier to access when the uterus is still enlarged.
(2) The patient will already be in hospital, whereas if you discharge her and ask her to come back, she may never return.
(3) Immediately after a normal delivery/miscarriage she will tolerate the minimal additional trauma of sterilization particularly well.
(4) If you have already opened the abdomen for some other reason, including Caesarean section (or perforation after a backstreet termination), tying the tubes is easy.

There are, however, some minor disadvantages in doing it at this time: (a) She may change her mind later, though in practice this is rare. (b) You may have not been able to exclude a serious abnormality in the newborn beforehand.

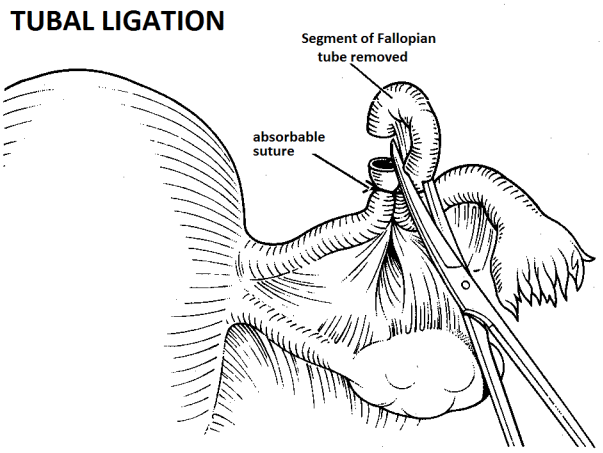


Fig. 19-2 TUBAL LIGATION.
Tie a loop of Fallopian tube and excise it. You can also use rings and clips.

INDICATIONS
(1) Mothers who are sure they want no more babies.
(2) Medical diseases making pregnancy dangerous, particularly severe heart disease, renal failure, HIV disease or severe diabetes.
(3) Previous Caesarean section where another Caesarean section would be impossible to organize.
(4) Ectopic gestation in a multipara.
(5) Severe psychiatric disease.

CONTRAINDICATIONS
(1) Extreme obesity.
(2) Excessive anxiety.
(3) A history of PID causing severe adhesions to the uterus of a patient not in the immediate post-partum period.
(4) Infertility.
(5) Pregnancy.
(6) Refusal to give consent.

CAUTION! Informed consent is essential, but does not usually legally need the agreement of the husband, although in some cultures leaving the husband out of the equation is unacceptable. Obviously it is better to discuss the option of TL early in pregnancy, but this does not mean you cannot introduce the subject during labour (especially if you have had no other opportunity), especially if you plan a Caesarean section.

A solution is to have a pre-printed area on every antenatal clinic card where nurses or midwives could record, early in the pregnancy, "wishes for a TL". This is especially important for a woman >30yrs who is para >2.

If a woman is enthusiastic at once and she knows what you are talking about, it is patronizing not to let her make use of the opportunity. Indeed, if you have to perform an emergency Caesarean section, *e.g.* for failure to progress in a para 4 woman who presented late in hospital, but you deliver a well baby, it is quite possible that any subsequent regret for consenting to TL is far outweighed by the chance of death in the next pregnancy (from uterine rupture, *placenta accreta*, haemorrhage during a difficult Caesarean section etc.).

In fact, most multipara regret not having been given the option of TL at Caesarean section. If in doubt, women will choose not to have a TL. There is no proof that women in Africa choose recklessly in favour of a TL.

DUDUZILE, a 40yr-old para 7, arrived in shock in the nick of time from a remote area with a bleeding ectopic gestation. A junior doctor operated on her, removed the affected tube but did not tie the other tube; he thought it unethical. When she understood that the next morning, she refused to leave the hospital till the other tube was likewise tied. She remembered her fear of dying and wished never to go through the same ordeal again.
LESSON After one ectopic gestation with a contralateral tube open, the chance of a further ectopic gestation is as high as 20-30%.

ARRANGEMENTS. It is best to perform TLs on a special operating list, and perform them early, so they are not displaced by other elective operations and emergencies. You may be able to run a 'TL camp' or set up adequate preparations at a health centre. Normally intervention under LA, with or without some sedation, is possible.

METHOD (GRADE 2.3)
Immediately before the operation, ask the patient to pass urine, to prevent you cutting into the distended bladder. *This is important*. Perform a careful bimanual examination to make sure that she is not already pregnant (although you can tie tubes during pregnancy without significant extra risk, to prevent the next pregnancy).

Use the semi-lithotomy position, with the thighs flexed to 45º and moderately abducted, the knees flexed, and the lower legs horizontal. Use Lloyd-Davies stirrups, or the cheaper 'Chogoria' supports (19-3). Make sure you can put her in a head-down tilt and that she does not slide off the table!

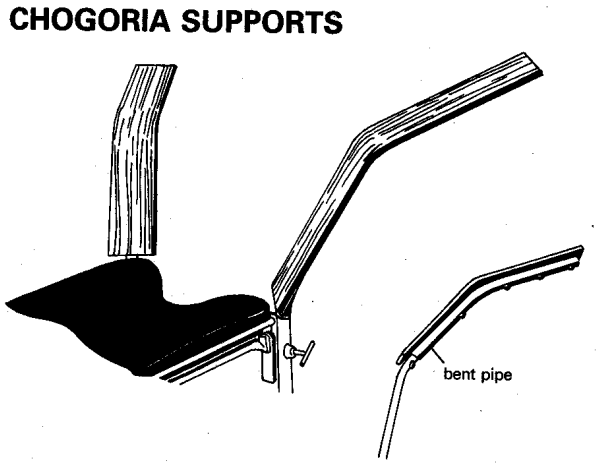


Fig. 19-3 CHOGORIA SUPPORTS hold the legs only partly flexed, so that you can have simultaneous access to the abdomen and the perineum. They are from a mission hospital of this name in Kenya, and are a cheaper locally made alternative to Lloyd-Davies stirrups, or to an attachment for an operating table that enables you to angle its lithotomy poles.

Clean the abdomen, perineum, and vagina, empty the bladder with a catheter (if it is not empty already), and cover her with an abdominal sheet. Pass a Sims or Auvard's speculum.

If >10 days have elapsed since the 1st day of the last period, consider doing a D&C or suction curettage (23.4), to prevent implantation in this cycle. You may need paracervical anaesthesia.

INCISION depends on the position of the fundus.
If delivery has occurred within 4 days, and the uterus is at the umbilicus or can easily be pushed there, make a 2cm horizontal incision in the inferior fold of the umbilicus. This is good cosmetically, and avoids the need for shaving. Also, the abdominal wall is thinnest here.

If she is not postpartum, make a short transverse incision just above the pubic hair. If you have inserted a uterine manipulator, moving it helps you to decide where to operate exactly. Ask an assistant, with the manipulator, to raise the fundus against the abdominal wall until you see and feel a bulge (19-4A). Alternatively, manipulate it yourself through the towels (experienced doctors can do this operation if need be without an assistant).

If you make an umbilical incision, stretch it and make a 2cm horizontal incision in its inferior fold. Spread (*don't cut*) the subcutaneous tissue moving laterally with scissors until you see the fascia. Insert 2 small narrow right-angled retractors, and pull them apart laterally, while using them to pull aside all tissues between the skin and the fascia.

Pick up the fascia between two haemostats (one as much as possible to the right and one as much as possible to the left), and inject another 5ml lignocaine just beneath the fascia to anaesthetize the peritoneum. Open the fascia horizontally with a knife over the ridge you create when you lift the haemostats. You will find the fascia and the peritoneum fused so lifting it up prevents you cutting too deep.
If the peritoneum is not open yet, do it bluntly by pushing closed forceps or scissors through it and pulling this instrument out in open position. The incision in the fascia should be large enough to admit your index finger. The skin will stretch, so you can make the skin incision shorter than the fascial one.

If you make a suprapubic incision, you have a similar approach but the incision is somewhat larger (reverse correlation with your experience). Enter the peritoneum as high as possible in your incision to stay away from the bladder.

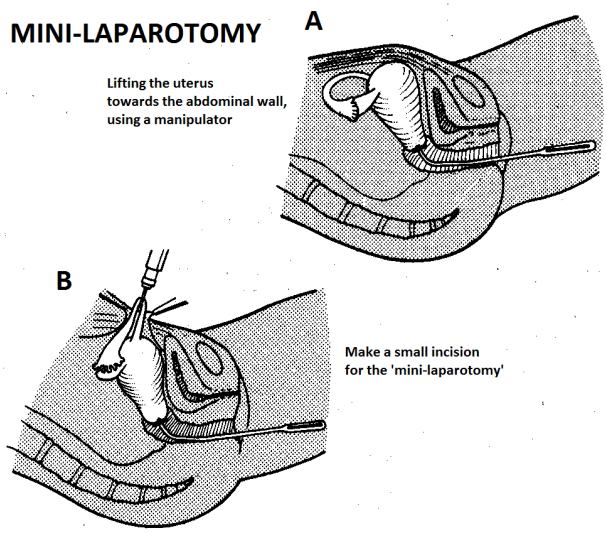


Fig. 19-4 MINI-LAPAROTOMY.
Use a special manipulator to push the fundus up against the abdominal wall, so that a very small incision can be made in the abdomen. *Don't use this immediately postpartum or post miscarriage.*

After delivery, use your finger to locate a tube and sweep it from behind the uterus medially, visualize it and grasp it. You can easily move the uterus from left to right by pushing against it through the abdominal wall to let the tubes approach the umbilicus. Alternatively, the incision can be moved with the retractors from left to right.

In the elective situation, using your finger is not such a good idea. It tends to hurt the patient, causing her to push. Bowel then appears in your incision and you can get stressed, operate more roughly, cause more pain, and so more pushing. Try to visualize the tubes and then pick them up (as distally as possible) with Babcock forceps. Your chances of seeing them increase with an easily directed light, a steep head-down tilt, a manipulator, the availability of a special instrument to fish out the tubes (a small ring on the end of a strait wire attached to a handle), patience, and an assistant talking to the patient, helping her to relax and breathe deeply. If all else fails, 100mg ketamine IV at this stage will help.

N.B. The tube is the middle (and top one) of the three lateral attachments to the fundus (dorsal ovarium, ventral round ligament) and has a lumen.

Introduce some LA between the two blades of the mesosalpinx near the tube where you plan to tie it. Excise a segment, having ligated a loop (19-2).

CAUTION! Check very carefully that there is no bleeding, cut the sutures on the tube, and then operate on the other tube in the same way.

Close the fascia with continuous #1 monofilament. Close the subcutaneous dead space to minimize oozing. Close the skin with subcuticular absorbable.

DIFFICULTIES WITH A MINI-LAPAROTOMY

If there is pain after you injected the LA, add sedation. A wide area of LA should prevent this.

In obesity, it will be difficult to pull the tubes into view through a layer of fat. Enlarge the incision and apply more head-down tilt. An umbilical incision may be easier than you expect, because there is less fat around it.

If you cannot find the tubes:
(1) The incision may be too far above the fundus; it should be slightly below it. Turning the uterus with your finger behind might help if you are desperate. You may find it helpful not to release the first tube, until you have moved across the fundus and found the other one. Try passing Cusco's speculum through the incision to help you look around.
(2) The uterus may be stuck down with adhesions. A careful initial pelvic examination should have excluded this. Dense adhesions require GA to enable you to operate safely. If the tubes are adherent to the uterus or the pelvis, you may have to make a standard incision, or abandon the operation. This is particularly likely to happen if there are adhesions following Caesarean section.

If you find any ovarian cysts, leave them if they are <5cm. Often normal ovaries have some physiological cysts. If a cyst is larger or a possible dermoid, collapse it by draining it with a syringe and needle, pull it into the wound, and excise it.

If you open the bladder, close it with absorbable sutures in two layers, and leave a catheter *in situ* for 7 days. Prevent a full bladder by having her empty it just before she enters the theatre. If you find it full at surgery, empty it with a catheter or a needle and syringe.

If you open the bowel, close it in two layers transversely, and observe closely for signs of sepsis, abdominal tenderness or distension suggesting a bowel leak.

Once you have mastered the mini-laparotomy technique, you can also use it for ectopic gestation (or ovarian cysts).

19.5 Using a laparoscope

A standard laparoscope is a <1cm diameter tube which you insert through a tiny incision near the umbilicus, and which you can use to inspect the abdomen. You can also perform a variety of minor operations through it, including tying the tubes.

Because a standard laparoscope with its associated equipment is fragile and expensive, a simpler and more robust instrument, the 'laprocator' (19-5), is used by the Johns Hopkins Program of International Education in its Gynaecology and Obstetrics (JHPIEGO) programme, and is specially adapted for use under difficult conditions. It is only suitable for tubal ligation, removal of a perforating IUD and diagnostic inspection of the peritoneal cavity with tissue biopsies, but not for the other procedures which are possible with a standard laparoscope. Unfortunately, like a standard laparoscope, it also needs special training, which is usually given at JHPIEGO courses, a laprocator being given free to all those who pass the course, and who can demonstrate that they have adequate facilities. It is described here, so that it becomes more widely known and it might be that you have experience in laparoscopy and you find a laprocator in a cupboard in your hospital and plan to use it. A laprocator is robust, reliable, and relatively inexpensive, and is popular with patients. You can use it with LA, but you may find using it more convenient with ketamine. You can use a cylinder of carbon dioxide but because you are not using diathermy inside the abdomen, you can use air instead. However, if you use carbon dioxide and not air, there is no risk of air embolism.

Alternatively, you can use a cystoscope (19-6), insufflating the abdomen with a standard sphygmomanometer cuff, for diagnostic purposes, using the working port for the biopsy instrument, but this does not allow you to perform a tubal ligation.

If you are skilled and have a good team, laparoscopic ligation is quick, and safe, and can be done on outpatients. The incision is so small that it soon becomes almost invisible. Because you use rings instead of diathermy, you will not easily injure the bowel.

There are disadvantages. A laprocator is still quite delicate, and the possible complications include perforation, air embolism, and bleeding, and introduction of infection if the instrument is improperly sterilized.

You can introduce the laprocator through a small laparotomy incision, or you can use a special trocar in combination with a special spring-loaded (Veress) needle to introduce the gas. If you are a beginner, start with the open laparoscopy method, which is safer and does not need a gas supply. The only disadvantage of the open method is that the skin incision is slightly longer, and needs two sutures instead of one.

(a) Laparoscopy has caught the imagination of doctors and patients. If you demonstrate it at health education talks, you can be sure that some mothers will come forward afterwards to have their tubes tied.

INDICATIONS
(1) Sterilization.
(2) Diagnosis of PID, endometriosis, and infertility.

N.B. A laparoscope is not much use in the diagnosis of ectopic gestation. The diagnosis is usually obvious anyway by the time the patient presents and you won't see anything but blood through your scope.
By doing a mini-laparotomy, you not only confirm the diagnosis but you can also repair the damage and perhaps collect blood for autotransfusion (5.3) without wasting time.

CONTRAINDICATIONS
(1) Most lower abdominal scars. However, if you are experienced, you can perform a laparoscopy if the scar was for a lower-segment Caesarean section, because it seldom causes adhesions between the bowel and the abdominal wall.
(2) A history of chronic PID with possible adhesions.
(3) Extreme obesity.

N.B. Mild obesity is an indication for laparoscopy, because the incision does not have to be larger if a patient is mildly obese, as it does in a mini-laparotomy.

PREPARATION
Place the patient into the semilithotomy position, as for a mini-laparotomy. Clean the abdomen, perineum, and vagina. Let the patient empty the bladder just before the operation. Pass a uterine manipulator or vulsellum and attach it to the cervix. Move the uterus up to the abdominal wall. Wait until she is relaxed and not coughing. Tilt the head downwards.

USING THE VERESS GAS NEEDLE (GRADE 2.3)
If you are right-handed, stand on the patient's left. Make a small cut in the skin of the lower border of the umbilicus, and dissect down till you can see the peritoneal layer. Lift this up and, holding the abdominal wall with your left hand, with your right hand insert the Veress needle through the peritoneum almost at right angles to the skin, pointing it in the direction of the pouch of Douglas. Hold it by the barrel, so that the blunt trocar is free to slide up and allow the cutting needle to enter.

CAUTION! Use some of the following methods to check that the end of the Veress needle is indeed in the peritoneal cavity:
(1) You are able to move its point freely from side to side. Be careful as you do this, and *don't use force*, because you may tear adhesions.
(2) When you lift up the abdominal wall, you can hear air rushing in through the needle.
(3) A drop of saline, placed over the hub of the needle, is sucked in when lifting the abdominal wall.
(4) After injecting 5ml of saline through the needle, nothing can be sucked back.
(5) Gas flows freely into the peritoneal cavity with little resistance.
(6) A small volume of gas obliterates the normal dullness to percussion over the liver.

N.B. There will be a normal range of insufflation pressures for your machine, shown in green on the dial. If the pointer moves to the red area, the needle is probably in the wrong place.

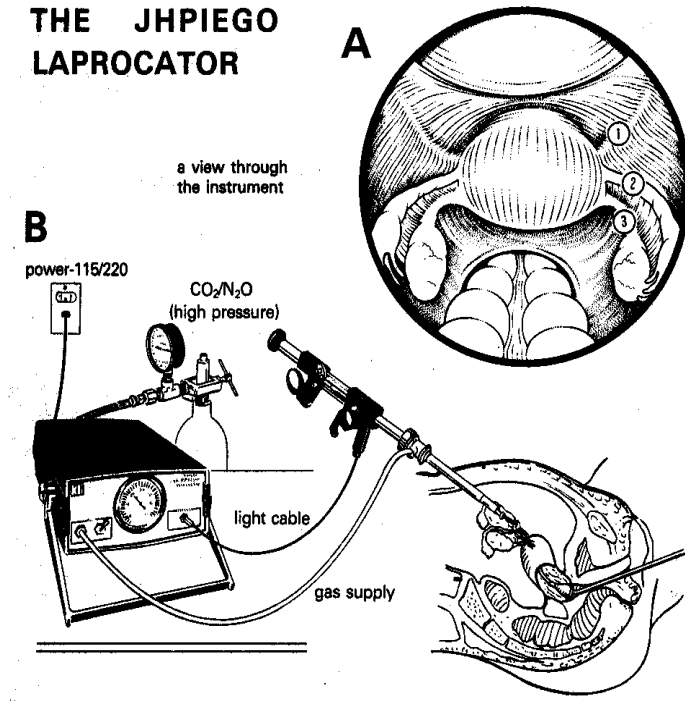


Fig. 19-5 THE JHPIEGO LAPROCATOR.
A, view through the eyepiece. B, instrument in use. (1) the round ligaments. (2) the tubes. (3) the ovarian ligaments.

Let the gas flow into the peritoneal cavity. A multipara who is being sterilized needs up to 4L (2L is usually enough). A nullipara who is having a laparoscopy for diagnosis needs 2-3L. Many insufflators don't measure volume, but carbon dioxide flows at the rate of c.1L/min, so allow it to flow for 2mins.

Remove the Veress needle, and enlarge the skin incision with a scalpel, until you have a 1·5cm horizontal incision at the lower border of the umbilicus. Insert the trocar and cannula. Push it in the same direction as you (should have) pushed the Veress needle. *If you haven't made the opening big enough, you will have to push quite hard, especially if the trocar is blunt: this is dangerous, and the trocar is difficult to control*. Keep it in the midline. When it is through the peritoneum, withdraw the trocar, and insert the cannula fully. Then insert the laparoscope. Connect the gas tube to the cannula for insufflation.

Look for the Fallopian tubes. *Make sure they are indeed the tubes by confirming that they*:

(1) join the uterus at the *cornua*, whereas the round and ovarian ligaments join below the *cornua*.

(2) are in the middle behind the round ligaments and in front of the ovarian ligaments.

(3) end in *fimbriae.*

(4) easily form a loop when pulled up, much more easily than the round or ovarian ligaments.

N.B. You can clean the laparoscope lens by gently wiping it on tissues within the abdomen, but beware: *a long contact may burn tissues if the bulb is hot*, and it may be safer to take it out and clean the lens with a warm moist gauze.

If you have difficulty manipulating the tubes, try inserting the gas needle in the midline 5cm below the umbilicus *under direct vision of the laparoscope*. Use the needle to help you manipulate the tubes. Otherwise push the cervix down using a vulsellum forceps and so elevate the fundus and identify the tubes.

Apply one ring to each tube. Withdraw the laparoscope. Open the valve to expel the gas, and remove the cannula. Close the skin with one suture or a skin clip.

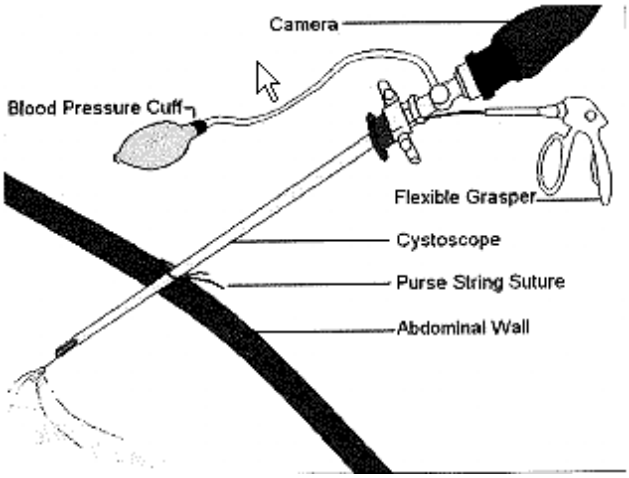


Fig. 19-6 USING THE CYSTOSCOPE FOR LAPAROSCOPY.
Fix the cystoscope with a purse-string suture on the abdominal wall. Insufflate with air using a sphygmomanometer cuff. Use the biopsy channel for instrumentation.
After Gnanaraj J, Diagnostic laparoscopies in rural areas: a different use for the cystoscope. Tropical Doctor 2010(3):156.

(b) Open laparoscopy with the laprocator

METHOD. Proceed as above under direct vision, so you are able to introduce the laprocator with its cannula, but without its trocar. Use 2 towel clips to tighten the skin around it and prevent gas leaking.

Fill the peritoneum with 2L of air. The laprocator control box has a small air reservoir which is filled by a rubber pump. Air is only slowly absorbed, so take care to let it all out when you have finished. If you allow air to get into the wrong place, for example into the extraperitoneal tissues, it will obscure the view. You will not be able to try again after a few minutes, because air takes hours to be reabsorbed, unlike carbon dioxide which is quickly absorbed.

(c) Difficulties with the laprocator

If there is extensive bleeding, cross-match blood, perform a laparotomy and search for the source of bleeding (usually mesenteric vessels). Pack the area if the source is not immediately visible, and then after 5mins, carefully remove the packs to look again.

If you cannot see the tubes, try the manoeuvres described; if these fail, perform a laparotomy.

If you mistakenly put a ring on something which is *not* the tube (e.g. the appendix, round ligament or even ureter), you can usually pull it off again by catching its edge with

one prong of the laprocator forceps. If this fails, perform a laparotomy: *don't assume the ring will not cause harm!*

If you perforate bowel with the trocar, perform a laparotomy and oversew the perforation with two layers of 2/0 absorbable (14.3).

If you perforate the bowel with the insufflations needle, observe the patient closely. Unless peritonitis develops, you don't need to perform a laparotomy.

19.6 Vasectomy

Although a vasectomy is a simple operation, it must be done well, because its success as a family planning procedure depends on there being very few side-effects.

The normal vas is about 2·5mm in diameter. When you pinch it between your finger and thumb, it has a characteristic firm cord-like feel. It is difficult to feel immediately behind the testis, but between the upper pole of the testis and the inguinal ring you can feel it quite easily, and deliver several centimetres of it through a small incision in the scrotum. Rarely, it is double, which is one reason why vasectomy occasionally fails.

After you have incised the skin, you will meet the superficial fascia containing the *dartos* muscle. Deep to this lies the connective tissue which surrounds the spermatic cord. When you reach the vas, you will find that this also has a sheath of its own. Take care:
(1) *Don't injure the veins of the spermatic cord* (the pampiniform plexus), which will bleed during the operation, and possibly afterwards also.
(2) *Don't tie the testicular artery*, or the testis will atrophy.

FORCEPS, vasectomy. Get these if you plan to do many vasectomies.

CONTRAINDICATIONS to vasectomy as an outpatient include: a varicocele, a large hydrocoele, a local scar, an inguinal hernia, genital tract infection, diabetes, recent coronary heart disease, and filariasis.

Don't do this procedure if the family situation is unstable, the man has fathered fewer than 2 children or he is <30yrs old.

Always examine the scrotum before you advise a vasectomy: *you may not be able to feel the vas!* There may be a double vas present on one side! There may be significant pathology there, *e.g.* condylomata.

PREPARATION
Explain the purpose of the procedure is to prevent sperms reaching the ejaculate; emphasize that there is no effect on libido. Advise that this is a permanent measure: reversal is extremely difficult even with microscopic techniques.

Inform the patient that it will take c.12 ejaculates to clear all live sperms distal to the vasectomy, and so he cannot assume he is sterile immediately after the procedure. Tell him that very occasionally the vas can rejoin. Get him to sign to an appropriate consent form which includes these points.

Ask him to soak in a bath and shave the scrotum before the operation, and bring with him a tight-fitting undergarment to support it afterwards. Take careful aseptic precautions.

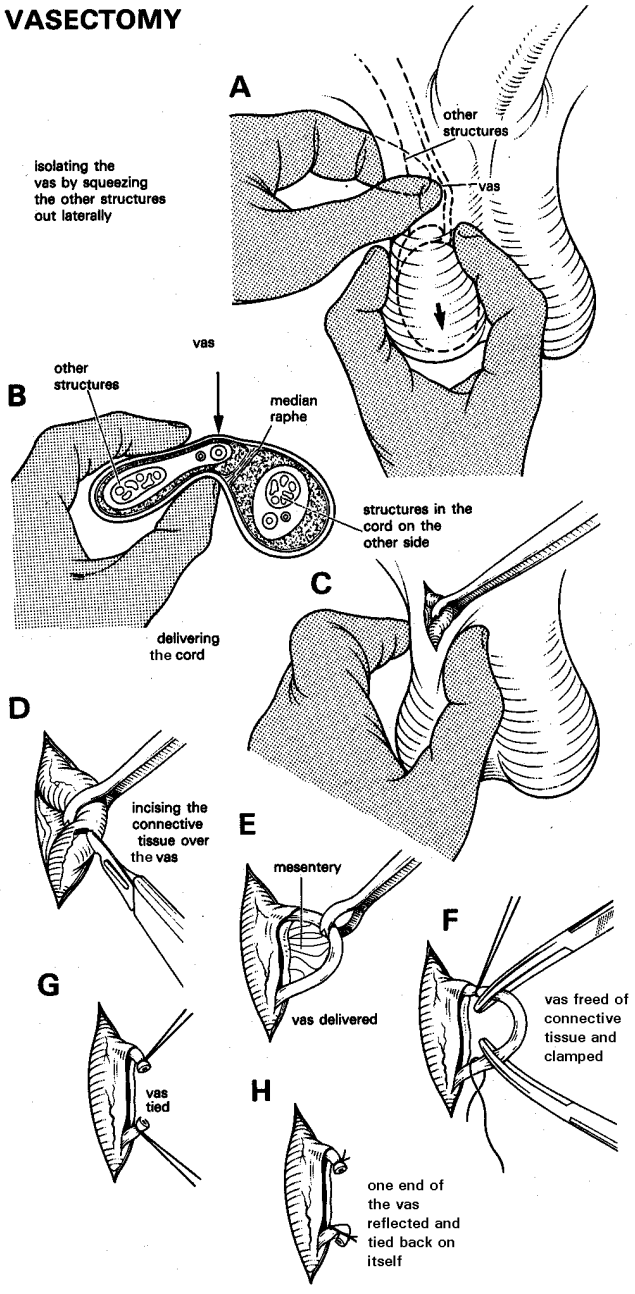


Fig. 19-7 VASECTOMY.
A-C, isolate the vas from the other structures in the cord. B, *N.B.* the pampiniform plexus and testicular artery are in the 'other structures'. D, incise over the vas. E, deliver the vas. F, free and clamp the vas. G, tie the vas. H, reflect one cut end of the vas.

VASECTOMY (GRADE 2.1)
Stand on the right. Find the vas where it is easily palpable in the scrotum. Pull on the spermatic cord just above the testis, with the thumb and index finger of your right hand. Assuming you are right-handed, use the thumb and fingers

of your left hand to manipulate the cord, so as to push the vas upwards and laterally. Isolate the vas from the other structures, by squeezing them out of the way (19-7A).

Hold the vas well above the testis with your thumb over it and two fingers underneath it. If the skin is thin, you will be able to see it. Pulling on it will cause him some discomfort, and pain referred to the abdomen. This is a useful sign that you have indeed found it (19-7B).

CAUTION! Make sure you have isolated and anchored the vas in the manner described. This is the critical step. Doing it without causing discomfort needs practice.

ANAESTHESIA
With the vas now anchored, find an area in the skin which is free of cutaneous blood vessels, and use 1% lignocaine to raise a small wheal. Then push the needle deeper and inject 1-2ml as close to the vas as you can, while holding it away from the other structures in the cord. If he has persistent discomfort while you are handling it, inject more solution into its sheath.

CAUTION! Don't infiltrate the other structures in the cord. This is unnecessary and dangerous, because you may injure the pampiniform plexus. If there is adrenaline in the anaesthetic solution, it may constrict the vessels, and make the testis temporarily ischaemic and painful.

DELIVERING THE VAS. While still firmly anchoring the vas, incise the skin 1cm over it transversely down onto the vas. If you inadvertently divide the vas, it is not a problem! Grasp the vas with the forceps (an Allis tissue forceps can substitute for a special vasectomy forceps) (19-7C).

If you cannot lift out the vas, gently cut deeper or push the tip of mosquito forceps through the incision, and split the *dartos* vertically. Then push the vasectomy forceps into the incision and lift out the vas. Confirm that the vas has not slipped away by feeling it with these forceps: it has a characteristic feel, and you will see the tiny lumen of the vas when you cut through it.

CAUTION! Don't mistake the vas for thickened bands of cremaster muscle, thrombosed veins, thickened lymphatics, or calcified worms.

ISOLATE THE VAS FROM ITS SHEATH by levering the tip of the forceps upwards by lowering its handle. Use a #15 blade to incise the connective tissue over the vas vertically in line with it. Make sure that the connective tissue is completely divided by continuing the incision into the vas itself (19-7D).

Hold a segment of the exposed vas with another forceps. If you are confident this is indeed the vas, release the first forceps. If you have judged the site and depth of your incision correctly, you can now easily pull out the vas, leaving only a thin mesentery on its medial surface (19-7E).

Use mosquito forceps to make a small window in a piece of the mesentery of the vas which is free of blood vessels. Isolate a 1-3cm segment of vas between clamps keeping far away from the epididymis. Tie its clamped ends with absorbable suture, placing your ties beyond the clamped area (19-7F). Leave one end outside the connective tissue by suturing it to the outside sheath and the other end reflected inside in order to minimize the risk of natural re-anastomosis.

Excise the isolated segment (19-7G), and keep it for histological examination. You may not need to send this but it is worthwhile keeping in case there are subsequent problems.

CAUTION!
(1) *Don't put the ligatures over the crushed area*.
(2) *Don't tie them too tight*, or they will cut out.
(3) To begin with, leave the ends of the sutures long, so that, if the cut ends of the vas bleed, you can pull them back into the wound. Pull on the testis to separate the ends of the vas. Inspect the wound. If it bleeds, pull out the ends of the vas, and tie any bleeding vessels with absorbable suture. Then cut the ends of the ligatures short and drop them back.

CAUTION!
(1) *Don't damage the pampiniform plexus*.
(2) Control all bleeding carefully. A small vessel can form a big haematoma later. Bleeding can also come from the skin edges, from the fascial sheath covering the vas, or from the pampiniform plexus.

If the incision is <1cm, the skin edges may come together without any sutures. Otherwise, suture them with catgut, *not long-lasting absorbable as the suture becomes mushy and often infected*.

Repeat the same procedure on the other side of the scrotum through a separate incision. You may prefer to move to the opposite side of the patient.

Place swabs on both wounds, and hold them with a crepe bandage tightly wound round the scrotum, held in place by tight underwear. *Don’t use adhesive tape on the scrotal skin!*

HAEMOSTASIS MUST BE ABSOLUTE

CHECKUP
Warn the patient that he may not become sterile for up to 3wks. His wife should continue to use a contraceptive:
(1) until 2 examinations of the ejaculate have shown no sperm, or
(2) until he has had 12 ejaculations after vasectomy.

To examine the ejaculate, ask him to produce a specimen by masturbation, or from a condom after intercourse. Examine this for sperms under a low-power microscope. There should be none.

If sperms are found, repeat the test after a further 3wks, advising precautions as above. If they are again found, re-explore the scrotum as below.

DIFFICULTIES WITH VASECTOMY

If you cannot find the vas, *don't continue the operation under LA* as an outpatient. The patient will need some sedation.

If you lose the cut ends of the vas after dividing it, the ligature may have slipped, or you may have released the forceps holding the vas too soon, and let them be drawn quite a distance into the scrotum. *Don't injure any blood vessels.* If there is no bleeding, leave things well alone.
If there is bleeding, try to recover the ends by systematically palpating the vas, and feeling for its ends with forceps.
If you cannot find the cut ends, strap the scrotum tightly leaving the wound open and admit the patient for bed rest. Tell him that you have had difficulty, and watch for haematoma formation. Check later to see that the ejaculate becomes sperm-free.

If a haematoma forms, it may spread into the scrotum, the thighs, or the abdominal wall. If it is small, it will disappear spontaneously. If it is larger, you may have to admit him and evacuate it.

If deep infection in the scrotum develops, suspect HIV disease or diabetes and drain the sepsis as for Fournier's gangrene (6.23).

If histology fails to confirm vasectomy, explain that you need to re-operate under GA or ketamine.

If the patient's wife becomes pregnant, either vasectomy has failed, or he is not the father! If sperms are present in the ejaculate, you can either re-explore the scrotum under GA or ketamine, and perform the operation again, or offer the wife sterilization. If sperms are not present in the ejaculate, consider carefully what you should or should not tell him!

20 The surgery of pregnancy

20.1 Surgical problems in pregnancy

Train the staff in your clinics to manage most of the minor complications of pregnancy. In early pregnancy they should refer to you incomplete miscarriages, especially if septic (20.2), as well as ectopic pregnancies (20.6,7,8). Rarely, you may have to treat an abdominal gestation (20.9), a delayed miscarriage (20.2), or gestational trophoblastic disease (23.10).

Late in pregnancy, after the 24th week, your main concerns will be antepartum haemorrhage, from placenta praevia (20.11) or placental abruption (20.12). Both of these need differentiation from incidental bleeding from lower in the birth canal. Another problem will be a dead foetus, whose management before 12wks differs from that later on (20.4).

It is important to be clear about definitions. The following WHO categories are generally accepted:
Extremely low birth weight: <1,000g
Extremely preterm birth: ≥20, <28 completed weeks
Very low birth weight: <1,500g
Very preterm birth: ≥28, <32 completed weeks
Low birth weight: <2,500g.

Oocytes (=eggs) are stored in the ovaries (300,000 present at puberty); some mature and are then sometimes released (ovulation: maximum 500 over a lifetime) and of those, only a few, if any, are fertilized, after which they stop being called oocytes and become a **zygote**, which becomes an **embryo** after further division and later a **foetus**.
Implantation is attachment and penetration of the endometrium by the embryo (then also called a **blastocyst**) that starts 5-7 days after fertilization.
Gestational sac: a fluid-filled structure associated with early pregnancy which may be inside or outside (in case of ectopic gestation) the uterus. The total size, unlike crown-rump (*i.e.* head to buttock) length (CRL), is a poor indication for **gestational age**, which is the time since ovulation (or perhaps better, fertilization).
The **ultrasound-based definition of foetus** is where the foetal heart movement is seen as positive and/or the CRL is >1cm (38.3).
The **embryonic period** is the first 8 *post-fertilization* weeks (=10wks' gestational age on ultrasound) during which organogenesis takes place; after that mainly growth and reorganization occurs.
The **perinatal mortality** (PNM) is foetal or neonatal death occurring during late pregnancy (≥20wks), during labour or up to 7 completed days after birth.
The **perinatal mortality rate** (PNR) is counted as per 1,000 births (alive or dead).
An **empty sac** is an anembryonic pregnancy; *i.e*, a sac without clear structures and no foetal heart movement.
Gestational trophoblastic disease (GTD, 23.10): complete or incomplete, *i.e.* partial, hydatidiform mole, or molar gestation.
Heterotopic gestation is an intra-uterine plus an ectopic (tube, ovary, abdominal, cervix) gestation.
Gestation of unknown location defines a +ve pregnancy test but with localization not (yet) possible with ultrasound

20.2 Evacuating an incomplete or delayed miscarriage

SOME TERMINOLOGY

The nomenclature still often used for early pregnancy has been revised but not completely agreed upon internationally. This development was needed because of the widespread use of ultrasound, very sensitive urine pregnancy tests, artificial reproduction technology and serum human chorionic gonadotropin (β-HCG) testing. It is good to use and teach the new terms for better communication and also because the term "abortion", used usually for spontaneous early pregnancy loss, is confusing or even insulting for the patient. In some countries police personnel are allowed to read doctors' records and the patient might be in trouble if you write "abortion". So we prefer to use "miscarriage". Induced abortion implies a termination of pregnancy, whether by surgical or medical means.

(a) Abortion, by the WHO definition, is the *death and expulsion of the foetus (or embryo) from the uterus either spontaneously or by induction up to but not including 20 completed weeks of gestation*. This means up to and including 19^{+6} (after that, it becomes the 21st week). If the gestational age is unknown, the embryo/foetus should be <400g in weight to call it an abortion, according to the WHO definition.

N.B. The specific number of weeks to define induced abortion may vary from one country to another, depending on local legislation. It is agreed, however, that after 23-24wks where a gestation is potentially viable, termination of pregnancy should not be known as an abortion.
A cardinal number indicates quantity; examples are 1,2,3, etc. An ordinal number depicts rank in a series: 1st, 2nd, 3rd, etc.
Ordinal numbers are one higher than the corresponding cardinal numbers. A child 13 months old illustrates the difference: having passed her birthday, she is 1 (cardinal) year old, but she is in the 2nd (ordinal) year of life.
Gestational age is best expressed in completed cardinal weeks and days instead of ordinal weeks or trimesters. Therefore 30^{+6} means 30 completed weeks plus 6 completed days. 24h later, the age is 31^{+0}.

(b) Delivery, by the WHO definition, is the expulsion or extraction of ≥1 foetuses from the mother at ≥20 completed weeks. If the foetus is dead at ≥20 completed weeks, it is a **foetal/intra-uterine death** and will most likely become a stillbirth (unless the mother dies and stays undelivered).
(c) Live birth is the complete expulsion or extraction of a product of fertilization from its mother irrespective of gestational age as long as there is *any* sign of life after delivery.

(d) A full-term birth is delivery from ≥37 to ≤42 completed weeks, *i.e.*>259 to <294 completed days. **Post-dates** implies more than 40^{+0} wks, and a **post-term birth** is a live birth or stillbirth after 41^{+6} completed weeks. A **pre-term birth** is a live birth or stillbirth at ≥20 up till 37 completed weeks.

(e) Neonatal death occurs within 28 completed days after delivery (after ≥20 completed weeks or when ≥400g at birth). *N.B. "Trimester" is a term which should largely be abandoned: it is rather unhelpful, vague and causes confusion.* Use completed days or weeks.

(f) Miscarriages (excluding those caused by an incompetent cervix) most often go through these 4 stages:

(1) **Threatened miscarriage** (there is bleeding and perhaps cramps, but the cervix is still closed),

(2) **Inevitable miscarriage** (the cervix is open but no products of conception have been expelled; most of the time there is no foetal heart movement),

(3) **Incomplete miscarriage** (part of the products have been expelled),

(4) **Complete miscarriage** (all the products have been expelled, bleeding has virtually stopped, the cervix is closed, and the uterus is now much too small in relation to the duration of the pregnancy).

(g) Gravida (G) refers to the number of times a woman has been pregnant, **para** (P) the number of births ≥20wks' gestation, and **vivo** (V) the number of living children.

If the pregnancy more or less 'falls out' without going through the preceding stages and the foetus is recognizable and alive at first (light reflections on a wet chest change with its heartbeat), then there is probably an incompetent cervix; this is unlikely to happen before 16wks. A miscarriage caused by an incompetent cervix can also start with ruptured membranes. An incomplete miscarriage and an inevitable one (if there is no foetal heartbeat on ultrasound while the foetus is >10mm) should be evacuated with the help of instruments, misoprostol or both.

N.B. In an inevitable miscarriage, increasingly after 12wks, suction curettage alone might not succeed in removing the foetus.

In the first 12wks the distinction between an inevitable and an incomplete miscarriage is unhelpful, because you can manage them both in the same way. After 16wks, the distinction is important, because an inevitable miscarriage is not ready for instrumental evacuation (access to misoprostol treatment makes evacuation without instruments quite feasible), whereas an incomplete one must be evacuated. Before 14-16wks it is difficult to tell, by observation of the products, if a miscarriage is complete or not, because the foetus, the placenta and membranes are not sufficiently well formed for you to identify them completely. Even with ultrasound, it is often not easy to make the distinction between retained products or blood clots in the uterine cavity.

(h) Recurrent miscarriage is ≥2 consecutive pregnancy losses.

(i) Delayed miscarriage may be late (12 to <20wks) or early (<12wks) but excluding preclinical/biochemical pregnancy. This term is used when there are low positive urinary/serum β-HCG values and it is not possible to localize the gestation with ultrasound (gestation of unknown location, GUL).

(j) Early miscarriage or **gestational loss** represents a loss <12wks: this is an ultrasound-based diagnosis with a persisting intra-uterine empty sac, loss of foetal heart movement and/or failure of crown-rump length growth over 1wk (38.3).

(k) Septic miscarriage is an incomplete miscarriage with signs of intra-uterine and perhaps extra-uterine infection.

(l) Post-abortal sepsis is pelvic infection after a completed miscarriage or termination of pregnancy.

(m) Retained miscarriage is an intra-uterine death before 20wks, after which the pregnancy is not expelled.

(n) Carneous mole is a continuation of a retained miscarriage, in which the dead conceptus is surrounded by shells of organized blood clot (a blood mole) and has become firm.

(o) Threatened miscarriage. Recommend refraining from sport, jolting movements (*viz.* horse riding, off-road transport, sexual intercourse) and heavy-duty work. Bed rest at home has no proven benefit. Arrange admission if:

(1) There is much bleeding (regardless of the gestational age).

(2) Gestation is >14wks.

(3) There is a bad obstetric history or she lives far away and cannot get help if bleeding becomes much worse, especially during the night.

(p) Uncomplicated uninfected inevitable or incomplete miscarriages

(1) **Before 14wks:**

Monitor the pulse, blood pressure, and temperature, peripheral circulation, and the amount of bleeding. Measure the Hb, and group blood if indicated. Perform an ultrasound scan if possible (38.3). Induce complete evacuation with misoprostol (400µg buccal or sublingual, or 600µg oral or rectal) or oxytocin (10IU IV) or ergometrine (0·25-0·5mg IM).

If bleeding was copious, restrict oral intake and prepare to evacuate the uterus as soon as possible.

Retained miscarriages (with intact membranes) have much less chance of becoming infected than induced miscarriages, so there is no hurry: use 800µg of misoprostol PV, followed if necessary by 400µg 3hrly x3 till complete expulsion occurs.

If expulsion does not happen, repeat the procedure after 3 days. Such treatment may be carried out at home for gestations <10wks if access to hospital is easy in case of brisk bleeding.

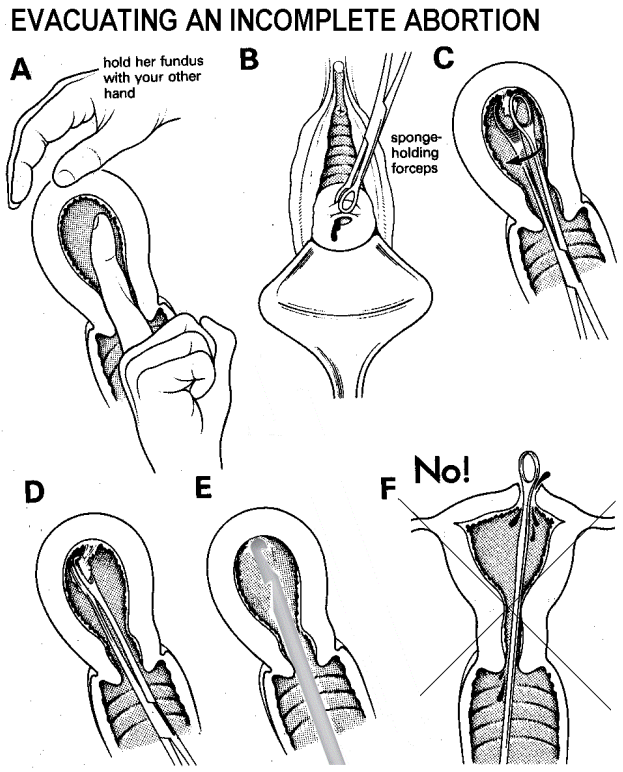


Fig. 20-1 EVACUATING RETAINED PRODUCTS OF CONCEPTION. A, explore the uterus with your finger while your other hand is holding the fundus. You may find it easier to use 2 fingers or your middle finger. B, grasp the cervix with sponge forceps and use them to pull it down. C,D, while holding the uterus with your other hand, introduce ring forceps, grasp and remove any products of conception, reinsert the forceps and do the same thing again. E, *gently* aspirate the uterus with a plastic flexible Karman suction catheter. F, this is the disaster you are trying to avoid!

Many miscarriages don't need evacuating, but those that do, need evacuating quickly, so don't let incomplete miscarriages wait unnecessarily. Evacuating a pregnant uterus differs from curetting a non-pregnant one (23.4) in 2 important ways:
(1) After a partial miscarriage the cervix is open, so there is rarely any need to dilate it.
(2) The wall of a uterus, especially if infected, is so soft that you can perforate it just as easily as the small atrophic uterus, giving rise to postmenopausal bleeding.

BLEEDING BEFORE THE 20th WEEK

Miscarriage (induced elsewhere, or spontaneous) usually presents with vaginal bleeding.

DIFFERENTIAL DIAGNOSIS includes:
(1) ectopic gestation (20.6)
(2) gestational trophoblastic disease (23.10).

N.B. Bleeding in late pregnancy may be due to placenta praevia (20.11) or abruption (20.12).

N.B. Remember gynaecological causes of a bloody vaginal discharge: *trichomoniasis*, *candidiasis*, venereal warts, cervical polyps, cervical ectopia (defined as uterine columnar mucosa extending further than average on the pale external cervix), and cervical carcinoma.

N.B. There may be no pregnancy, but dysfunctional uterine bleeding (24.3), or bleeding from polyps or fibroids (24.7). Much bleeding remains unexplained.

SITI (27 years) was admitted with what looked like a threatened 16-week miscarriage. It seemed to settle, and she was discharged, but she bled in the bus on the way home and was readmitted. Foetal parts were extracted through a dilated cervix, and traumatized pieces of bowel were seen through it. The diagnosis had to be adjusted to complications caused previously by an (instrumentally) induced back-street termination. A laparotomy showed a laceration in the descending colon, old clots and pus in the peritoneal cavity, and a rupture of the uterus. The lacerations in the descending colon and uterus were sutured and the abdomen closed. Some days later she passed faeces through the cervix. She was re-explored, and a proximal defunctioning colostomy was fashioned, after which she eventually recovered. She was of course counselled at that stage on the use of contraception. If this termination was performed because she thought the family complete, then a tubal occlusion should have been done together with the uterine repair. There is quite a risk, *if* she ever gets pregnant, that it will be an ectopic or that she will rupture the uterus.
LESSONS This true story is an extreme case. It shows the magnitude of the disasters that can follow the mismanagement of what might seem to be quite a minor condition. She was fortunate to escape with her life. The many lessons include: (1) Somebody had tried to terminate a 16wk gestation, which is dangerously late for a non-expert outside a well-equipped theatre. The availability of misoprostol in urban areas (including the black market) will often now make these late terminations far less dangerous because fewer skills are needed. (2) If an induced miscarriage is incomplete, evacuation is mandatory. She should not have been discharged before the uterus had been emptied. (3) Whenever the colon has been damaged and faecal soiling is present, a proximal defunctioning colostomy is indicated. Had this been done at the first laparotomy, she would not have required another operation. (4) If an ultrasound scan had been performed, the diagnosis would not have been missed or at least it would have been clear that there was no foetal heart movement in the presence of retained products.

EVACUATION. Mandatory indications are:
(1) Considerable bleeding (evacuation is urgent).
(2) Bleeding more than during normal menstruation, which continues >24h.
(3) Where retained products of conception are obviously still present on vaginal examination or ultrasound (38.3).
(4) Any indication of infection or physical interference to the pregnancy.

(2) **After 14wks:**

If the cervix is open at least one finger, there is no foetal heart activity on ultrasound or sonic aid, but the products of conception, especially the foetus, have not been expelled, assess and monitor as above. *Don't evacuate the uterus with instruments until the foetus has been expelled*: you can expedite this with misoprostol.
If evacuation is incomplete, complete it. If however the foetus and placenta are expelled together, and the membranes are complete, there will be nothing left to evacuate.
If the foetus is still in the uterus and there is a serious infection, *don't waste time!* Use misoprostol and IV antibiotics and also organize an evacuation immediately even though you may need instruments to remove the foetus because it is too large for evacuation by suction curettage alone. The misoprostol might not evacuate the retained products, but will dilate the cervix and make evacuation easier. *Don't wait >2h after administering antibiotics before doing the evacuation!*

(q) Infected miscarriage
If there is fever, a foul vaginal discharge, and perhaps signs of peritonitis, the miscarriage is septic (23.2).

EVACUATING AN INCOMPLETE MISCARRIAGE (GRADE 2.1)
Misoprostol may make evacuation unnecessary, so try it first (unless there is sepsis, 23.2). The products of conception may evacuate subsequently, and bleeding may stop. Anyway, it will dilate the cervix, maybe allowing you to perform a digital evacuation.

EQUIPMENT. A catheter. 2 ovum forceps or sponge-holding forceps without rachets (one for swabbing the vagina and the other for removing the contents of the uterus), uterine curettes blunt and sharp, preferably a few sizes of each. A vaginal speculum (Sims', Auvard's, or Collin's). A set of Hegar's dilators (only occasionally necessary). *Don't use a sound*, because this can readily perforate the uterus. If obtainable, Karman suction curettes are best; these are boilable but will melt in an autoclave. This means that when cleaned, boiled and kept in betadine they are, certainly compared with the cervix and vagina, for all practical purposes sterile.
Have intra-uterine contraceptive devices (IUDs) readily available.

Occasionally, if the pregnancy was <10wks and was unintended and not infected, an evacuation can be combined with inserting an IUD. That might prevent a recurrence, possibly with more complications. If there was an obvious unintended pregnancy in a multipara who is not very young, you might counsel for an evacuation plus tubal ligation. Combine an IUD with antibiotics: in fact it is a good idea to administer antibiotics with every evacuation, especially because you can never be entirely certain the miscarriage was not induced. Remember the option of inserting an IUD when you perform an evacuation, *unless there was gross infection*.

CAUTION! Don't operate until an IV infusion of Ringer's lactate or saline is running if haemorrhage is severe, if there is hypovolaemia or anaemia. Resuscitation may be life-saving: proceed with this at the same time as the evacuation.

ANAESTHESIA
Ketamine is useful but GA is not absolutely necessary. Remember if you use 100mg pethidine with 10mg diazepam IV in the same syringe, which works well unless the patient is anaemic, septic or very small, this may produce respiratory depression.

An alternative is LA: use 20ml 1% lidocaine preferably in combination with 1:80,000-1:100,000 adrenaline para- or intra-cervically. Adrenaline can substantially decrease the blood loss *in pregnancies of >14wks*, during and after the procedure. It is therefore often useful even when GA is used. *Don't use halothane*: this may relax the uterus and cause brisk bleeding.

N.B. 20ml of 2% lidocaine can be absorbed so fast in the well-perfused uterine area as to be dangerous (epileptic fit, bradycardia, myocardial depression): so, dilute it.

Use a long *thin* needle (the cervix tends to bleed somewhat from the needle hole) and make sure the needle is well and truly pushed on the syringe because force is needed to inject into the cervix and *if the needle comes off you will get blood and LA sprayed in your face*.

Remember to dispose of the needle directly in a proper way: if you put it on your instrument tray, you might easily prick yourself or the nurse when handling the instruments later (5.3).

POSITION
Use the lithotomy position with the buttocks over the end of the table, so that you can insert your instruments comfortably in any direction. Make sure the bladder is empty before you start: ask the patient to pass urine just before the procedure.
First, perform a bimanual examination using a disposable glove with two fingers in the vagina and your other hand on the abdomen. Check:
(1) The state of the cervix and its degree of dilatation.
(2) The size of the uterus and the products of conception palpable inside it.
(3) Any adnexal masses (*don't miss an ectopic gestation but don't use an examination under GA to diagnose one!* 20.6).
Then put on sterile gloves.

METHOD
Clean the suprapubic area, vulva, and perineum with chlorhexidine, and put a drape under the buttocks and on the abdomen. Take careful aseptic precautions. Use a swab on a sponge-holder to clean the vagina.

If you can get your finger into the cervix, use it to empty the uterus (finger curettage). A finger is much safer than a curette, because you can feel where you are, so *avoid using a curette if you can*. Put half your hand into the vagina and use your right index or middle finger. At the same time push down the fundus of the uterus with your left hand on the abdomen, so that your finger can reach right into it. Ideally this requires good muscular relaxation. If you are using LA, be gentle, talk to the patient kindly, and persuade her to relax. Loosen all the retained tissue with your finger. If you can empty the uterus this way, there is no need to curette it. Moreover, if there has been instrumental interference to the pregnancy resulting in a uterine perforation, you can usually diagnose this easily without causing further damage.

N.B. Fishing around with any instrument in a large flabby uterus for a few fragments of tissue is likely to do more harm than good, especially if you use a non-Karman curette. Furthermore, if you start with instruments, you might not only pull out loops of bowel, but also be blamed for causing the perforation which was present already!

If you cannot get your finger into the cervix or reach the fundus, grasp the cervix with a ratcheted sponge-holder or vulsellum (20-1B). With your left hand, pull the cervix well down with the instrument attached to the cervix to straighten the uterine cavity. Keep pulling during the whole procedure. You hardly ever need a speculum at this stage anymore because the ligaments are lax and/or the vagina gaping. Without a speculum, it is easier to pull the uterus straight and hence avoid a perforation. Introduce another pair of non-ratcheted sponge-holders into the uterus with your right hand. Slide them in gently until you can lightly feel the top of the fundus. Open them, turn them through 90º, close them, and remove them (20-1C).

Do this several times, to remove pieces of placenta hanging from the uterine wall, until the uterus is empty. If you can, check with ultrasound and/or use the suction curette with the largest diameter (1cm normally) to make sure the uterus is empty.

If you cannot insert your finger or a metal curette, as occasionally happens in the first 14wks when the cervix is not sufficiently dilated but the uterus seems enlarged, dilate it. First insert a small dilator, and then progressively larger ones. *Don't dilate the cervix in this situation further than necessary, especially if you intend to insert an IUD.* A rule of thumb is that the diameter of the Karman cannula should till <13wks be 2mm less than the number of wks if the foetus is still in the uterus. The means at 12wks a 10mm cannula is fine; at 6wks 4mm will do. Of course, at 18wks, 10mm will do if you only mean to extract a piece of placenta.

CAUTION!
(1) *Don't put a sound into a pregnant uterus*. If you want to know how long the uterus is, insert a large Hegar dilator, suction curette or sponge-holder and mark how far it goes in with your finger.
(2) Be gentle, or you will perforate the fundus. Your exploring finger will have shown you how deep it is. With your left hand on the abdomen, explore the uterus again with your finger to make sure it is empty.

If you still cannot empty the uterus fully, use a suction curette to remove the remaining pieces of placenta. While it is still well contracted (sometimes with the help of 400µg misoprostol rectally or oxytocin 10IU), use the largest suction curette you have or very gently scrape the inside of the uterus (20-1E). Let it almost rest in your hand as you use it. Leaving the retained products of conception behind is serious, but perforating it (20-1F) is more so.

If you do perforate the uterus with a suction curette, *first disconnect the suction tubing and then withdraw the curette,* otherwise you might pull bowel or adnexa out!

You will know that the uterus is empty by:
(1) A characteristic grating feeling.
(2) Your failure to remove any more tissue.
(3) Resistance to movement with a suction curette: the retained products act as a lubricant, so when they are no longer there, you will feel more resistance.
(4) An ultrasound performed during or directly after the evacuation. *N.B.* 10mins later, retained products are indistinguishable from blood clot.

Finally, with a large uterus that is still bleeding, perform a bimanual compression (22-10) to encourage contraction and expel clots from the uterus. Put two fingers into the anterior vaginal fornix, and your other hand onto the abdominal wall. Compress the uterus between them.
Return the patient to the ward with a vulval pad. Inspect this from time to time during the first 3h after the evacuation.
If bleeding recurs, inject another dose of oxytocin 5IU IV or IM, or use 400µg of misoprostol rectally.

POST-OPERATIVELY, monitor for further bleeding and check the vital signs. If all is well, discharge the patient and advise her on contraception, which should always be part of the ward routine. Advice is often not good enough. Providing the means at the same time is much better. If you have not inserted an IUD or an implant at the end of the evacuation, offer Depo-Provera at this stage or other contraceptive medication. Discuss the option of an early tubal ligation. Ensure that good, preferably long-acting, contraception is available to those whose pregnancy was (obviously) unintended.

If there has been a suspicion of vaginal interference to the pregnancy, or venereal infection, use an appropriate broad-spectrum antibiotic (*e.g.* doxycycline and metronidazole). If you have the resources, it is a good idea to use antibiotic prophylaxis for everyone. However, if there is overt sepsis, IV antibiotics are required (23.2).

N.B. Syphilis can cause miscarriages: if you have the resources, check for this on the spot and have the results ready within 30mins (just like with an ANC visit), otherwise only 50% will return for treatment. Treat with long-acting penicillin and make sure the partner is also treated.

N.B. It is obvious that some patients will have a complete family at this stage *e.g.* para 5 of age 40yrs, or para 3 HIV+ve. Some patients will be very grateful if you combine a uterine evacuation with a tubal ligation. Regret is usually only seen in young women <30yrs old. If the logistics of your hospital make it possible, you should give women a choice.

(r) Difficulties evacuating the uterus (23.4)
If there is profound hypotension, the cause may be severe blood loss because the placenta has become stuck or is half hanging out of the cervix (common). The external os may be tight, while the internal os and cervical canal dilate to accommodate the pregnancy. This is quite different from a cervical gestation (20.8).
Don't wait to put up an IV line. Remove the placenta with a gloved finger *on the ward* without anaesthesia. If this fails, pass a Sims, Collin's or Cusco's speculum. If you see products of conception in the cervix, remove them with sponge forceps. Blood loss usually stops miraculously. Shock may be caused by a vasovagal attack: you may then be fooled into thinking a blood transfusion is necessary.

If there is heavy bleeding, start resuscitation and administer misoprostol or oxytocin and at the same time evacuate the uterus with a finger *on the ward*. Even if evacuation is not complete, it will help stop the bleeding.

If bleeding does not stop after evacuation and you have excluded a uterine perforation, it is probably due to poor contraction of the uterus, or there may still be products of conception in the uterus. Often there is no obvious reason. Make sure the uterus is empty. Administer 800µg misoprostol rectally or oxytocin IV (40 units/L), massage the uterus to stop it bleeding, and repeat bimanual compression. Be patient at this stage: 5-10mins of bimanual compression may be necessary, but it will usually succeed.

If these measures fail to control bleeding, suction curette the uterus if you have not already done so. Sometimes packing the uterus helps; *don't pack the vagina* as that only conceals the problem; it will not usually remove the cause of the bleeding. A torn cervix is occasionally the cause and suturing might be a technical challenge. In that case, packing the vagina near the cervix might solve the problem.

If even this fails to control bleeding (very rare), tie both uterine arteries or perform a hysterectomy. Very rarely a cervical ectopic gestation (20.8) is the problem.

If serious anaemia results, *don't jump to transfusion unless the Hb is <40-50g/L.* Always supply iron to take home. The younger the patient, the easier it is for her to cope with a low Hb. *It is better to spend a few days extra in hospital because of dizziness than to get a transfusion contaminated with HIV or hepatitis B or C.* In young, anaemic, otherwise healthy women on oral iron, the Hb can increase 20·50g/L/wk. Older women and those with sepsis, malaria, or heart disease cannot, however, so easily deal with very low Hb levels.

N.B. Sometimes women abort because of malaria and therefore combine blood loss with haemolysis. This can be an extremely dangerous situation if missed. The high fever may then be diagnosed as sepsis as a result of the miscarriage (induced or otherwise) instead of the cause of the miscarriage. Severe malaria can make the Hb drop in a day by 50g/L. The jaundice seen with severe malaria is also seen sometimes in sepsis.

If you find injuries to the vagina, cervix or uterus, **or physical interference with the pregnancy is suspected**, and there is shock, severe sepsis or more severe anaemia than simple vaginal blood loss could explain, or there is free gas in the abdominal cavity, the uterus is probably perforated. Perform a laparotomy immediately, and inspect the uterus. *Don't forget to counsel and get consent for a tubal ligation.*

If there is no improvement after evacuation, reconsider the diagnosis. She may have an ectopic gestation, or be severely anaemic, or have a collection of pus. If you find an abscess in the pouch of Douglas, drain it (10.3).

If you think you have perforated the uterus,
(i) after emptying the uterus, look if there is fat, omentum or bowel on the forceps or in the vagina. If not, return the patient to the ward. Keep her nil orally on IV fluids with gentamicin and metronidazole, and observe the pulse, temperature, blood pressure, urine output and suppleness of the abdomen carefully. The perforation will probably heal easily, especially if it occurs before 14wks. If there are, unusually, increasing signs of infection or bleeding, perform a laparotomy to close the wound in the uterus.
(ii) before emptying the uterus, try a digital evacuation and accept that, perhaps, evacuation is incomplete. Observe carefully as before.

If there is evidence of omental or bowel injury, start resuscitation and perform an immediate laparotomy, and close the uterine perforation. Repair (11-5) or resect (11-7) the bowel and omentum.

If there is severe bleeding or an extensive tear, tie the uterine arteries at several locations in the area just after they enter the uterus (22-14). If this fails, a B-Lynch suture (22-13, 22.11) or hysterectomy (22.17) may be necessary. If you have closed a uterine tear, warn that the uterus is in danger of rupturing in later pregnancies and an elective Caesarean section (21.9) would then be mandatory.

If you feel a fibroid in the uterus (uncommon), it may have been the cause of the miscarriage (unusual). Leave it for 3 months before you treat it.
If it is pedunculated and submucous with a narrow neck, *don't be tempted to twist it off vaginally at the time of the miscarriage*. This can cause severe bleeding. Leave it for 1 month while treating with iron supplements. If the cervix is closed, first use misoprostol 400µg vaginally 2h prior to excision (23.7).

If there are very few curettings, the miscarriage is probably complete. There is however a possibility that your diagnosis may be wrong, and that she has a CHRONIC ECTOPIC GESTATION (20.7).
Material removed or spontaneously aborted from the uterus with an ectopic gestation has no connective tissue-like structure. It is endometrium/decidua. If you put it in an ordinary household sieve, you can easily make it disappear if you rub it with a brush under the tap. In contrast, even the products of conception at 5wks have so much structure that this is not possible.

If you feel that she has a uterine septum, clean out each side of the uterine cavity.

BE CAREFUL WITH THE CURETTE!

20.3 Termination of pregnancy

Legislation related to termination of pregnancy differs enormously throughout the world. In some countries, doctors don't dare to terminate a pregnancy even to save a woman's life. In others, terminations are allowed on request up to 22wks.
Historically these operations were of course quite dangerous before modern techniques, prostaglandins and antibiotics were available. The extensive use of misoprostol (replacing sticks, roots, catheters, soaps, poisons and uterine massage) has now made even late terminations far less dangerous.
Up to 7-9wks' gestation, a combination of 200mg mifepristone (blocks progesterone receptors) followed 12-72h later by 800µg vaginal misoprostol tablets (prostaglandin E1 compound) is very effective. In most countries, mifepristone, the 'abortion pill', is not available. However, misoprostol (perhaps in repeated doses) without mifepristone is also quite successful.
The disadvantage of this so-called medical termination is that it is far less easy to combine with the insertion of an IUD than an early suction evacuation. In most countries induced terminations are performed with the help of suction curettes only up to around 13wks. GA is not essential but ketamine can be useful.

You should hesitate to remove pregnancies instrumentally which have proceeded further as you will need to use a combination of suction and forceps extraction. This needs experience, proper training and preferably guidance by ultrasound.
Induced terminations past 12 weeks' gestation are prohibited under most conditions in many countries. *You must acquaint yourself with the laws in force in your country before intervening.*

20.4 Foetal death: retained miscarriage and intra-uterine death

An embryo or foetus can die at any time during pregnancy. What you can do about it depends on whether death occurs before or after 20wks. Before this time this is termed a retained miscarriage, afterwards an intra-uterine death.

(a) Before 20wks, a dead foetus is usually expelled without the mother knowing that the pregnancy has ended. Occasionally, however, uterine emptying is delayed for several weeks with failure of normal growth in size of the uterus and the mother feeling symptoms of early pregnancy diminishing. Alternatively, there may be a threatened miscarriage which stops bleeding spontaneously, and is followed by a brownish discharge and no further true blood loss.
Although the loss of a pregnancy may be tragic, a missed miscarriage has few physical risks, there is little risk of a clotting defect this early in pregnancy, and provided nobody interferes with instruments, there is very little risk of infection.

(b) After c.20wks:
(1) The mother is aware of the intra-uterine death of the foetus because foetal movements stop, or don't occur when they should (18-22wks in a primipara, 16-20wks in a multipara).
(2) The foetal heart movements are not visible (6·5-8wks) on ultrasound or not heard (10-16wks) with a Doppler. Remember that using a foetal stethoscope is unreliable in obesity or polyhydramnios and enables you to hear the heart only at 20-28wks.
(3) The height of the fundus, as found by palpation, fails to match that expected from the dates. Instead, it either remains stationary or falls. For this sign to be useful, you must measure the height of the fundus above the symphysis pubis accurately with a tape measure. So, when you suspect foetal death, impress this on the midwives.
(4) Radiological signs of foetal death (after 28wks) show overlapping of the bones of the foetal skull (Spalding's sign), hyperflexion of the spine, and gas in the great vessels, though it is rare that there is a real indication for radiographs in this setting.

N.B. β-HCG tests for pregnancy take 4 or even 8wks to become -ve, so they are of little value.

There is a 50-90% chance that spontaneous delivery will occur within 4wks after foetal death, whatever the duration of the pregnancy. But, as long as a dead foetus remains inside the uterus, there is the remote but serious risk of a serious coagulation defect and catastrophic bleeding. This risk is low initially, but increases with time, particularly 4-6wks after death.

Rupturing the membranes to induce labour is dangerous, because the dead foetal tissues are easily infected by anaerobes and antibiotics will *not* reach the foetal tissues if there is no foetal circulation.

Use oxytocin and/or prostaglandins for a missed miscarriage or intra-uterine death. Throughout pregnancy there is sensitivity of the uterus to prostaglandins, although the optimal dose varies according to gestation, but its sensitivity to oxytocin increases with each gestational week and oxytocin in early pregnancy is ineffective. Misoprostol is easiest to administer (22.2).

(c) Missed miscarriage
If a mother's uterus is small for her gestational age, perhaps with a brownish vaginal discharge, suspect foetal death. Monitor the growth of the uterus carefully: it will not grow, and may even become smaller. Pregnancy tests become -ve after some weeks. Methods of detecting the foetal heartbeat vary in their sensitivity: ultrasound scanning (38.3) detects it certainly at 8wks, Doppler ultrasound at 10-16wks, and an ordinary stethoscope at 20-28wks.

DIFFERENTIAL DIAGNOSIS includes:
(1) A normal pregnancy of shorter duration (wrong dates),
(2) A slow-leaking ectopic gestation,
(3) A false (imagined) pregnancy (pseudocyesis),
(4) Fibroids.

MANAGEMENT
If spontaneous miscarriage does not follow after 4-6wks, proceed as follows:

If the uterus is smaller than 10wks, put 400µg misoprostol in the posterior vaginal fornix, or buccal cavity, and repeat this 3hrly for 12h. Spontaneous evacuation will usually occur; if not, dilate the cervix to Hegar *(maximum 10)*, and then use a #6-10 Karman curette, depending on the largest Hegar used, with maximum vacuum. Continue until the uterus is empty, and you can feel the uterus tight round the curette.

If the uterus is larger than 12-13wks, *don't attempt an ordinary dilation and curettage*. Instead, use misoprostol.

N.B. You can dilate the uterus to Hegar 10, and use a #10 Karman curette, which works up to 12wks but not beyond because the foetal parts become too large! Attempting to perform a standard dilation and curettage (23.4) on a uterus larger than this can cause disastrous bleeding, and perhaps infection.

(d) Intra-uterine death (*i.e.* after 19^{+6}wks)
The mother notices that foetal movements stop, or don't occur when they should (at around 18wks). Or, a midwife fails to hear the foetal heart after 24wks.

DIFFERENTIAL DIAGNOSIS
(1) A normal gestation of shorter duration (wrong dates).
(2) Gestational trophoblastic disease.
(3) Polyhydramnios (a uterus large for dates due to excess amniotic fluid).
(4) Multiple gestation with small foetuses.
(5) An abdominal gestation.
(6) Ascites, an ovarian tumour, fibroids, or a false pregnancy.

MANAGEMENT
Confirm the absence of the foetal heartbeat with ultrasound or Doppler (38.3). If you are not sure, repeat the ultrasound after 1wk.
CAUTION! A pregnancy test is no use at this stage. It may be +ve when the foetus is dead.

Do nothing for 2wks after foetal movements have stopped. Explain carefully why you are doing nothing. The patient may find this difficult to understand and her husband may try to persuade you to act prematurely. Explain that, if you attempt induction by the method below, it may fail and she may need a few days' rest before you try again.

If she is still undelivered 2wks after foetal movements have stopped, consider induction. Before you start this, check the clotting time and platelet count, if the foetus might have died ≥4wks before. Misoprostol is successful in >90% of cases. Start with very low doses for grand multipara or where there is a uterine scar. If it fails, introduce the tablet(s) inside the cervix. Fever, shivering, and gastrointestinal symptoms while having prostaglandins are probably side-effects rather than due to infection.

If this also fails, provided gestation is from 13wks (before which it is unnecessary), until 28wks, try an infusion of 5U oxytocin in 500ml of Ringer's lactate or saline, at 25drops/min (22.2). You may find that labour does not start until the following day. If this fails, repeat the infusion the next day with 25 units in 500ml. If necessary, wait and repeat it after 7 days. If this does not work, wait 7 days more and try a 3rd time. You may have to use up to 100 units in 500ml (the absolute maximum). Usually, much less is necessary.

CAUTION! EXTRA SPECIAL CARE is necessary when you use oxytocin!
(1) You may have to use large doses. Oxytocin has an antidiuretic effect, and fluid overload is a danger. So:
(a) increase the concentration of the infusion, rather than the volume you use,
(b) use Ringer's lactate or saline, rather than 5% dextrose,
(c) *infuse a maximum of 3L IV fluid in 24h*,
(d) keep a fluid balance chart; if there is a positive fluid balance of >2L, stop the infusion.
(2) Oxytocin (and misoprostol) can rarely rupture the uterus as early as 18wks, so *don't use more oxytocin than you need.*

If drowsiness or convulsions ensue whilst on an oxytocin infusion, suspect water intoxication. Stop the infusion and let the kidneys excrete the water. Infuse 100ml 5% saline slowly IV.

If an escalating oxytocin infusion fails, and the products of conception have not been expelled within 2-4wks of presentation, check that you have not missed an abdominal gestation. Having excluded this, try the Foley catheter option: using a Cusco's or Collin's speculum and sponge forceps, pass a sterile Ch14-16 Foley catheter with a 30ml balloon gently through the cervix into the extra-amniotic space. A Foley catheter of this size will always enter a pregnant cervix. Now inflate the catheter balloon and leave it *in situ* 24h.
If the method you have used has not succeeded in 2 days of trying, try another method, or wait for 2-3 days and try again.
CAUTION! Don't rupture the membranes. It may hasten delivery, *but it is not worth the risk*.

(e) Foetal death at term or during labour (21.5,8)
A dead foetus is usually easy to deliver when death is the result of gestational hypertension or abruption, because the foetus is usually small and is often macerated. But if death occurs because labour was obstructed, delivery is more difficult. Caesarean section might seem to be the obvious answer. Unfortunately, if the head is impacted deep in the pelvis, removing it from the uterus at Caesarean section is difficult. There is also the serious immediate risk of septic shock and peritonitis, and the later one of a uterine scar. Provided the head is well down in the pelvis, an operative vaginal delivery, if necessary a destructive one (21.8), will be safer.
N.B. Make sure you exclude a dead extra-uterine gestation.

If the foetal head is high and hydrocephalic, drain its cerebrospinal fluid. You can do this *per vaginam* or, if the membranes are still intact, with a large IV cannula through the abdominal wall straight into the uterus (making sure the bladder is empty).

Otherwise, Caesarean section is the only option, but even then a hydrocephalus is often so large that you need to drain the head before you can extract it via a routine uterine incision, or you have to make your incision higher, larger or maybe even vertical and any of these scars is in real danger of rupturing in a following pregnancy.

MPHO (16yrs), gravida I, was admitted in labour in a district hospital with stalled progress. The doctor diagnosed CPD with an enormous caput. At Caesarean section, the routine incision was, by a large margin, not large enough to deliver the head now found to be hydrocephalic. The doctor perforated the skull and 2L fluid escaped. The head could now be delivered. The baby also had a large spina bifida and club feet, and was wrapped in towels and put on a table. During abdominal closure, the baby, who everybody thought had died, started crying. The wound in the skull healed afterwards but it was clear the legs were permanently paralyzed. The baby died 3 months later probably from repeated urinary tract infections. The mother was severely stressed and depressed during this time. Any further delivery should be under close medical supervision.
LESSON Hydrocephalus drainage only needs a small needle. Mothers of malformed babies need counselling before delivery if at all possible.

(f) Difficulties with a dead foetus <30wks

If you are not sure about foetal death, perform an ultrasound (38.3); alternatively, wait, and review in 2wks. By this time, it should be clear if the foetus is dead or not.

If delivery is complicated by severe bleeding, disseminated intravascular coagulation (DIC) is a possibility (3.5). Maintain the blood volume, and try to administer fresh blood, as well as packed red cells and fresh frozen plasma. If bleeding is not controlled when the uterus is empty, use an oxytocin infusion with 400μg misoprostol PR. Try compressing the uterus, pack it for 24h, and then remove the pack. This is a useful temporary measure for any bleeding uterus, and may save the need to perform a laparotomy.

If this fails to control bleeding, apply a B-Lynch suture (22-13); failing this, tie the uterine arteries (22-14). If this also fails, put a tourniquet round the cervix (22.11), which will give you time to arrange a hysterectomy (21.17) if necessary.

If you know or suspect that an IUD has been *in situ* >4wks, check the platelets. If they are very low, that is an indication for administering heparin (paradoxically). There is a risk of bleeding because of DIC. Use the dose of whatever heparin you have available normally used for prophylaxis. The platelet count will rise rapidly; start induction the next day.

20.5 Recurrent mid-term miscarriages

It is not easy to help women with a history of repeated early miscarriages. These are often the result of some foetal abnormality for which nothing can be done. The best advice is to keep trying. Most women will eventually achieve a successful pregnancy.

Although recurrent miscarriage (RM) affects only 1-3% of couples, it has a major influence on the wellbeing and psychosocial status of patients. Anatomical malformations, infectious diseases, endocrine disorders, autoimmune defects as well as acquired and inherited thrombophilia are established risk factors in RM. Treatment strategies like aspirin and low molecular weight heparin are standard, although only a few placebo-controlled trials have proven their benefit in respect to the live birth rate.

Mid-term miscarriages are different. They are not usually caused by recognizable foetal abnormalities. Some are due to maternal illness (HIV disease, syphilis, hypertension, uterine fibroid vaginitis, cervicitis, diabetes, etc.), or to a congenital malformation of the uterine cavity. A low level of progestagens may play a role. As in early pregnancy, often no cause can be found. The prognosis of a mother with repeated mid-term miscarriages depends on the cause, and is excellent if syphilis can be treated, or cervical incompetence corrected surgically. Hypertension and diabetes are more difficult to treat, and the outcome of the pregnancy is less certain. Mothers in whom no cause can be found have a reasonable prognosis: about 70% of their pregnancies go to term.

Here we are concerned with the management of patients with 'suspected cervical incompetence'. This means that the cervix opens spontaneously between 14-30wks, without the uterus contracting. Sometimes this is owing to a previous forceful dilatation of the cervix, or to a previous traumatic delivery. Usually, there is no obvious cause.

The diagnosis is difficult. It is usually made by the history alone. A typical patient gives a history of ≥2 spontaneous mid-term miscarriages, without much uterine contraction (until the membranes have ruptured), or bleeding. The first symptom is a watery vaginal discharge, often followed by a sudden loss of amniotic fluid. Soon afterwards the foetus is delivered, often still alive. The diagnosis is only certain in the present pregnancy if the uterus is found to be effacing and dilating, without any uterine contractions. When this is happening, it is often too late to intervene surgically.

If you have a vaginal ultrasound probe, it is easy to measure the length of the cervix. If the cervix is ≤1·5cm before 32wks, you have an indication for a cervical suture if there is a history of repeated consecutive mid-term miscarriages.

N.B. In multiple pregnancies, however, 2-4cm dilation is quite frequent without an imminent delivery although, of course, multiple pregnancies are associated with pre-term deliveries.

True cervical incompetence is probably quite rare. *Don't make this diagnosis too often* or you will operate upon many patients unnecessarily. So only operate on those patients with a highly suggestive history.

N.B. Cervical incompetence does not cause miscarriage before 14wks.

A course of doxycycline (or erythromycin) and metronidazole before the next pregnancy or during this pregnancy may help.

McDONALD'S CERVICAL CERCLAGE (GRADE 2.2)

The simplest method is McDonald's, which is a variation of the original Shirodkar suture. If you do it for the right indications, it has a good chance of succeeding.

Timing is critical. If you do it too early (<14wks), an early miscarriage due to a foetal abnormality may arise, and the suture is wasted. If you do it too late (>24wks), a miscarriage may have already happened.

Don't insert a suture between pregnancies. This will cause more trouble than it is worth. Remember that the benefits of the procedure are related to good selection, especially by examination of cervical length.

SIFLOSA (20 years) had a McDonald suture inserted at 14wks, following 3 mid-term miscarriages. Her pregnancy continued uneventfully until term, when she was admitted for delivery. Unfortunately, the consultant who inserted the suture was on leave, and it was not noticed by the duty team. She complained of severe pain during the second stage of labour, but this was ignored. Labour proceeded normally, and she delivered a live baby without help. Immediately after delivery she complained of urinary incontinence and collapsed. No one took any notice of this, and she was discharged after 2 days. On examination 2 months later in another hospital, she was found to have a high 1cm vesico-vaginal fistula, which was contiguous with the cervix, which was torn and ragged. This was successfully repaired abdominally.

LESSONS (1) Always explain clearly to the patient that she must have the suture removed at 37wks or in labour. (2) Take her complaints seriously.

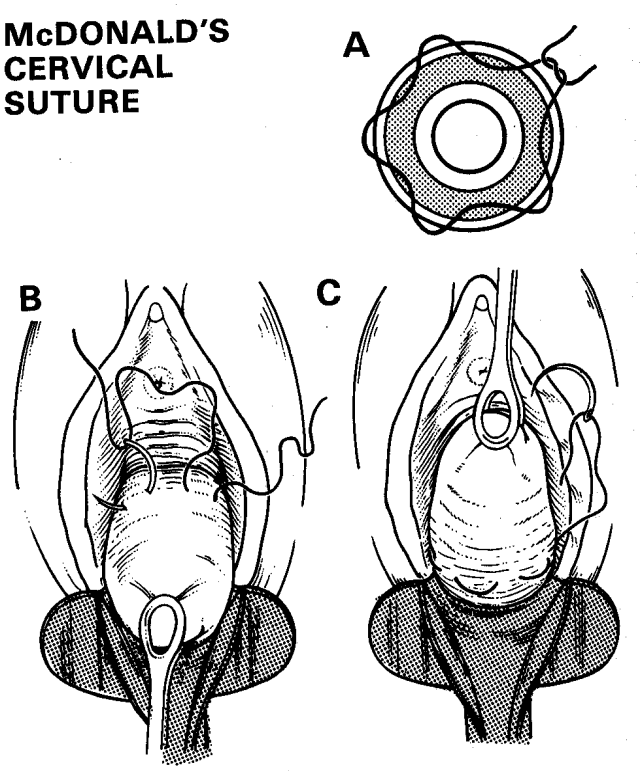


Fig. 20-2 McDONALD'S CERVICAL CERCLAGE SUTURE. A, the position of the suture. B, inserting it anteriorly. C, inserting it posteriorly.
Partly from Bonney V, Gynaecological Surgery, Baillère Tindall, 2nd ed 1974 with kind permission.

INDICATIONS
Two or more consecutive, almost painless miscarriages between 16-28wks especially when the foetus was alive just after birth, and/or there was initially premature rupture of membranes. There may be a scarred widely open patulous cervix. Preferably insert the suture at 14wks, when the danger of an early miscarriage is passed.

CONTRAINDICATIONS
(1) Drainage of amniotic fluid or rupture of the membranes.
(2) Vaginal bleeding.
(3) Established premature labour which does not respond to β-mimetics or indomethacin.
(4) Local vaginal or possibly intra-uterine infection.
(5) Foetal anomalies if you can detect them.
(6) An IUD or a retained miscarriage.
(7) Pregnancy >28wks.

N.B. Exclude HIV disease, syphilis, hypertension, diabetes, and uterine abnormalities such as uterine fibroids.

CAUTION! Don't insert these sutures unless:
(1) The mother has access to a hospital where at all hours of the day and night there will be someone present who will see her, and who is competent to remove the suture.
(2) You have explained precisely what you are going to do, and that the suture must be removed at 37wks, or when labour starts.

ANAESTHESIA
(1) Light GA
(2) Ketamine.
(3) Spinal. You must be able to retract the cervix and dilate the vagina widely to insert the sutures.

METHOD
Check the foetal heart with ultrasound or Doppler. Confirm the gestational age (38.3).

Insert a speculum. Grasp the anterior and posterior lips of the cervix with the same sponge forceps. Insert a #2 monofilament nylon (or special cerclage suture) superiorly in the outer surface of the cervix, near the level of the internal os, about 3mm under the surface of the cervix staying more or less at the same depth in the cervix for 90-120º, and then let your needle come out. Continue to reinsert the sutures in the cervix near the place where your previous insertion exited the cervix at regular intervals as shown, so as to encircle it.

Then tighten the suture round the cervix and knot in such a way that when it is tightened it would still be easy to insert scissors between the knot and the cervix. This is so that, later at 37wks or when in labour, you can cut on one side of the knot. The canal must be just patent as the suture is tied. As the patient is pregnant, *don't insert a dilator*. Admit her for 1wk: most failures occur in this time.

Write on the notes outside and inside in large red letters:

REMOVE CERVICAL SUTURE
NOT LATER THAN 37WKS

Make a drawing to show where the knot is to facilitate removal when it is time.

Review every 2wks, and insert a speculum or examine digitally to check that the suture is still in place. Occasionally it comes out and needs reinsertion.

At 36wks, admit the patient (because she may not be able to present herself in time when labour starts) and remove the suture in early labour or at 37wks.

N.B. If labour does start with the suture *in situ*, it may cause a severe cervical tear, cervical incompetence, a vesico-vaginal fistula or rupture of the uterus.

CAUTION! Remove the suture immediately if:
(1) signs of imminent miscarriage develop.
(2) the membranes rupture in the absence of labour.

20.6 'Acute' ectopic gestation

In many parts of the world one in every 50-200 gestations is ectopically (*i.e.* abnormally) implanted, of which 99% implant somewhere along the Fallopian tube. Very occasionally implantation is in the abdominal cavity (20.9), or in the cervix. Trouble occurs either because the tube ruptures, or because the gestation aborts through the abdominal end of the tube, into the abdominal cavity. How soon there is trouble depends on where implantation occurs.

Ectopic gestation is more common if the tubes are affected by previous PID, if an IUD is *in situ*, or if there has been previous tubal surgery.

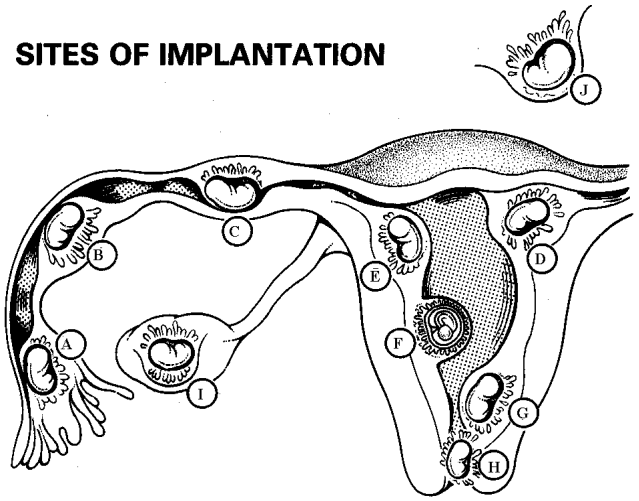


Fig. 20-3 SITES OF IMPLANTATION. A, the fimbria (the most common abnormal site). B, the ampulla. C, the isthmus. D, the uterine part of the tube. E, the angle. F, the body of the uterus (normal). G, close to the internal os, leading to placenta praevia. H, the cervix. I, the ovary. J, elsewhere in the abdominal cavity (including the rectum).

The common sites (20-3A,B) are the distal ⅔ of the tube. Here, the results may be:
(1) an acute or subacute rupture 6-10wks after the last period,
(2) a tubal miscarriage, in which the foetus is expelled into the peritoneal cavity from the free end of the tube, which is not ruptured. Instead, chronic bleeding may continue slowly into the pelvis, to cause a pelvic haematoma (haematocoele).

In the isthmus (20-3C), it ruptures earlier at 4-6wks. In the uterine part of the tube (20-3D), it ruptures early. In an angle of the uterus, or cornu (20-3E), it may proceed to 20wks (20.8). Both close to the internal os (20-3G), resulting in placenta praevia, and in the cervix (20-3H), it leads to antepartum vaginal haemorrhage. On the ovary (20-3I) or elsewhere in the abdomen (20-3J), it may rupture around 14wks. If an ectopic gestation survives to 20wks without causing serious symptoms, it is probably in one of the less common sites, perhaps in an angle.

Patients with an ectopic gestation form 5 groups:
(1) Those who have had a massive bleed into the abdominal cavity. These are the acute and subacute cases described below.
(2) Those with little abdominal bleeding. A few of these 'chronic' ectopic gestations (20.7) will have a massive haemorrhage later, but many will never lose >200ml of blood into the abdominal cavity.
(3) A miscarriage into the abdomen which resolves itself.
(4) The gestation attaches itself to an area in the abdomen or sometimes inside the broad ligament where there is enough room even to grow to term!
(5) Those presenting early because they think they are pregnant, often symptomless, where an ultrasound finds the uterus empty while there is a pregnancy seen elsewhere, or either the history or the β-HCG test indicates there should be a recognizable intra-uterine pregnancy present. There are also various intermediate forms.

Symptoms start when an ectopic gestation grows so large that it ruptures out of the tube that contains it. The periods are usually a few days to a few months late, and the patient may rightly think she is pregnant. Or, she may not think she is pregnant because:
(1) the tube may rupture before she has missed a period.
(2) vaginal bleeding due to decreasing hormone levels may begin at about the time of the expected period.
(3) she may have an IUD *in situ*, be taking a contraceptive, or have had a tubal ligation and assume she cannot be pregnant. If the period of amenorrhoea is short, before the symptoms start, gestation is likely to be in the isthmus, and the effects of rupture worse.

(a) An acute rupture presents as a sudden severe lower abdominal pain, with signs of hypovolaemia. Pain and internal bleeding may be severe enough to cause vomiting and collapse. Peripheral shutdown, tachycardia and drop in blood pressure ensue as shock progresses. Some mild dark red or brown vaginal bleeding usually follows 24h after the onset of the pain, as the decidua are shed if the bleeding has not been catastrophic. Emergency surgery is mandatory.

(b) A subacute rupture typically presents with a history of 3-7 days of weakness, anaemia and abdominal swelling, usually with little pain. The lower abdomen may be tender, with rebound tenderness and guarding, but these signs are often minimal. Blood irritating the diaphragm may cause referred pain at the tip of the shoulder. The presentation may be with diarrhoea and vomiting in up to 40% of cases. Fainting episodes may not be mentioned. Treatment is fairly urgent; you should perhaps cross-match blood first.

(c) A chronic ectopic gestation presents as lower abdominal pain with a growing swelling which is easily confused with PID and does not require urgent treatment (20.7).

The diagnosis is usually easy when there has been massive bleeding in the abdominal cavity but it can be very difficult, especially if there is only a little bleeding.

Remember that any woman with a menstrual irregularity (a period or more missed or periods which have been lighter than usual) combined with abdominal pain and adnexal tenderness on one side may have an ectopic gestation. Anaemia, dizziness, shoulder pain, and a tender mass are all extras which encourage the diagnosis, but are not necessary for it.

A -ve sensitive urine pregnancy test excludes an ectopic gestation, provided the test is from a reputable manufacturer and is not date-expired. If the pregnancy test is +ve and the patient is in pain and you cannot confirm an intra-uterine gestation because it is too early or you have no proper ultrasound, you may be better off performing a laparoscopy or mini-laparotomy as an ectopic gestation is potentially fatal. Even if your diagnosis is wrong, and there is salpingitis or appendicitis in the absence of an intra-uterine gestation, you will have correctly intervened even if for the wrong reasons!

Don't let anyone who might have an ectopic gestation go home; if you opt for observation, make sure you:
(1) monitor her carefully,
(2) are able to operate at very short notice,
(3) have discussed the option of sterilization if you decide to operate.
As so often, 'look and see' is better than 'wait and see'. These are rewarding patients, because they seldom die if you treat them correctly, even if they have bled severely. So be watchful.

DON'T FORGET ECTOPIC GESTATION IN A WOMAN OF CHILDBEARING AGE

The main failures are not to:
(1) consider pregnancy as a possibility,
(2) place importance on known risk factors,
(3) think of an ectopic gestation as a diagnosis,
(4) arrange suitable follow-up.

ACUTE AND SUBACUTE ECTOPIC GESTATION

EXAMINATION. Look for general signs of blood loss (shock and anaemia), and for signs of bleeding within the abdomen. Tenderness may be mild, distension, rebound tenderness and guarding are variable, and may be absent. If there is a large tender mass in the lower abdomen, bleeding has been confined there by adhesions. If you suspect free fluid in the abdomen, try to confirm this (as below). There is often a low-grade fever.
Gently perform a vaginal examination. The important signs are pain on moving the cervix, tenderness in the posterior fornix and pouch of Douglas, and perhaps acute adnexal tenderness, which is worse on one side (highly suggestive).

CAUTION! Don't do a vigorous vaginal or bimanual examination if you are not able to start surgery in 20mins, e.g. *when visiting a rural clinic or when the theatre staff have just gone home:* you may worsen or even restart bleeding!

TESTS
Do a sensitive PREGNANCY TEST, making sure your kit is not out of date!
Check the Hb: it is normal to begin with and only drops as tissue fluid or IV replacements dilute the circulating blood. A few days after a severe bleed, however, you may find an Hb as low as 30g/L.
Check for ORTHOSTATIC HYPOTENSION with a Schellong test: take the pulse when the patient is sitting up; if it is >25 beats/min faster than when she lies down for at least 10mins, this is a good sign of hypovolaemia.
Look for free fluid by ULTRASOUND (38.2,3). In case of doubt, run 200ml of normal saline *via* a giving set and cannula into the abdomen. Then ask the patient to lie on her left and then right side. Then place the bag on the ground. If clear fluid runs back in the system, you can exclude a *ruptured* ectopic gestation. If blood runs down, especially with small clots in it, this strongly supports the diagnosis. *Don't reject the diagnosis, though, on a doubtful result.*
You may see the typical echogenic ring of an ectopic gestation, *but don't waste time looking for this if the patient is unstable!*

DIFFERENTIAL DIAGNOSIS
(a) OF 'ACUTE' ECTOPIC GESTATION

If there is blood present:
(1) Uterine rupture (21.17),
(2) Unsuspected abdominal trauma,
(3) Abdominal tuberculosis (16.1),
(4) Ruptured haemorrhagic ovarian cyst (23.9),
(5) Ruptured abdominal aneurysm (35.8),
(6) Acute haemorrhagic pancreatitis (15.13).

(b) OF 'SUBACUTE' ECTOPIC GESTATION
Most causes of an acute abdomen (10.1, 12.1), and other causes of anaemia.

If the amount of blood lost PV fully explains the extent of shock and anaemia, suspect a miscarriage.

LAPAROTOMY FOR ECTOPIC GESTATION

Prepare the equipment needed for autotransfusion (5-1).

N.B. ABOUT TRANSFUSION: A healthy woman who loses blood fast without having an infusion of fluid will die, if she does so, *not* from lack of red blood cells but from lack of circulating volume. This is the basis of hypovolaemic shock, *i.e. an insufficient response to acute volume deficit.* If then she arrives in shock and is operated immediately and her circulating volume is replaced with normal saline just before and during operation, she will have enough red blood cells left to survive.
However, if she has had volume replacement before arriving in hospital, or in hospital while waiting to be operated upon, or whilst bleeding over a prolonged period, then her circulating volume will *not* be the problem. Note that about ⅓ of saline infused IV will remain in the circulation, therefore losing 2L blood will be compensated in terms of volume by 6L saline or Ringer's lactate. With volume replacement but continuous bleeding, the cause of death is lack of oxygen because of lack of red blood cells.
So, in theory, a patient arriving in your hospital with a ruptured ectopic, operated upon immediately (with a very low dose of ketamine) while the first litre of saline is being infused rapidly, *does not need a blood transfusion*. If the patient has had IV fluids for some time, then it is much more difficult to tell if she needs red blood cells. Also because the blood in her abdomen is now partly diluted blood, she might have received the saline infusion before she was really in danger, and she might have bled after the infusion was set up.

If the total volume of blood (clots) in the abdomen is <1-1·5L (the younger she is, usually the stronger), she does not really need to be (auto)transfused unless she was previously severely anaemic, *e.g.* due to malaria or sickle cell disease. If possible, these patients (with infusions running) should be operated immediately and perhaps autotransfused.

If the patient is stable at the end of the operation and has enough circulating volume and you are certain you have stopped the bleeding, then a blood transfusion is *often not needed*.

What often happens, though, is that it takes so long to organize cross-matching of blood that the first bag is connected to a stable patient 4h or so after the operation. Because she has not actually died, she probably did not really need the blood. However, the first signs of problems are 'oxygen hunger': cardiac failure typified by crepitations over the lung bases, an impossibility to lie horizontally, and confusion. *Rapid transfusion in this situation may well prove fatal due to cardiac overload.*

The first step then is to administer 20-40mg furosemide IV. Fluid overload can also produce this syndrome rather than lack of red blood cells. Check the Hb: if <50g/L, transfuse 1 unit of red cells if available.

Remember transfusions are often just giving you an extra margin of safety. Therefore transfusion is justified if donors are abundant and safe. *If this is not the case, you should be extra-careful with your haemostasis* and manage with smaller safety margins. The above applies also to women bleeding from incomplete and/or induced miscarriages. In those cases bleeding can be often stopped immediately even without access to a fully equipped theatre. Therefore miscarriages very seldom need transfusions.

Make doubly sure bleeding is not still continuing!

N.B. Some teenage or premenopausal women arrive very pale after weeks of dysfunctional uterine bleeding (DUB: 23.3). Other women have a fibroid prolapsing through the cervix.

Often women who are bleeding are extremely anaemic. The reflex of some health workers is to give them a fast infusion of saline when there is nothing wrong with their circulating volume. This extra fluid gives extra work to the heart which is already working to capacity because the heart muscle is anoxic having to circulate the remaining red blood cells much more frequently. This fluid might actually kill the patient as a result of inducing cardiac failure.

The priorities are therefore in severe anaemia:

(1) Administer 20-40mg furosemide IV *if there are already signs of cardiac failure*.

(2) Stop any bleeding (suction curetting with 6mm Karman curette without anaesthesia or twisting off a pedunculated fibroid).

(3) Treat with iron supplementation.

(4) Lastly administer packed red cells slowly *only* if the patient is in mortal danger, and then only in addition to a further 20-40mg furosemide IV.

RESUSCITATION FOR AN ACUTE ECTOPIC GESTATION

Insert 2 large-bore IV infusions immediately and administer saline or Ringer's lactate rapidly. Cross-match blood. *Don't be too enthusiastic to restore the blood pressure above 90mm Hg systolic*, because you might promote more bleeding. Your first priority is to stop the bleeding: resuscitation is to prepare the patient as best you can in the time available. The more fluid you give, the more anaemic the patient will become!

It is possible with an acute massive bleed (*e.g.* after a vaginal examination performed on admission or in the ward) to insert the needle of a blood letting system as used by blood banks, through the abdominal wall into the pool of blood in the abdomen just before the operation. The bag with citrate in it may fill within minutes: hand it to the anaesthetist for autotransfusion (5.3). Scrub up, gown up and operate while the patient is given that blood via a filtered giving set.

Ketamine is ideal for anaesthesia. *Don't use thiopentone for induction:* the blood pressure might crash!

NTABISENG (35yrs) was suspected of having an ectopic gestation in a small mission hospital. Her Hb was 80g/L. The nurse anaesthetist there refused to take the risk of giving anaesthesia and referred the patient to the nearest government district hospital where she arrived 2h later. The Hb being 60g/L by now, the nurse there even more strongly refused to give anaesthesia. The patient was now transported to the provincial hospital where on arrival 3h later her Hb was 40g/L. Neither surgeon nor anaesthetist wanted to intervene, so she was now referred to a central hospital but did not arrive alive.

LESSON The message is clear: *don't think others in more sophisticated surroundings can do better with a patient who is much worse.*

PREPARATION

If you have time, insert a urinary catheter: leave it *in situ*. Since one ectopic gestation is followed in 30% of cases by another, discuss whether the patient wishes any further pregnancies and, if she does not, ask for permission to tie the normal tube.

TSITSI (39yrs) was a para 6 and lived more than a day's travel away from the hospital. She arrived in severe shock from an ectopic gestation over the weekend but was operated with success. The next year, the same thing happened but because there were floods, her journey to hospital took much longer and she died *en route*.

LESSON If she had had the other tube tied, or access to proper contraceptives or had at least an IUD inserted, this totally avoidable death could have been avoided.

PINCH THE BROAD LIGAMENT TO STOP THE BLEEDING

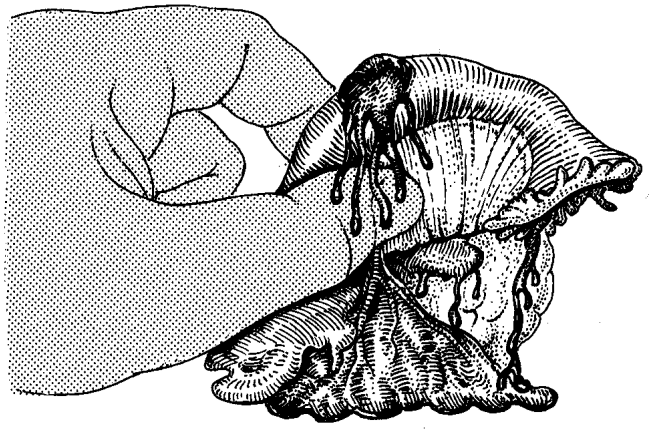


Fig. 20-4 PINCH THE BROAD LIGAMENT TO STOP THE BLEEDING. As soon as you open the abdomen while the patient is in the Trendelenburg position (otherwise the blood will spill over and is not available for autotransfusion), lift out the uterus if possible, find the ruptured Fallopian tube and if it is still bleeding significantly, grasp the mesosalpinx between your finger and thumb, so as to compress and later clamp the vessels and stop the bleeding.

METHOD
Place the patient in a 15º head-down (Trendelenburg) position. Make a subumbilical midline (11.2) or, if you are experienced, a Pfannenstiel incision. There will be blood in the abdominal cavity, which should not spill out and be lost for autotransfusion.

Put your hand into the pelvis and feel for the uterus and lift it out if possible. Find the ruptured Fallopian tube, and if it is still actively bleeding, grasp its broad ligament between your finger and thumb, so as to compress the vessels in it (20-4). Apply long curved haemostats across the tubes on either side of the ectopic gestation (20-5) so that the points meet and you leave no part of the broad ligament unclamped. Preserve the ovary.

N.B. You can put the distal clamp either over the distal tube (20-5X) *or over the remaining broad ligament* (20-5Y), *which will result in removal of the distal tube.*

If you leave the fimbria, it may prove possible later to reconstruct the tube, provided there is >4cm of it remaining, if the patient becomes infertile. On the other hand, it is possible that a zygote fertilized in the contralateral tube might be trapped in the distal part of the amputated tube, resulting in another ectopic gestation.

After you have clamped the ruptured Fallopian tube, the anaesthetist can now raise the BP without just adding to the amount of fluid pumped into the abdomen. Think about autotransfusion (5.3) if there is >1·5L blood in the abdomen: collect it all while slowly returning the patient to the horizontal position. Suck out and discard the last drops of blood, so you can see where to place ligatures at the right place.

Examine both the tubes to make sure that there isn't a double ectopic gestation. The other tube may often contain a little blood and appear violet-blue, but this is not an indication to remove it. The embryo will probably only be about 1cm long, so you won't usually find it. Or, you may find quite a large unruptured amniotic sac containing it. If the other tube seems severely damaged, record it and tell the patient. However, many patients who are told that they cannot become pregnant anymore achieve pregnancy nonetheless.

If there is a subacute ectopic, the ruptured tube will be covered with blood clot and adherent to the surrounding structures. Free it from them with scissors or a finger.

If the patient has no other children and the tube looks reasonable while the other tube is hopelessly stenosed, it is on occasion (provided the patient has easy access to a functional hospital) best to incise the tube at the place of the swelling lengthwise opposite the mesosalpinx. Remove the gestation bluntly, by combining squeezing and using the back of dissecting forceps, and then *very thoroughly* compress the area to stop the bleeding. Put a few sutures in the mesosalpinx around the blood vessels supplying the area. Repair the incision in the tube with 5/0 sutures.

If, however, the other tube looks satisfactory apart from a closed distal end, and the patient is stable, it is occasionally responsible to open that distal end bluntly with a small artery forceps and fix the 'petals' with 5/0 nylon to keep the tube open.

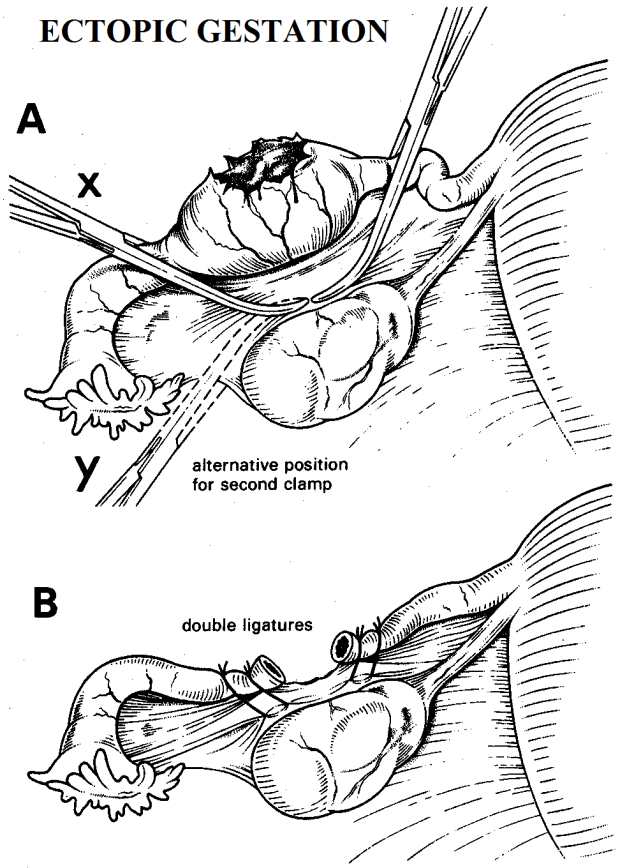


Fig. 20-5 ECTOPIC GESTATION. A, put clamps on either side of the ruptured tube. Try to preserve its fimbrial end if you can (X). If necessary, you can put the second clamp in position Y. B, remove the ectopic gestation, and put 2 ligatures round the Fallopian tube ends.

Remove the ruptured part of the tube by cutting along the free side of the clamps. Place 2 long-acting absorbable ligatures under the joints of each clamp. Tie them with a sliding knot (4.8). Leave the ends of these ligatures long, and hold them in haemostats. Place double ligatures on both sides, to make sure that no arteries are missed.

CAUTION!
(1) Tie these ligatures carefully, or else post-operative bleeding will ensue.
(2) If bleeding continues after you have applied 2 ligatures, re-apply the clamps and repeat the procedure.
(3) *Don't do anything else which is not essential*.

Often, however, it is most reasonable to excise the affected tube completely; if you do so, remove the fimbrial end thoroughly. Lavage the peritoneal cavity thoroughly with warm sterile water. If the patient has previously consented, tie the other tube. Close the abdomen without drainage.

Examine the specimen. In the middle of an ill-defined placenta and blood clot you will see the amniotic sac.

If there is suspicion of gestational trophoblastic disease (23.10), send the specimen for histology.

POST-OPERATIVELY, monitor the urine output until the patient is out of danger. Treat the anaemia with folic acid orally and/or iron or, if the Hb is <50g/L, with blood transfusion. Very rarely will you need >1 unit blood transfusion.

Difficulties with acute and subacute ectopic gestations

If you cannot find the tube with the ectopic gestation, *don't panic*. Allow yourself time to scoop out blood and clots. Make sure you have tipped the head of the table down (the Trendelenburg position), so as to make the blood *and* the bowel move away from the pelvis. Feel for the uterus in the midline in the hollow of the sacrum. Lift it into the wound. If it is stuck down by adhesions, divide them with gentle traction, or cut them with scissors. Having found the uterus, feel for the affected tube. If this is also stuck down by adhesions to the omentum or bowel, separate them (usually not too difficult).

If the tube is stuck to the broad ligament on the same side (more difficult), try to get your fingers under it and the ovary, and lift them into the wound, by scraping the tip of your fingers along the back of the broad ligament. If necessary, cut adhesions between the tube and the rectum.

If the ovary is stuck to the tube, or you have torn it as you mobilized it, you sometimes have to remove it.

CAUTION! Before you remove the ovary (if you have to), make sure you separate adhesions between it and the broad ligament. *If you don't do this, you may clamp the broad ligament too low down, and so include the ureter*.

If adhesions obscure everything, search for the uterus, or the infundibulopelvic ligament (23-21). On the right this comes away from under the caecum and appendix, and on the left from under the mesosigmoid.

The blood supply to the tube and ovary comes from:
(1) The ovarian vessels in the infundibulopelvic ligament.
(2) The ascending branches of the uterine vessels.
If you can put a clamp across the infundibulopelvic ligament, and another one across the tube and broad ligament next to the uterus, you will interrupt the blood supply to the ectopic.

If there is a raw area in the peritoneum which oozes, after you have removed the ectopic gestation, it will usually stop spontaneously, if there are no obviously bleeding vessels. Try compressing if firmly for 5mins. If it continues to ooze, insert a drain for 24h, and monitor the patient carefully.

If you find inflamed tubes with some pus discharging from their fimbriated ends, or evidence of inflammation without pus or abscess formation, this is salpingitis (23.1), not an ectopic gestation. *Don't excise the tubes*: take a pus swab and administer broad-spectrum antibiotics.

If there is a chronic pyosalpinx, excising it will be very risky if it has stuck to the bowel, but this may be possible if it is not too friable and adherent.

If one tube is grossly swollen and full of pus, it is better to remove it anyway if you have opened the abdomen: try not to spread infection, and lavage the pelvis afterwards with warm water. If there is bilateral severe pyosalpinx in a woman with no children, you might prefer to aspirate the pus with a needle and treat with antibiotics.

If there is a tubo-ovarian abscess (23.1), drain it.

If you find the appendix adherent to the tube, peel it off. If you damage it, perform an appendicectomy (14.1).

If there is no ectopic gestation, and you find copious bleeding from the ovary as a result of ovulation, control bleeding with sutures.

N.B. Very occasionally there is blood in the abdomen from a spontaneous rupture of the spleen (15.17) or even from the needle hole you created trying to diagnose an ectopic gestation!

If there is a 2nd gestation in the uterus (very rare), removing the ectopic gestation may not disturb it. If amenorrhoea continues, its presence will soon be obvious.

If there is a large purple haematoma in the broad ligament, the ectopic gestation has ruptured into it, and not into the peritoneum, and may be quite large (12-16wk size or larger).

CAUTION!
(1) *Don't burrow into the lower part of the broad ligament*: you may damage the large venous plexuses there, or the ureter.
(2) *Don't try to control bleeding by suturing deeply*, unless this is absolutely essential: you may tie the ureter by mistake.
Try one of the following 3 methods:
(1) Clamp and divide the round ligament on the same side 2-3cm from the uterus. Check the anatomy (23-20,21).
(2) Clamp the tube and ovarian ligament close to the uterus, but *don't divide them at this stage* (if the anatomy is confused, leave this and do it later). Cut the peritoneum from the round ligament in the direction of the infundibulopelvic ligament. This will open the top of the broad ligament. As you approach the infundibulopelvic ligament, find, clamp and divide the ovarian vessels without including the ureter! This will have isolated the blood supply to the ectopic gestation. Now you can clamp and divide the tube and ovarian ligament. If the ectopic is not already free, a little blunt dissection should free it from the base of the broad ligament. If oozing from the base of the broad ligament does not stop spontaneously, clamp and tie the bleeding vessels.
(3) Or, mobilize the uterus by removing blood clot and dividing light adhesions. Apply two large artery forceps to the tube as before (20-5), but *don't excise the ectopic gestation at this stage*. Cut a ½cm opening in the back of the broad ligament, and squeeze out the haematoma by pressing it from below.
Monitor the patient.

There are several consequences possible:
(1) The haematoma does not reform because the artery forceps have controlled the bleeding. Excise the ectopic gestation, complete the operation in the usual way, and then suture the hole in the broad ligament.
(2) The haematoma reforms. Open the broad ligament more widely, look for a bleeding point, and tie it off.

If you find no specific bleeding point, but only a general ooze, compress the area with a pack, and wait 10mins by the clock. If this controls bleeding, complete the operation.

If a pack fails to control the bleeding, tie or underrun as many bleeding vessels as you can. Be careful to feel for the ureter to avoid including it in a ligature. Trace it from where it enters the pelvis over the sacroiliac joint (23-20). It has a characteristic firm feeling, and you can roll it between your fingers.

If you realize that you have forgotten to perform a tubal ligation when indicated, insert an IUD before the patient leaves hospital.

If you find a 'chronic' ectopic (20.7)**, an angular or cervical** (20.8)**, or an abdominal gestation** (20.9), see below.

20.7 'Chronic' ectopic gestation

When there is extra-uterine implantation, inadequate attachment usually causes sudden bleeding. However, 3 types of ectopic gestation do not:
(1) One which has, so far, only caused a small bleed, with the risk of massive haemorrhage later;
(2) One in which repeated small bleeds have caused a haematoma (pelvic haematocoele, 20-7) containing 100-500ml of blood and clot. This is the 'chronic' ectopic gestation: some of these resolve without treatment, but *don't wait for this to happen*. You can never be sure that there won't be a massive haemorrhage, which may be catastrophic;
(3) A complete tubal miscarriage which has stopped bleeding.

Presentation may be varied:
(1) Lower abdominal pain, perhaps combined with pain on micturition, defecation, or sexual intercourse.
(2) A little dark vaginal blood loss (less than a normal period), perhaps preceded by amenorrhoea, and sometimes with the passage of a decidual cast, which can be mistaken for a miscarriage and thus the ectopic gestation remains.
(3) An enlarging mass in the lower abdomen, adjacent to or centrally lying on the uterus, or in the pouch of Douglas. Occasionally, if the adnexae have a long pedicle, this mass is entirely outside the pelvis. Moving the cervix is painful, but this is not such a reliable sign as in an acute rupture. The uterus is usually slightly enlarged.

The diagnosis of a chronic ectopic can be difficult, and is often missed. Its symptoms are like those of PID; *if the patient has had several similar attacks of pain without any missed periods, she probably does have PID* (23.1).

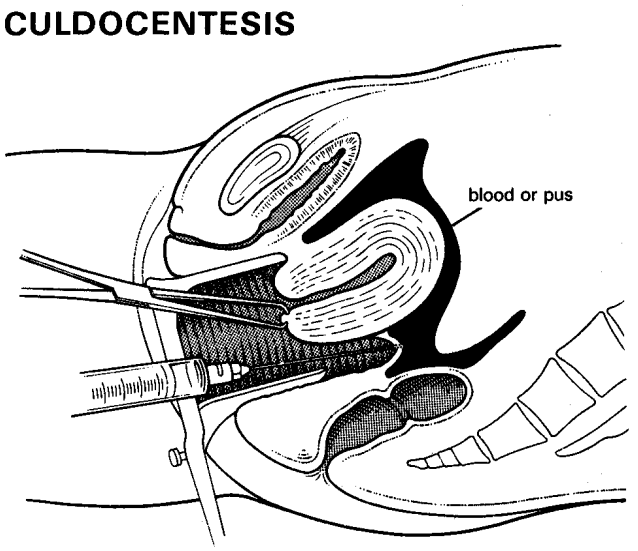


Fig. 20-6 CULDOCENTESIS can be used to confirm the presence of blood or pus in the pelvic peritoneum, and to distinguish between PID and a pelvic haematocoele (chronic ectopic gestation) as the cause of a pelvic mass. If you find clear fluid, this might be from a gestational sac in the pouch of Douglas.

CULDOCENTESIS (20-6) is the confirmatory test for rupture of a chronic ectopic gestation, or a pelvic abscess. You will be able to aspirate blood if the haematocoele is in the pouch of Douglas, but not if it is, rarely, elsewhere.

THERESA (24yrs) was seen in hospital complaining of heavy prolonged bleeding for 5 days. She had missed two periods and said that she had passed clots. She was anaemic, the uterus was slightly enlarged, and the cervix was closed and still bleeding. A doctor diagnosed her as having an incomplete miscarriage, and performed a dilation and curettage. There were few curettings, so he thought that she must have had a complete miscarriage. He prescribed iron tablets and discharged her, but she continued to bleed and to have low abdominal pain. So she went to another hospital where the doctor felt a tender mass on the left side of the uterus. He thought at first that she had an ectopic, but he read the discharge card from the first hospital, which said that she had had an incomplete miscarriage, and a 'D&C'. So he was misled and diagnosed PID with a tubo-ovarian abscess. He prescribed antibiotics, and discharged her. Nearly a month later she went to a private clinic run by a medical assistant. He correctly diagnosed an ectopic gestation, before even doing a vaginal examination, and referred her. The Hb was 40g/L. She had had 5 children, and wanted no more, so at laparotomy the tubes were tied.
LESSONS (1) *Don't be misled by other people's clinical opinions*. (2) Supposed miscarriages may be ectopic gestations. (3) PID can produce symptoms which are very like those of a chronic ectopic gestation. (4) This patient has some of the features of a subacute (severe anaemia), and some of those of a typical chronic ectopic gestation (a history of chronic pain); this shows that there is no sharp differentiation between these 2 conditions. (5) Before you diagnose PID, stop and think whether this could be a chronic ectopic gestation.

Think of a chronic ectopic gestation whenever you see a patient with irregular, missed, or prolonged periods, especially if she has low abdominal pain which began with feelings of fainting, and particularly if she has previously had an ectopic gestation.

LARGE PELVIC HAEMATOCOELE

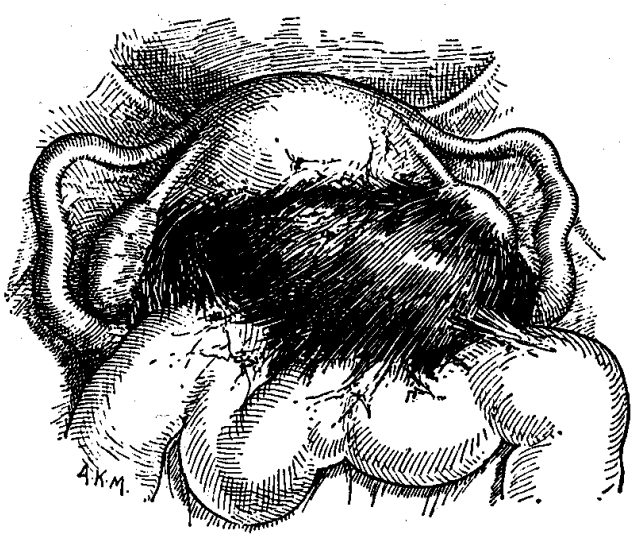


Fig. 20-7 A LARGE PELVIC HAEMATOCOELE (CHRONIC ECTOPIC GESTATION)**. You will only make the diagnosis if you think of this whenever you see a patient with irregular, missed, or prolonged periods.**
From Young J, A Textbook of Gynaecology. 5th ed. 1939, Fig. 101. A & C Black.

DIFFERENTIAL DIAGNOSIS
Suggesting PID (23.1)**:** no missed periods, no anaemia. Fever, lower abdominal pains often worse around menstruation. Vaginal discharge which may be mild. A partner treated for STI. A high ESR. Localized peritoneal irritation (with grimacing on coughing).

Suggesting a threatened, incomplete, or complete miscarriage: significant vaginal bleeding. You can make a decidual cast disintegrate between your fingers, unlike placenta/trophoblast which will have *villi* clearly visible if you float it in a glass of water.

Suggesting a fibroid uterus in pregnancy: a solid mass with much less discomfort.

Suggesting DUB (23.3)**:** irregular periods, anaemia, no mass palpable (although an ovarian cyst may be present).

LAPAROTOMY. Perform a salpingectomy (20.6) and remove all the debris of the dead gestation.

DIFFICULTIES WITH A CHRONIC ECTOPIC GESTATION
If there are many dense adhesions between the ectopic gestation and the surrounding organs, scoop out as much blood clot as will easily come out without tearing and pulling. *Don't try to remove firmly adherent clot;* there will be much oozing. *Don't try to remove the whole 'wall' of the haematoma cavity:* you may injure the bowel.
If you injure the rectum or sigmoid colon, suture the injury and wash the pelvis thoroughly with warm water.
If you injure the small bowel, close the perforation if it is healthy or resect the damaged portion if it is ragged or badly inflamed (11.3). You may, rarely, have to fashion a diverting ileostomy (11.5).

20.8 Angular (cornual) and cervical ectopic gestation

An ectopic gestation occasionally implants towards the medial end of the Fallopian tube. If it implants at the point where the tube enters the uterus, it ruptures early, but if it implants in the intramural part of the tube near the uterine cavity (angular or cornual gestation), it may not rupture until 20wks (20-3). In either case, the whole angle of the uterus becomes a bleeding mass.

If, rarely, an ectopic gestation implants in the cervix (cervical gestation), the cervical os will be open and contain a thin-walled cavity in which you can feel fragments of chorionic tissue. This cavity bleeds massively, and may resemble a miscarriage, where the cervical os is closed tight. Whereas there is little bleeding after a miscarriage has been evacuated, a cervical ectopic gestation continues to bleed profusely (20.2).

MANAGEMENT OF AN ANGULAR ECTOPIC GESTATION

Perform a laparotomy; you will find a purple bleeding mass arising from one angle of the uterus. Bleeding can be torrential. Place a Foley catheter as a tourniquet low down towards the cervix, around the base of the uterus (22.11) if adhesions around it are not too dense. Otherwise get an assistant to compress the angle of the uterus firmly with his fingers. The uterine and ovarian arteries supply this area, so bleeding can be very severe and many patients with this diagnosis will die before they reach hospital.

Remove all products of conception bluntly with your finger and then suture the open area with large bites of #2 long-acting absorbable suture. Alternatively, perform a partial resection, aiming to remove the mass by incising the uterus around its sides. *Be careful that you don't remove too much of the outer layer of the uterus*: otherwise there will be too large a defect to close despite taking big bites.

If control of haemorrhage is inadequate, you may have to proceed to subtotal hysterectomy (21.17). At any rate, unless the patient desperately wishes for more children, ligate the remaining tube, as the risk of sudden rupture of a subsequent gestation at around 28wks is great.

MANAGEMENT OF A CERVICAL GESTATION

Pack the thin-walled cavity in the cervix tightly to stop bleeding, and resuscitate the patient. If the ectopic gestation is early, packing may be all that is needed. Bleeding may have stopped when you remove the pack 24h later.

If a pack does not control bleeding, there are 3 more manoeuvres you can do before hysterectomy: Suture the descending cervical branches of the uterine arteries (as with a cone biopsy).

Pull the cervix firmly down and insert one long-acting absorbable suture from the 2-4o'clock and one from the 8-10o'clock positions, as high as you can at the level of the cervico-vaginal junction. Provided you don't go above this level, the ureters will be safe. Also, if you stay very near or, better, inside the cervix with your suture, you will not tie (but perhaps kink) the ureter. *Don't try any dissection*.

Then insert a large (50ml or more) Foley catheter into the bleeding cavity in the cervix, inflate the balloon, and leave it for 24h. Fluid from the uterus will be able to drain through the tube. It helps to infiltrate the cervix with 10-20ml 1:80,000 to 200,000 adrenaline solution.

If this fails, perform a laparotomy and tie the uterine arteries bilaterally (22-14, 23.15) after they enter the cervix and uterus bilaterally. This way you cannot tie the ureters. You can insert 2-3 sutures on each side 0·5-1cm medially to the lateral margin in the cervix (first from anterior to posterior then the same suture posterior to anterior entering 2-3cm in the direction of the fundus) parallel to lateral margin and then tie them. Do the same in the lower uterine area. *Be careful not to penetrate the bladder* as sometimes it needs to be dissected down. If this too fails, perform a hysterectomy (21.17).

20.9 Abdominal gestation

An ectopic gestation occasionally slips backwards down a tube, or bursts out of it without causing excessive haemorrhage, and embeds itself elsewhere in the abdominal cavity. Sometimes, an ovum is fertilized outside a tube on the surface of an ovary, and then implants itself in the abdominal cavity. Such an ectopic gestation may die at any stage, or proceed to term. An abdominal gestation is thus a rare consequence of a simple ectopic gestation, so that in areas where ectopic gestations are common and not immediately attended to, the incidence of abdominal gestations is increased also. An abdominal gestation causes comparatively few symptoms. Often the diagnosis depends on the sum of many clues.

A patient with an abdominal gestation may present with:
(1) Persistent abdominal pain from c.26-28wks onwards of variable severity, which is not well localized.
(2) The 'uterus' (in reality the gestational sac) is ill-defined, and feels 'odd', when you palpate it. The foetal parts may be abnormally easy or abnormally difficult to feel. The foetal lie is often abnormal, and may be persistently transverse or oblique.
(3) The foregoing features accompanied by the failure of the 'uterus' to enlarge, typically at 32wks, and foetal death.
(4) The foregoing features combined with a 'uterus' that distends more than it should, so that you suspect polyhydramnios.
(5) Postmaturity (>40wks).
(6).Foetal death which is neither expelled spontaneously nor with misoprostol nor oxytocin (20.4).

Other, rarer presentations are:
(7) An abdominal mass after 26wks adjacent to an empty uterus (or a uterus enlarged to the size of a 12-16wk gestation), which is quite separate from it, and which you may think is a fibroid.
(8) A distended abdomen which is like a full-term gestation, and a mass which is less cystic and rubbery than a normal gestation, with accompanied 'menstruation'. On questioning, the patient may later admit having missed some periods, possibly several, in the past.
(9) An abscess in the abdomen rupturing through the abdominal wall. You see foetal bones emerging!
(10) An abscess ruptures into the bowel with passage of foetal bones in the faeces.
(11) A firm mass which you presume to be an ovarian tumour in an elderly lady: it turns out to be a petrified foetus (lithopaedion).
(12) Loss of weight and general ill health.

The diagnosis depends on recognizing that the patient is or was pregnant and that the gestation is not in the uterus.

The history is seldom helpful, but:
(1) she may have had episodes of pain in early pregnancy;
(2) she may have a history of a previous ectopic gestation; or
(3) if she is an experienced multipara, she may say that the pregnancy 'feels different'.

MARY (19 years) was found to have a transverse lie at 7 months. External version failed, so she was allowed to go to term. At 40wks she had abdominal pains, but the lie was still oblique. On pelvic examination the cervix was in a curious position in front of the foetal head. At Caesarean section she did not seem to have a uterus, instead the membranes were close against the abdominal wall. After a live baby girl had been delivered, the placenta was found to be attached to the left Fallopian tube. It was left in place and as many of the membranes as possible removed. She recovered uneventfully.
LESSONS (1) If something rather unusual happens, think of the possibility of an extra-uterine gestation. (2) If you cannot easily remove the placenta, leave it.

(a) Diagnosis
Perform a vaginal examination feeling with the finger through the cervix (often easy in a multipara at term). You will note an empty space, the membranes or presenting part being missing. Sometimes you can feel the fundus opposite from the inside.

(b) Ultrasound: note the gestation attached to an empty uterus (which you may think, at first, is a fibroid).

N.B. You may miss an abdominal gestation on ultrasound if you concentrate on the foetus itself without verifying that it is inside the uterus.

(c) Radiographs
(1) The foetus may be in an abnormal attitude and remain in it over a long period.
(2) In an upright lateral film the foetal parts may overlap the shadow of the maternal spine. This is rare in a normal pregnancy.

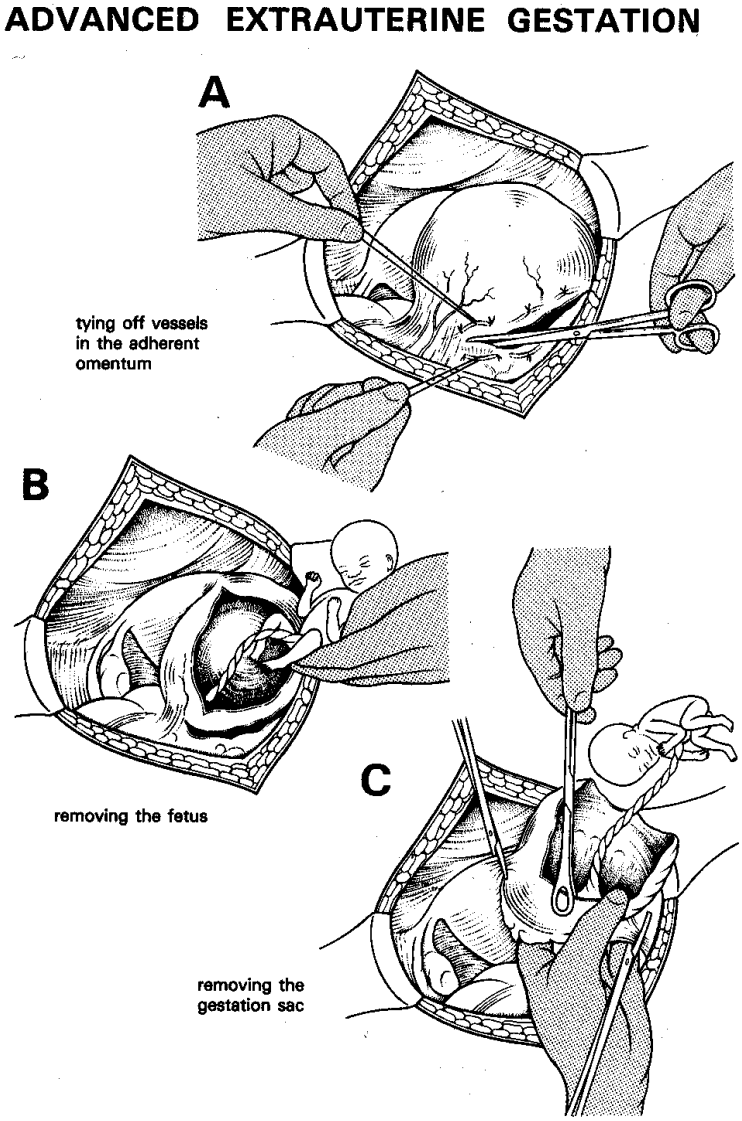


Fig. 20-8 AN ADVANCED EXTRA-UTERINE GESTATION in the omentum. A, tying off the vessels in the omentum. B, removing the foetus. C, in this patient the entire sac is being removed; if it is not easy to remove and has not started to bleed, leave it.
From Bonney V. Gynaecological Surgery. Ballière Tindall, 2nd ed 1974 with kind permission.

The foetus can implant itself anywhere, but because the placenta is so large, it is nearly always attached to bowel or omentum somewhere. The common sites are:
(1) the pouch of Douglas.
(2) the broad ligament, where it is attached to the uterus.
(3) the pelvic wall.
(4) the adnexa.

After 24wks, if the foetus is still alive, keep the mother in hospital to wait until 34-36wks, so as to improve the chances of foetal survival. Often, she has few children or none, and will be grateful for a live child. Bleeding before term, though uncommon, is a real risk, so this is a useful option only if there is always a theatre team available at short notice and there is easy access to blood.

After 24wks, with a dead foetus, postpone intervention for 3-4wks after the foetal movements have stopped, so that the vascularity of the placental bed is reduced.

If the foetus has been dead >6wks, check the clotting time and platelet count before you operate, because of the possibility of DIC (3.5), but plan surgery at the next available opportunity. This is difficult surgery, so refer if you can.

N.B. Sudden severe abdominal pain at any time may indicate haemorrhage, so operate immediately.

GERALDINE (38 years), after years of infertility, finally became pregnant. She kept having abdominal pains but was the last to complain about it. She was so happy and besides she lived far from the nearest clinic. She was referred to the central hospital after she was checked by a midwife in the clinic for oblique lie. That evening she complained about abdominal pains. The junior doctor was summoned and arrived after quite a delay and diagnosed PID in pregnancy and prescribed antibiotics. The ward nurse had phoned the consultant because the junior doctor took so long in coming and, arriving 5mins after the junior doctor had left, he examined the patient properly abdominally and vaginally. He found what he thought was an acute abdomen due to either an abdominal gestation or a uterine rupture. Luckily the patient got to theatre within 15mins. Upon opening the abdomen, much arterial bleeding was seen, but a live baby was delivered. The bleeding caused by the placenta being partly separated from the left adnexa was stopped by compression; the placenta was then removed together with the left adnexa and a large part of omentum. Some large Z-sutures were placed to secure haemostasis against the inside of the abdominal wall.
LESSON *Don't diagnose PID in pregnancy unless you have convincing proof*: other diagnoses are far more likely.

MANAGEMENT
Before 24wks, perform a laparotomy. This may be difficult, so try to refer.

LAPAROTOMY FOR ABDOMINAL GESTATION (GRADE 3.5)
Make sure you have blood cross-matched, and equipment for autotransfusion (5.3).

INCISION. Listen over the abdominal wall for a vascular bruit. This may tell you from where the placenta is getting its blood. Place your abdominal incision away from this site. If you can palpate the foetus, make an incision at that spot. If there is no obvious incision preferred, make a midline incision, extending above the umbilicus.

Open the abdomen with care, because bowel may be adherent to the abdominal wall. Search for the amniotic sac and placenta. Open the sac through a thin area where there is no placenta. If necessary, remove any bowel and omentum from the front of the sac. Dissect away the sac and remove the foetus.

If the placenta is not fixed to the bowel or some other essential structure, and you think you could shell it out quite easily, then remove it. But if it is fixed to the bowel, to the mesentery, to the parietal peritoneum over a large area, or some other vital structure, leave it. Disturbing it will cause severe bleeding.

If the gestation has arisen in a tube and/or ovary, and the sac has a vascular pedicle which you can clamp and divide, remove the sac completely with the placenta.

The placenta may have started to separate and cause bleeding, giving you no choice but to remove the rest of the placenta as gently as possible. If you have adequate blood available for transfusion, removing the placenta will reduce the risk of post-operative adhesions, which may be even more difficult to deal with later.

CAUTION!

(1) *Don't dissect in the region of the placenta*. This may cause catastrophic bleeding, especially if the foetus is still alive.

(2) *Take care not to injure the mesentery, or its blood supply:* part of the bowel may necrose, causing fatal peritonitis.

(3) *Take special care not to injure the large bowel!*

If you decide to leave the placenta, cut and tie the cord as short as possible and expel from it as much blood as possible. Then remove as much of the sac as you safely can.

(4) *Don't insert a drain*: the placenta will be absorbed anyway, and a drain might only introduce infection. Close the abdomen in standard fashion (11.8).

If you cannot control bleeding, pack the bleeding area tightly, taking care *not to include bowel loops in your pack*. Close the abdomen with one end of the pack sticking out of a separate opening lateral to the rectus muscle. It is often feasible to pull out the pack 12-24h later by injecting LA around the opening and gently pulling on the pack. If bowel is adherent or does come out, or there is evidence of further bleeding, proceed to a formal re-laparotomy.

20.10 Antepartum haemorrhage (APH)

In about 50% of patients who bleed antenatally, you never find a cause. Most often the bleeding is not severe and stops spontaneously. However, sudden severe bleeding may suddenly ensue without warning, especially if the cause is due to either (1) placenta praevia, or (2) placental abruption.

It is therefore very important to try to make the diagnosis of these potentially fatal conditions; much the best way is by use of ultrasound (38.2,3), which you should do your utmost to acquire. Otherwise you might have to make the diagnosis by a gentle vaginal examination, which itself may precipitate a massive haemorrhage. You therefore must *only do this using a speculum, never your fingers, unless you are in the operating theatre* with equipment prepared for transfusion and emergency Caesarean section. Alert your anaesthetist. Make sure you monitor your patient carefully! Act always on the side of caution.

BLEEDING AFTER THE 28TH WEEK

Admit the patient, keep her in bed, and monitor her carefully. Record the amount of all the blood lost. Look at its texture: is it mixed with mucus, is it bright red? Measure and record the pulse, blood pressure, and Hb.

Decide on the probable duration of gestation (*don't use the surfactant test* (22.1), because the amniocentesis needle may go through a low-lying placenta).

Record the foetal position, presentation, and lie. Feel for rhythmical contractions. Listen to the foetal heart. *Don't do a vaginal examination with your fingers!*

CAUTION! If you find an abnormal lie, *don't try to correct it*.

She may be:

(1) An emergency with severe bleeding (≥500ml), in shock, or in labour.

(2) A non-emergency with none of these things.

Ask yourself 3 questions:

(1) Has the uterus ruptured (21.17) due to obstructed labour?

(2) Has the scar from a previous Caesarean section (21.14) ruptured? Both these are uncommon causes of vaginal bleeding before labour and even then most bleeding will be intra-abdominal.

(3) How likely is she to have placenta praevia?

Suggesting placenta praevia:

(1) Painless bright red bleeding which may be mild to severe, especially after 32wks, and tends to stop and start again.

(2) A soft non-tender uterus that relaxes between contractions, *if there are any contractions at all*.

(3) An audible foetal heart.

(4) Shock commensurate with the measured blood loss.

(5) A high-lying head or a transverse lie.

N.B. You can only exclude placenta praevia if the head or the breech are deeply engaged in the pelvis.

Suggesting placental abruption:

(1) Painful bleeding which is slight to moderate and which does not look very fresh.

(2) The presenting part is not higher than you expect, and the lie is usually stable.

(3) A tense, tender, woody-hard uterus with poorly defined foetal parts.

(4) Absent foetal heart and movements.

(5) Shock which is worse than expected from the visible blood lost.

(6) Constant lower abdominal pain.

CAUTION! Beware of diagnosing abruption in a patient who has had a previous Caesarean section; rupture of the uterus is much more likely, even if not in labour.

N.B. A placenta praevia is somewhat more likely if there is a previous Caesarean scar because the placenta can grow in the scar. Such a bleed would usually be painless, as opposed to the bleeding related to abruption or rupture.

Suggesting insignificant vaginal causes:

(1) There is <10ml of blood lost.

(2) Bleeding occurs with contractions.

(3) There is no pain between contractions.

(4) Blood is usually mixed with mucus.

(5) Bleeding stops when the membranes rupture.

Suggesting uterine rupture (21.17):

ULTRASOUND (38.3). Look for the position of the placenta.

If there is no obvious abruption, or you don't have an ultrasound (or you suspect the placenta is hidden in the shadow of the foetal head), and the patient is not in labour,

you may perform a very careful speculum examination to see where the blood is coming from, and to diagnose the incidental causes of bleeding.

N.B. It is not easy, and can precipitate bleeding if you do it roughly. Even probing to find the cervix can cause bleeding if there is a placenta praevia.

Resuscitation may need to start immediately. Cross-match blood and make sure that there are always 2 units ready.

Speculum examination

You should know how long it will take you to prepare for transfusion and Caesarean section, before you do this!

Pass a sterile speculum if the membranes have ruptured. Use gentle speculum examination only.

CAUTION! Never perform a vaginal examination with your fingers except in theatre with preparations for a Caesarean section ready: if there is a placenta praevia, you may cause massive bleeding.

Look for:
(1) Cervical erosions.
(2) Cervical polyps.
(3) Vaginitis.
(4) Carcinoma of the cervix.
(5) Varicose veins (rare).
(6) Placenta in the upper endocervix.
(7) The presenting part, *i.e.* a hairy vertex or a buttock!

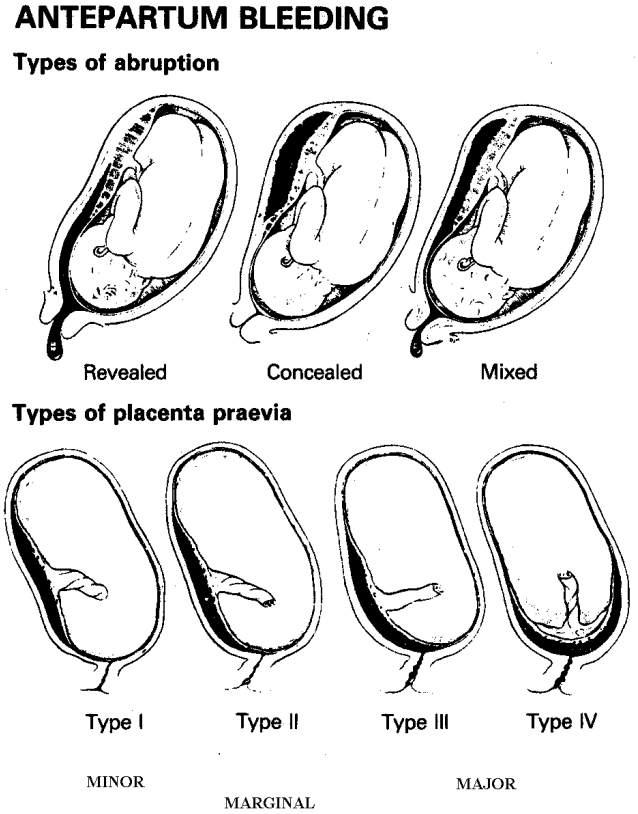


Fig. 20-9 ANTEPARTUM HAEMORRHAGE. The 3 types of abruption, and the 4 types of placenta praevia.
From Moir JC (ed) Munro Kerr's Operative obstetrics Ballière Tindall, 7th ed 1964 Figs 30.1,2 with kind permission.

If there is a placenta praevia, you may see a normal cervix, a haemorrhagic mucous plug, a blood clot in the external os, active bleeding from the cervix, or an open cervix with placental tissue prolapsing out of it. Ask an assistant to press the foetal head into the brim of the pelvis.

Explore the lateral fornices of the vagina very gently with your finger; determine whether there is thickening between the presenting part and the lower uterine segment. If so, ask yourself if this is just on one side of the os (marginal placenta praevia), or all around it (major placenta praevia).

N.B. If you mistakenly do a digital examination inside the os, there will be a boggy feeling of placenta in front of the foetal presenting part, followed often by torrential bleeding as you remove your finger!

If there is an abruption, you will see blood coming out of the cervix. If you mistakenly do a digital examination, *you won't be able to feel the placenta*.

N.B Finding an incidental cause (such as a small polyp) does not mean there cannot also be a placenta praevia, *so beware!* Does the incidental cause look as if it could have caused the bleeding observed? It is wise to assume there might be a placenta praevia until proved otherwise.

If there is *trichomoniasis*, you will see a red vaginal wall and a pale green frothy discharge. Treat this with metronidazole, and aim to treat any sexual partners also.

If there is cervical ectopy, they will usually disappear after delivery and need no specific treatment. Treat any associated *trichomoniasis* or chlamydia. Ectopy seldom causes more than staining of the underwear or spotting, which may be related to sexual intercourse.

If there are vulval varicosities, local pressure will probably stop bleeding. If necessary, insert a suture. Varicosities sometimes occur at the vulva or introitus of older multipara.

If there is a cervical polyp, *don't twist it off during pregnancy*: it may bleed severely. Leave it alone, and deal with it after delivery (23-8).

If there is carcinoma of the cervix, found in the presence of labour, perform a Caesarean section. If the lesion is confined to the cervix, proceed to a hysterectomy if you have the skill.

If there are enormous *condylomata accuminata*, they can easily bleed, especially with HIV-related thrombocytopenia, *but are not an indication for Caesarean section.* You should start ARV therapy (5.8) before resorting to excision of the warts (5.6).

20.11 Placenta praevia

Placenta praevia describes a low insertion of the placenta in the uterus, alongside or in front of the foetal presenting part. Its incidence is increased where there has been previous uterine injury, including curettage, Caesarean section, other uterine surgery, or termination of pregnancy. It is thus more common in multipara, in twin pregnancy, and with increasing age.

The placenta is more likely to be inserted in a scarred part of the uterus and often the placenta is then also abnormally adherent (*placenta accreta*, *increta* or *percreta*); in these cases a delivery without an experienced operator and blood available for transfusion might easily result in a disaster. You should therefore inform a woman, if there is placenta attached to a scar, that a hysterectomy might prove necessary.

The chance of an abnormally adherent placenta is increased when there is a short interval between a Caesarean section and the next pregnancy. Placental migration from the lower segment occurs from about 15wks onwards while the lower segment of the uterus unfolds; this might cause some bleeding. Migration is less likely to happen if the placenta is posterior or over a Caesarean section scar.

Placenta praevia exists when the placenta is inserted wholly or in part into the lower segment of the uterus. If it lies over the internal cervical os, it is considered a major placenta praevia; if not, a minor placenta praevia (20-9). Vaginal ultrasound is somewhat more accurate, less inclined to over-diagnose placenta praevia than abdominal ultrasound (38.3), but requires extra equipment.

Most patients with placenta praevia bleed before labour starts. If you make the diagnosis of a major placenta praevia, admit the woman concerned from 32wks onwards, or much earlier if there is not always fast reliable transport available or if there is no constant companion present. The first bleed may be slight, and subsequent ones increasingly severe, as the area of placental separation increases. Most bleeding is painless and with bright red blood.

If you have no ultrasound to confirm the position of the placenta, to find out from where bleeding is coming, examine the patient in theatre, and get fully prepared for an elective or emergency delivery. The correct timing of this is vital. You can do it early, soon after she presents. Or, if she is not bleeding severely, you can postpone it, and manage her non-operatively in hospital until she reaches 36wks, by which time the chances of foetal survival are almost as good as they would be at term. Most of your patients with placenta praevia will present before the 36th week, so non-operative treatment will improve your perinatal mortality, but it is only justified if Caesarean section is instantly available 24h/day, 7days/wk!

Unfortunately, a major placenta praevia often does not bleed until labour starts. Even so, a high presenting part, or a persistent transverse lie, should lead you to suspect it before. Placenta praevia also increases the risk of puerperal sepsis, and of postpartum haemorrhage, because the lower segment, to which the placenta was attached, contracts less well after delivery.

Management of placenta praevia
Once you have made the diagnosis, you must balance the risks of continuing the pregnancy with delivering the foetus.

At 32wks with minimal bleeding, the balance is in favour of bed rest and careful monitoring (with blood cross-matched and available if necessary). At 36wks you should perform a Caesarean section. Start iron supplements from the time of diagnosis to optimize the Hb level.

N.B. Don't be tempted to do a cerclage or use tocolysis if there are also contractions.

If labour has started, and you cannot immediately perform a Caesarean section, provided the cervix is fully dilated or almost so, and the membranes are presenting at the os, you may be able to arrest bleeding in a multipara with a minor or marginal anteriorly lying placenta praevia by rupturing the membranes, which often results in bringing the presenting part down to press against the placenta. *However, remember delivery will take longer than a Caesarean section, and may compromise foetal viability.* It is of course justified if the foetus has already died. For a posteriorly lying placenta praevia, this will work only if the foetus is very small.

N.B. Don't try this if the os is not dilated >5cm, or with a major placenta praevia.

If the foetus has died, and the placenta is partially prolapsing through the nearly fully dilated cervix, remove the placenta or go past or through it and deliver the foetus by leg traction. The foetal buttock will press on the placenta and reduce bleeding whilst the cervix dilates fully. This may be life-saving for the mother, especially when there is a breech presentation. Otherwise you may be able to insert a hand into the uterus, and turn the foetus round (internal version, 22-8) so that the buttock presses on the placenta.

20.12 Placental abruption

The concordance between clinical and pathologic criteria for the diagnosis of abruption is poor. This should include evidence of retroplacental clots, or vaginal bleeding accompanied by a worrying foetal status or a hypertonic uterus, together with ultrasound visualization of the abruption.

'PLACENTAL ABRUPTION' AFTER A PREVIOUS CAESAREAN SECTION IS ACTUALLY A UTERINE RUPTURE

Abruption (20-9) is not common, and is not easy to treat. The longer you leave a patient undelivered, the worse the prognosis. If there is severe abruption, there is at least a 25% chance of disseminated intravascular coagulation (DIC, 3.5), unless you intervene before 48h. So aim for a vaginal delivery if the foetus has died as soon as you make the diagnosis.

If there is severe abruption, DIC will make it dangerous. The foetus is often dead, and is usually growth-retarded and premature, so cephalopelvic disproportion is seldom a problem.

Here is a rule of thumb with abruption and a dead foetus with a longitudinal lie: if you would be able to do a Caesarean section, *you don't need to do it* (because the situation is not desperate, and clotting factors are still adequate); if you do need to do a Caesarean section (because bleeding is severe), *you can't do it* (because there are almost no clotting factors left in the patient's blood and your incision will cause further bleeding).

The principles of management are therefore:
(1) Replacement of blood loss.
(2) Delivery without delay, preferably vaginal.
(3) Prevention of: postpartum haemorrhage, DIC, and renal failure.

Management of severe abruption
Start a rapid IV infusion of 0.9% saline, or Ringer's lactate, through a wide-bore cannula. Take blood for clotting time and an emergency cross-match. If the systolic BP is <80mm Hg, infuse 1·5-2L of fluid fast; if the BP is 80-100mm Hg, infuse 1L fast. Continue infusion to try to maintain the BP at 100mm Hg.

Start whole blood transfusion as soon as it is available. If you cannot maintain the BP, use un-cross-matched O-ve or even, *in extremis*, O+ve blood. If you have fresh frozen plasma (FFP), infuse 2-4 units rapidly, and add fibrinogen 4g twice, if available. Be aware that:
(1) Heparin is contraindicated.
(2) *Don't use plasma expanders, such as dextran,* because these may precipitate further DIC.
(3) *Don't try to insert a central venous line:* bleeding from puncture of a major vein may, in combination with DIC, be fatal.

Rupture the membranes. Insert a urinary catheter and monitor the urinary output, which should be >50ml/hr. Start a partogram and continue careful monitoring. Stimulate labour with an oxytocin infusion, *unless there is a previous Caesarean scar, or the patient is para ≥2*. Labour is usually fast. Try to effect delivery in 6-8h. Once in the active phase, labour should progress rapidly.

If the cervix is unripe, insert 200µg misoprostol rectally (*not vaginally as it may be washed out*) but then *don't use oxytocin within 3h of misoprostol.*

If the foetus is already dead, which is often the case, expedite delivery by attaching clamps on its head, or inserting a Foley catheter in the foetal rectum. Inflate the balloon with 50ml water in the case of the breech presentation, and apply steady traction with the help of a weight and rope (21.8). This has saved many a mother's life, because pressure of the presenting part speeds up cervical dilation and so delivery.

N.B. The tense, tender, woody-hard uterus will make contractions difficult to monitor.

If the patient is obese or a multipara, with an unfavourable cervix, she is particularly at risk; assess the progress of labour by careful vaginal examination.

The 2nd stage is usually rapid: the dead baby, the placenta, and clot may however all be expelled suddenly, and tear the vagina, perineum, or cervix. *These tears may bleed severely in the presence of DIC.* Try to prevent this by controlled expulsion of the head. If bleeding does occur, correct the clotting disorder with whole blood, FFP and fibrinogen, and pack the vagina (22-10C). Repair the lacerations (21.15,16) and remove the packs 4h later.

After delivery of the placenta, there are often problems, because of a clotting defect, and because there may be *an atonic uterus*. There is a serious risk of postpartum haemorrhage, so encourage uterine contraction immediately after delivery, and administer IV ergometrine with oxytocin ('syntometrine'). Add 15U oxytocin in 500ml of Ringer's lactate or saline, and infuse this fast to keep the uterus well contracted. Add also 800µg misoprostol rectally. Massaging the uterus and expressing the clots at this stage are essential. If bleeding continues, compress the uterus bimanually (22-10A).

Don't leave the patient alone trusting that the medication will do its work. She needs continuous hands-on attention for at least 1h after delivery. A short period of inattention might result in relaxation of the uterus and the loss of a critical quantity of clotting factors.

N.B. Avoid a 'trial of a Caesarean scar' (21.14) when there is an abruption, because you will not be able to recognize or exclude uterine rupture (21.17).

INDICATIONS FOR CAESAREAN SECTION include *only*:
(1) A previously scarred uterus.
(2) Failure to progress, despite artificial rupture of her membranes, oxytocin and traction.
(3) A patient who is bleeding to death *with normal clotting time*. Caesarean section is a desperate step and may save her life.
(4) A live foetus >2kg, with signs of foetal distress.
(5) The transverse lie of a foetus at term for whom vaginal delivery is impossible.

N.B. If you decide on a Caesarean section, do it immediately! *Don't delay till clotting factors are lost!*

If bleeding does not stop after emptying the uterus, deliver it out of the abdominal wound and administer oxytocin and misoprostol. If bleeding continues, put a tourniquet round the lower segment using a rubber urinary catheter (22.11). This may give you time to replenish the circulation, organize blood transfusion, and for the clotting factors to recover.

If bleeding continues, apply a B-Lynch suture (22-13) and if necessary ligate the uterine arteries (22-14) or proceed to hysterectomy (21.17).

21 The surgery of labour

21.1 Two different worlds of obstetrics

If labour does not proceed normally, intervention to help the woman may be necessary. How best you should do this, and what methods you should use, depend greatly on where she is. This has been beautifully described:

> Obstetrically, there are now two worlds, with pockets of one world in the other, and every gradation between the two. In the advantaged industrial world Caesarean Section is now so safe that it has done much to change the whole pattern of obstetrics there. In that world obstetric services are good, and theatres and blood banks well organised. If a woman needs a Caesarean Section, it is done by a skilled obstetrician and an experienced anaesthetist. Antenatal care is available everywhere, transport is easy, and most women are sufficiently educated to understand why they should have a hospital delivery if they need one. Most of them only plan to have 2 or 3 children anyway, and are not frightened by the possibility that Caesarean Section might reduce their chances of having any more. Just because it is so safe, it is used electively for up to even 40% of women as a means of anticipating difficulty, rather than dealing with disaster. It is done so efficiently that seriously traumatic vaginal deliveries and perinatal deaths from birth injury have almost disappeared.
>
> Most women in low- and middle-income countries are less fortunate. A really disadvantaged woman must have 6-7 children, in order to be sure that 3 or 4 will survive. If she has an obstructed labour in a distant village, she may arrive in your hospital after a long journey, dehydrated, ketotic, shocked, anaemic, or infected, or all of these things. If you have to perform a Caesarean Section, you may have to do it through infected tissues, so that it may be followed by peritonitis, which antibiotics may fail to control.
>
> When she has recovered, she may remember only a frightening operation followed by a difficult puerperium, and deliberately not seek hospital care when she becomes pregnant again. If the foetus died, she may blame the hospital for the death, and decide to have the next one at home. Unfortunately, Caesarean Section seldom removes the factor which caused it, the narrow pelvis, which may have been the reason for the Caesarean Section, will still be present. But the scar in the uterus is now its weakest part, so that the chances of it rupturing are great.
>
> *Lawson J. Embryotomy for obstructed labour. Tropical Doctor. 1974;4(4):188-91.*

How can you help a woman like this? She may have no antenatal care in the next pregnancy, and be unable to reach hospital for the next delivery. How can obstetrics be adapted to the needs, without being dominated by the practice of the industrial world?

The answer is to make good use of the alternatives to Caesarean section, and one of the main purposes of this chapter is to describe them.

Unfortunately, in many hospitals, the methods used to assist a woman who has prolonged or obstructed labour are unnecessarily limited. If an oxytocin infusion and a vacuum extractor fail – in some settings, not even those two life-saving options are employed to overcome non-progress in labour – Caesarean section is automatic, and no one thinks of other possibilities. If cephalopelvic disproportion (CPD) is mild, perform a symphysiotomy (21.7). If the foetus is dead, perform a destructive operation (21.8). At all counts it should be possible to learn how to manage a breech delivery (22.7) and other abnormal presentations without resorting to Caesarean section, except as a last resort.

One alternative which needs the hands of an expert obstetrician is the standard type of mid-cavity rotational forceps, such as Kielland's. *In the hands of anyone else, these forceps are so dangerous that the mother and her foetus will be safer if you use vacuum extraction, which you should learn anyway.* So you will find that the only forceps we describe here are Wrigley's outlet ('low') forceps. Perhaps the only acceptable use of the standard mid-cavity forceps by non-experts is their application to the aftercoming head during a breech delivery. For this purpose you can however usually use outlet forceps instead.

The first priority, when a woman is admitted in labour, is to examine her immediately, or to get the most experienced person available to do so. There should be a careful re-assessment ≤4hrly thereafter and observations in between, accurately recorded on the partograph (21-2). *Unless this happens, the whole process of labour management breaks down*.

N.B. In some areas, traditional midwives may use herbs to induce labour, and this may result in inappropriately early and forceful contractions!

Your team will need guidelines to know when to call you. For the most part, they are the same as those for which a health centre refers a woman to hospital. Make it a point that no one should hesitate calling you early, rather than calling too late, even if this means you will get many calls at first.

When you start any operative delivery, make sure that the midwife who is assisting you knows how to resuscitate the baby, and has the equipment ready for doing this. In some hospitals, the results of not doing so are seen only too tragically, in the numbers of handicapped children who attend the paediatric clinics.

Don't forget to relieve pain when you can, so make proper use of pethidine, ketamine and LA.

21.2 Obstetric anaesthesia

Anaesthesia is often the most dangerous part of a difficult delivery. In many district hospitals you can avoid GA in obstetrics; consider also avoiding it for Caesarean section. When the patient is bleeding, or is already hypovolaemic, or very ill, you'll need to manage GA expertly. Unless you use ketamine, you'll need to intubate the patient. *Full GA is dangerous in the circumstances of many labour wards, and the operating theatre may take dangerously long to get ready.*

You can perform most Caesarean sections under subarachnoid (spinal) anaesthesia, provided you take the necessary precautions. You can also use ketamine and LA. For a vacuum extraction, use a pudendal block, with LA for the episiotomy if used, though it is not always necessary. For a destructive operation, other than a transverse lie, use a pudendal block combined with IV ketamine. For a transverse lie, use ketamine or spinal anaesthetic. For manual removal of the placenta, use ketamine.

Epidural anaesthesia is excellent, but may be impractical, except in specialized, well-staffed, well-maintained obstetric units where rigorous aseptic procedures are in place. You will need staff to monitor the patient carefully post-operatively.

Pudendal block for an operative vaginal delivery
This is not 100% effective but may still be useful in certain situations. The danger of a needle-stick injury with a transvaginal or perineal pudendal block is high; you have to locate the ischial spines by palpation through the vagina canal on either side at 4 and 8o'clock.

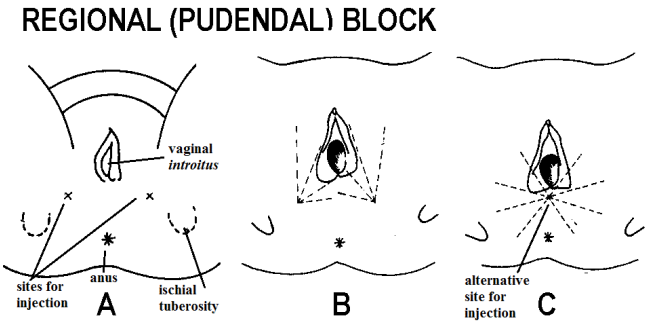


Fig. 21-1 REGIONAL BLOCK FOR AN OPERATIVE VAGINAL DELIVERY. A, position of injection sites for pudendal block through the perineal skin. B, direction of block from each side. C, or from a single central puncture site.
After Howie B. High Risk Obstetrics Macmillan 1986 Fig. 14-6,7 with kind permission.

Raise a skin wheal of LA halfway between the vaginal opening and the ischial tuberosities or centrally (21-1A,C). Use a 12cm x 1mm needle to reach the ischial spines. Inject 12·5ml of 0.5% lignocaine or 1% procaine (both with adrenaline) on each side. Supplement this with superficial infiltration for most operative vaginal deliveries. Withdraw the needle while you inject 25ml of solution in the directions shown. Use a total of 50ml. For an episiotomy and vacuum extraction, superficial infiltration alone may be enough.

CAUTION!
(1) Premedicate with pethidine.
(2) Distinguish the ischial spines from the ischial tuberosities.
(3) *Always aspirate with the plunger before you inject*. If you withdraw blood, move the needle to avoid injecting the anaesthetic solution IV.
(4) Allow the anaesthetic at least 3mins to act.

21.3 Delay in labour

Labour is seldom any problem if it goes at its proper pace. Most trouble starts when it is prolonged or the foetus cannot cope with the temporary lack of exchange caused by contractions interfering with the blood supply to the placenta. Even if the foetus can cope but reacts, for example, to cord or head compression by cardiac deceleration, it is difficult to be sure that no intervention is needed. Most unnecessary interventions have no serious consequences in rich countries but they may have deadly results in other circumstances, directly or a few years later.

(a) The partograph (21-2). In order to manage delay in labour, you must know as early as possible that it has occurred. To know this, you will need an effective method of monitoring labour. The most important part of this is plotting the dilation of the cervix in cm, and the descent of the head in fifths above the brim, against the duration of labour in h.

The purpose of the partograph is:
(1) To prevent neglected obstructed labour and ruptured uterus (which cause 70% of maternal deaths in some areas) by enabling peripheral health workers to monitor labour, to detect deviations from the norm more effectively, and thus to refer women at the optimal moment, before it is too late. **This is the purpose of the 'alert line'**. Ideally, you should only use the partograph to monitor those women whose labour is expected to be normal; *women with obvious 'risk factors' should already have been prepared for assisted delivery*.
(2) To monitor all women in labour in hospital, so that you know when to intervene. This is the purpose of the 'action line'. If the 'progress line' of a woman's cervical dilation moves to the right of the alert line, be extra-vigilant. **If the progress line reaches the action line**, *you must intervene, if you have not already done so.*

The partograph depends on the principles that:
(1) The duration of the 1st stage of labour (though this is difficult to define as it may include false contractions) should not last longer than 8h, hence the thick vertical line at this point.
(2) The latent phase ends and the active phase starts when the cervix is 4cm dilated (3cm was used in the past; it is best to stick to WHO guidelines if these are the rule nationally).
(3) During the active phase of the 1st stage, the cervix should dilate quickly at ≥1cm/hr.
(4) A lag time of 4h is usually acceptable between the slowing of labour and the need to intervene; this is the distance between the alert and the action lines.

N.B. This lag time of 4h is obviously too long if there is a scar on the uterus.

The WHO partograph uses fixed alert and action lines and transfers the patient to the alert line as soon as she reaches 4cm, as has been done for one particular patient (21-3C).

Dilation of the cervix and its relation to the action line is only one of the factors measuring the progress of labour and the necessity to intervene. It and the descent of the foetal head are the 2 most useful and the most easily plotted. Other important factors are:
(1) the presentation,
(2) the moulding (foetal skull bone overlap) score (21.5),
(3) the presence of foetal distress,
(4) the woman's condition,
(5) the duration and frequency of contractions.

Consider all these factors, and *don't be guided only by the dilation of the cervix* in relation to the action line and by the descent of the head, critical though these are. The position of the action line is to some extent arbitrary, and some obstetricians like the alert and action lines closer together.

In some circumstances there is a line halfway in between and parallel to the alert and action line. This is called the transfer line and is used, for example, in peripheral urban clinics to indicate the best time that the woman should be referred to a nearby hospital.

Intervention needs to be earlier in a multipara than in a primipara, so some partographs have 2 action lines, one at 3h for multipara and one at 4h for primipara.

Some hospital partographs leave out the action line altogether and take the alert line as the action line. The important point is that *the further the progress line is from the alert line, the greater should be your vigilance,* and usually the greater your need to intervene.

When the patient approaches the action line, assess all the factors listed above (and others) and decide what to do next, using the guidelines described for normal labour and for obstructed labour (21.4).

N.B. Some hospitals consider that the requirement for some intervention if there is no dilation ≥1cm/hr is 'too active', and leads to an unnecessarily high Caesarean section rate, which is not suitable for populations with an average of perhaps 4-6 children, and when Caesarean section has to be done under less-than-ideal circumstances in small hospitals, so they give the alert line a flatter slope. This is justifiable at 4-7cm of cervical dilation, but >7cm 4h delay is too long.

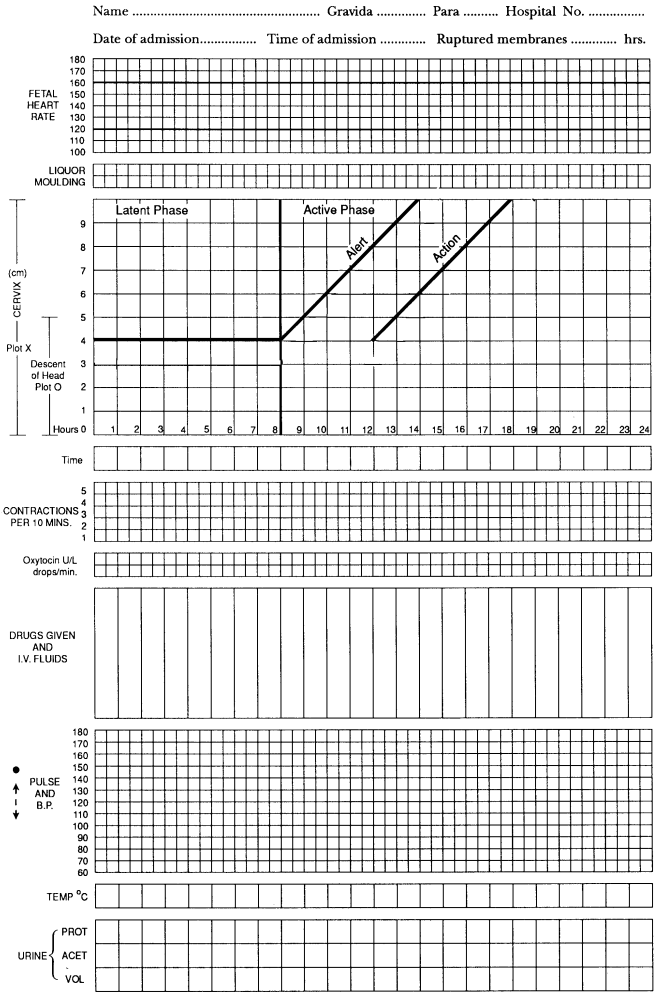


Fig. 21-2 THE PARTOGRAPH is a very useful tool for managing labour. The vertical scale on the left measures cervical dilation in cm and the descent of the head in fifths above the pelvic brim. 'LIQUOR' represents rupture of the membranes and release of amniotic fluid.

Partographs have proved so useful in reducing both maternal and perinatal mortality that not to introduce them might almost be considered criminal neglect. *If you don't already use them, you must! N.B. Partographs don't tell you about risk factors present before labour!*

ARE YOU AND YOUR RURAL CLINICS USING PARTOGRAPHS?

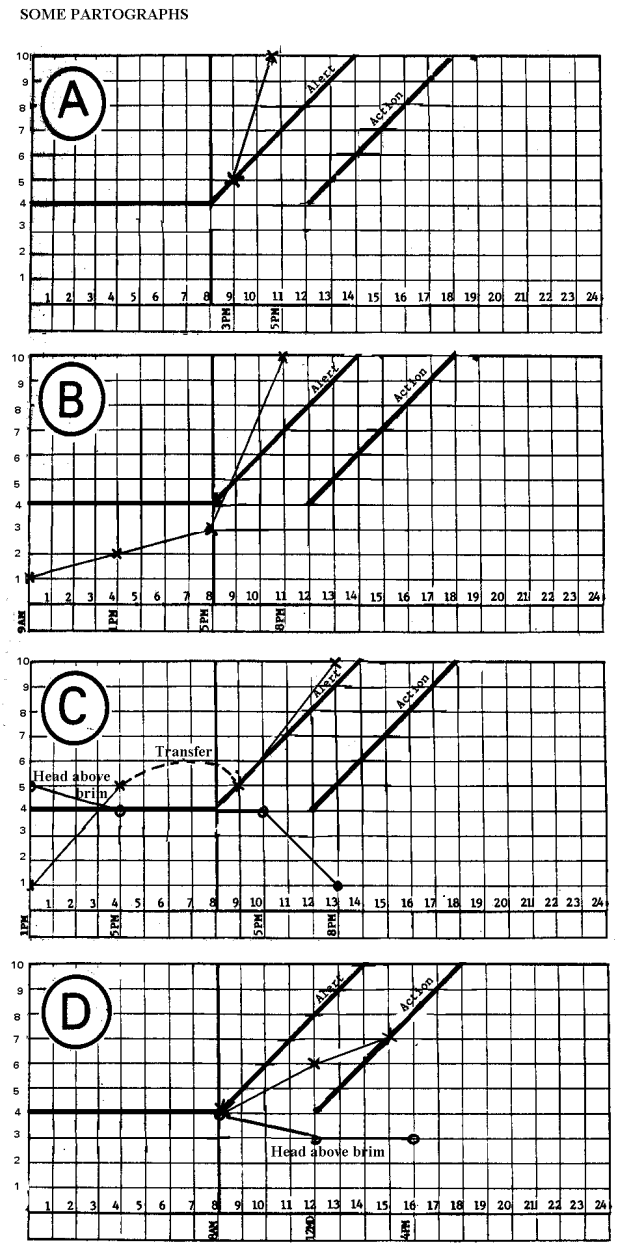


Fig. 21-3 SOME PARTOGRAPHS. If you don't have enough partographs for every woman, put a clean sheet of X-ray film over one of them, write on this with a marker pencil, and then wash the film clean for the next patient.

Woman A was admitted at 3pm 4cm dilated in the active phase of labour; the progress line remained to the left of the alert line and she delivered normally.

Woman B was admitted at 9am 1cm dilated; the latent phase lasted 8h and the active phase 3h. She quickly passed to the left of the alert line.

Woman C was admitted at 1pm 1cm dilated with the foetal head $^{5}/_{5}$ above the pelvic brim. At the next vaginal examination (5pm) the head was $^{4}/_{5}$ above the brim and she was 5cm dilated. She was therefore transferred to the 'alert line'; the cervix continued to dilate, the head descended, and she delivered normally.

Woman D was admitted to a health centre with the foetal head $^4/_5$ above the brim and the cervix 4cm dilated, so she was put on the alert line. At 12 noon she was only 6cm dilated and had moved to the right of the alert line, so she was transferred to hospital. When she arrived at 4pm she was still only 7cm dilated and had reached the action line. The head was $^3/_5$ above the brim, with a moulding score of 3 (21.5)**; it was not possible to put a finger between the head and the pelvic wall. So, following the indications outlined** (21.4)**, she underwent a Caesarean section. If however she were a nullipara, the foetal heart action excellent and an anaesthetist available within 1h, a trial of oxytocin (for a maximum of 1h) might still prevent a Caesarean section. Alternatively a symphysiotomy** (21.7) **may allow a normal delivery.**

THE CRITICAL AREA IN A PARTOGRAPH

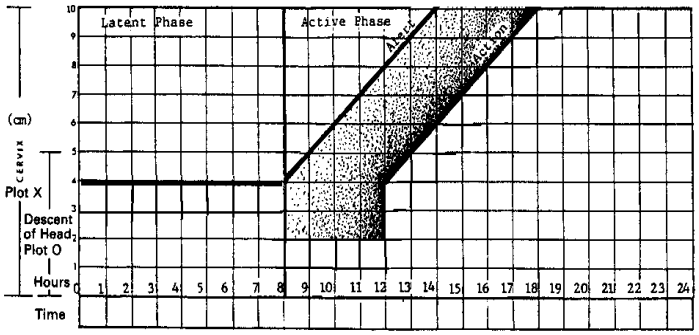


Fig. 21-4 THE CRITICAL AREA IN A PARTOGRAPH.
In a peripheral unit, if a woman's progress line reaches this area, she should be referred. In hospital, it is the area in which you should consider intervening; the darker the shading, the more important this is. ***Don't let her cross the action line without careful reassessment!***

METHOD

Make sure this is not actually obstructed labour (21.4) by excluding:

(1) severe moulding (21.5) and caput (soft scalp swelling, caused by the foetal head being pressed against the cervix),

(2) foetal distress,

(3) stretching of the lower segment, where the peritoneum becomes firmly attached to the anterior uterine wall (21.5),

(4) bloody urine.

N.B. DELAY IN THE LATENT PHASE (primipara and multipara) *is present if a patient who was 'admitted in labour' has not reached the active phase after 8h*.

Distinguish 'false labour' from a truly prolonged latent phase. Recognize false labour by:

(1) membranes still being intact, and

(2) the cervix of a nullipara remaining long and closed (or just admitting a fingertip), or the cervix of a multipara being not effaced, *i.e. thinned and shortened* (even though it may be 1-2cm dilated).

Explain that she is not in labour, and return her to the waiting shelter. If she insists that she feels painful contractions, administer pethidine 100mg IM, let her sleep, and then review her after 3h.

N.B. If you artificially rupture the membranes and start an oxytocin infusion, you inevitably pass the point of no return and increase the chance of needing to perform a Caesarean section. This is only really indicated if you want to stimulate labour for example because of pre-eclampsia, gestational diabetes, or sickle cell disease.

If the latent phase is truly prolonged, the cervix is completely effaced, but remains undilated at c.2-3cm, or effaces and dilates very slowly, you have 2 choices:

(1) Use sedation with pethidine 50-100mg IM, repeated if necessary, and allow mobilization.

(2) Administer very low dose misoprostol 20µg 3hrly (4ml of a 100µg (½ tablet) dissolved in 20ml water) if there is an indication to induce but not strong enough to risk a Caesarean section by rupturing the membranes. Once labour starts, stop the misoprostol!

(b) Delay in the active phase in primipara

If the progress line for a primipara approaches the action line, there may be simply a decrease in contractions, or obstructed labour.

Perform a Caesarean section if there is:

(1) gross cephalopelvic disproportion: head $^4/_5$ above the brim and marked moulding,

(2) a malpresentation, or

(3) foetal distress.

If there is no evidence of malpresentation or foetal distress, first correct dehydration and ketosis with an infusion of IV saline or Ringer's lactate, and provide adequate analgesia: either by a lumbar epidural block or use pethidine 100mg + promethazine 25mg IM.

Then *in a primipara* where there is no foetal distress, nor scar in the uterus (also from myomectomy for example), stimulate the uterus with oxytocin and try to decide if the CPD is significant.

N.B. Using oxytocin in women who have previously delivered is increasingly dangerous with each delivery. Meconium-stained liquor is in itself not a contraindication to the use of oxytocin but very good monitoring is mandatory. Often the first sign of impending uterine rupture, rare in primipara, is foetal distress. Women who have previously delivered vaginally are anyway less likely to have a decrease in contractions after real labour has started and oxytocin is less likely to be of benefit in this group.

(Of course, you can use oxytocin to start labour in a multipara whose membranes have already ruptured but it should be stopped as soon as labour is established. *Note this is then induction, <u>not</u> augmentation, of labour*.)

Start an infusion of a solution of 5U oxytocin to 500ml of 5% dextrose at 10 drops/min, and increase the rate of the infusion by 10 drops/min at ½hrly intervals, until there are contractions lasting 45-60secs at a frequency of 3-4 in 10mins. (This means the first increment will be 20drops/min, and 30mins later 30drops/min.) As soon as you witness good contractions, *don't increase the speed of the IV infusion anymore*. You may even decrease the drop rate if the foetal head is coming down and is well applied to the cervix.

If good contractions (3-4 in 10mins lasting >40secs) don't clearly give rise to progress, and the membranes are ruptured, then there is an indication for a Caesarean section unless the cervix is nearly fully dilated.

N.B. It is difficult to know what good contractions are, however. The frequency is easy to monitor, but the intra-uterine pressure is measurable only with a transducer in the uterus and even then, although low pressure is an indication for oxytocin, proper pressure does not mean the pressure is directed by coordinated contractions in the right direction. Consequently, estimate the length of contractions as an indicator of their strength: <20secs is weak, 20-40secs moderate, > 40secs is strong.

If you are sure labour has started, the cervix is dilated ≥4cm and the membranes are not already ruptured, rupture them. *Remember the risks of prolonged rupture of membranes!* Monitor the mother's progress and the foetal condition carefully. Monitor the heart and watch for signs of foetal distress, especially a slowing of the foetal heart.

N.B. Meconium staining of the liquor is common, but is an unreliable sign.

If you detect signs of foetal distress, stop an oxytocin infusion if one is running.

N.B. Oxytocin itself can, of course, cause such strong, long, or frequent contractions that foetal distress ensues. Stopping the infusion and restarting it later at a lower rate might still result in the vaginal delivery of a healthy baby. On the other hand, some foetuses just cannot cope with inadequate contractions; if the contractions are optimized with oxytocin to cause further cervical dilation, the foetus is in danger. This is then definitely an indication for a Caesarean section, with poor descent of the head or even when almost full cervical dilation has been reached.

If there is severe CPD, which you can recognize by (1) inadequate cervical dilation and foetal descent, especially associated with foetal distress,
(2) cervical dilation proceeding at <1cm/hr at the end of 4h, with no descent of the foetal head, or
(3) the foetal head remaining high, with moulding,
perform a Caesarean section.

N.B. Inadequate cervical dilation and foetal descent without foetal distress may suggest the option of a vacuum extraction (sometimes combined if necessary with a symphysiotomy, 21.7).

(c) Delay in the active phase in multipara

If the progress line for a multipara approaches the action line, this is serious, and you will need to assess the situation carefully. *Don't try to stimulate the uterus with oxytocin*, unless you are as sure as you can be that there is no CPD (21.6). This is difficult to be sure about, and if you are wrong, and there is CPD, the uterus may rupture. A good rule is: *no oxytocin for **augmentation** of labour in multipara* unless you supervise it personally (not over the telephone) *and certainly not for multipara >3, and for not longer than 2h!*

N.B. For a multipara whose membranes have ruptured, for example, it is acceptable to start labour carefully with oxytocin and reduce or stop the infusion as soon as labour is established. This applies also to a breech presentation.

If you are sure labour has started (the cervix is >3cm) and the membranes have not already ruptured, rupture them, *but take cautions regarding HIV exposure*. Labour goes faster in multipara and prolonged exposure to vaginal HIV is less likely. Under close supervision, it is reasonable to start oxytocin at 4-6cm cervical dilation and if good contractions ensue, then to rupture the membranes.

If you are in doubt, observe for 2h more with adequate analgesia, and then reassess the patient. Feel the contractions yourself. She may progress to full dilation even when there is major CPD. You can only detect this by finding severe moulding and caput, with failure of the head to descend, and no progress or movement (>20secs in the 2nd stage, 21-5B,C).

CAUTION! Some women have 6 or 8 normal labours, and then need Caesarean section for CPD with their next pregnancy. *Don't forget to offer a sterilization with the Caesarean section in such a situation*.

(d) Difficulties with delay in labour

If there is delay in the latent stage, look carefully for hidden CPD. Provided there is a vertex presentation, it is always worth sedating the patient and waiting a little to see what happens.

N.B. CPD is almost impossible to diagnose when the membranes are intact and rupturing the membranes might be a good idea if the cervix is very ripe. Even if you are using oxytocin properly, its use with an unripe cervix will increase your Caesarean section rate.

If there is foetal distress with an oxytocin infusion running, *stop it*; turn the patient onto the left side, do a vaginal examination to exclude prolapse of the cord, and make sure she is adequately hydrated and administer oxygen. If the signs are not relieved, proceed to Caesarean section.

21.4 Obstructed labour

The exact point at which lack of progress (21.3) becomes **obstructed labour** is arguable. This is the failure of the presenting part to descend despite good uterine contractions. What really distinguishes prolonged labour from obstructed labour are the secondary signs and complications that follow: severe moulding (21.5), caput (21.3), intra-uterine infection, prolonged suffering, fear of death, pain between contractions, foetal distress, a stretched lower segment (21-5), bloody urine, fistulae and rupture of the uterus. Whereas delay in labour is common and often readily treatable, and is comparatively harmless, obstructed labour is none of these things. *It should never be neglected if care is adequate.* Arguably, obstruction is not uncommon (*e.g.* a failed properly executed vacuum extraction) *but obstructed labour should never be neglected*.

(a) Recognized scenarios of obstructed labour are, for example:
(1) needing 3h to organize a Caesarean section after a failed vacuum extraction, without knowledge of how to perform a symphysiotomy (21.7); or
(2) having waited for 2h with the mother actively pushing, without having attempted a probably easy vacuum extraction and needing 3h to organize a Caesarean section.

(b) Causes of obstructed labour may include:
(1) An abnormality in the pelvis (a contracted pelvis): a true conjugate (antero-posterior diameter at the pelvic inlet) should be >9cm, and the diagonal conjugate (transverse diameter) 1·5-2cm larger.
(2) An abnormality in the foetus (hydrocephalus, etc.).
(3) An abnormality in the relationship between them, either:
(i) An abnormal lie or presentation (breech, brow, face, shoulder presentation, or prolapsed arm in a transverse lie),
(ii) An unfortunate coincidence of their relative sizes.

Cephalopelvic disproportion means the foetus may be too big for the mother, or *vice versa*.
(4) A marginal CPD and/or soft tissue resistance combined with contractions which are not optimal, or poor pushing by the mother.
(5) Rarer causes, such as stenosis of the vagina (particularly after FGM), locked twins, or a pelvic tumour, particularly fibroids or an ovarian cyst.
N.B. CPD is the most important cause (>65%), and an impacted transverse lie is the next most.

In well-nourished mothers (but not over-nourished) with an adequate pelvis, the occipito-posterior position is often involved in obstruction. This partly explains why the same woman often delivers the second, heavier, baby vaginally without problems.
In over-nourished women, the large size of the foetus and perhaps weaker contractions and fat in the pelvis are responsible for much obstruction.
Vitamin D deficiency seems to be involved in weak contractions. This is important for women whose diets are poor in vitamin D, and who cover themselves nearly completely when they venture outside. This is especially important if they live in climates with little sunshine and when they also have much pigment to block UV light.

(c) Antenatal care screening is mainly to detect women who are at risk from obstructed labour, although the sensitivity and specificity of this screening are quite limited.
The purpose of the partograph, however, is to detect it early in labour and it works (with cephalic presentations in single pregnancies) much better than screening.
In practice, when the presenting part stops moving through the birth canal, you may not be able to tell if this is because:
(1) the uterine contractions are weak (uterine inertia), or
(2) there is CPD.
Often in a primipara, there is a combination of inertia and CPD.

(d) Preventing obstructed labour depends on several factors:
(1) Good nutrition starting in childhood and good health promoted by vaccinations, hygiene and mosquito nets so that women reach their genetically determined height and the pelvis its genetically determined size before the first delivery.
(2) Avoiding teenage pregnancy where a girl has to deliver before the pelvis has reached its maximum size.
(3) Universal antenatal care, so that obstructed labour can be anticipated from the history and any risk factors identified.
(4) The monitoring of labour by skilled staff, so that a woman can be referred at the first sign of danger, before she obstructs.

Detailed preventive measures are:
(a) screening for risk factors, especially short stature, *and*
(b) the routine use of the partograph.
When adequate antenatal care is impossible, and where health centre and hospital beds are limited, the establishment of a 'mothers' waiting area or temporary village' is a useful alternative.

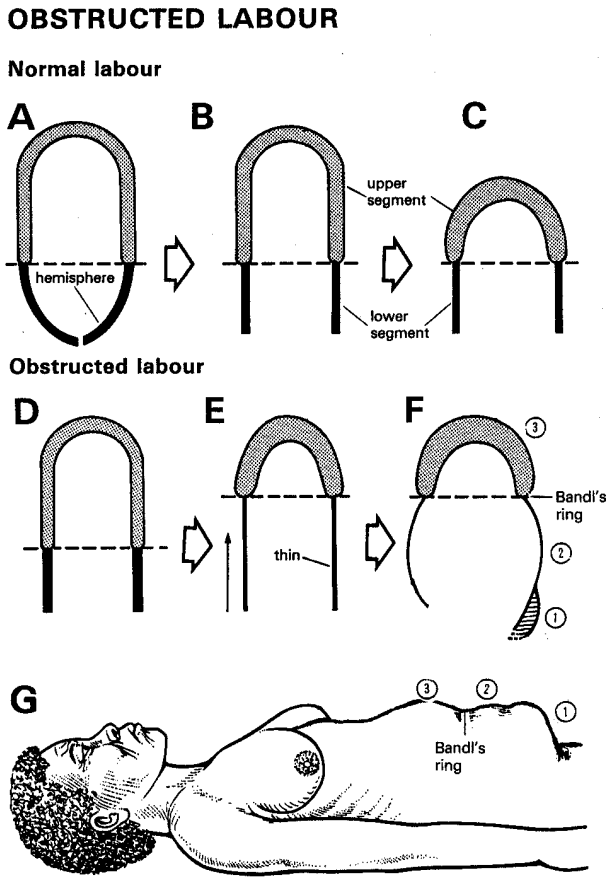


Fig. 21-5 OBSTRUCTED LABOUR.
A,B,C, during a normal labour, the hemispherical lower segment is converted into a cylinder: it thins but does not elongate. During the 2nd stage, the uterus shortens itself by contraction of the upper segment. D,E, during an obstructed labour, the uterus cannot empty, so the thinned lower segment elongates. F, sometimes a palpable (Bandl's) ring forms between the upper and lower segments. G, from the side, you may sometimes see or feel three distinct abdominal swellings: (1) the bladder, (2) the lower segment, (3) the upper segment. Bandl's ring separates the lower and the upper segments.
After Lawson JB, Stewart DB, Obstetrics and Gynaecology in the Tropics, Edward Arnold 1967 Fig 11.2. with kind permission.

(e) ***Neglected obstructed labour*** *is a major failure of obstetric care*. Unfortunately, it still happens, even in some hospitals. It often depends on the prevalence of CPD in your area, and the quality of your antenatal and obstetric care. Alas, the poorest communities with the worst health services are usually those with the most CPD.

A warning sign that labour is going to obstruct is a prolonged 1st stage, but this may be normal or even short. The membranes rupture, and amniotic fluid (liquor) escapes. The uterus contracts and retracts, and forces the foetus into its lower segment, which gradually becomes overstretched.

Foetal escape is obstructed, so the lower segment moulds closely round and thins. The contractions of the uterus become hypertonic, and relaxation between them poor. The placenta is poorly perfused, there is foetal distress, and the foetus dies.

Obstructed labour has 3 main dangers:
(1) Stuck between the foetal head and the pelvis, the vaginal, bladder, and rectal walls become squeezed, and eventually become necrotic, slough, and develop fistulae.
(2) Often in a primipara, the contractions stop while the head is wedged in the pelvis, causing sepsis and/or fistulae.
(3) In a multipara, the uterus keeps contracting till it ruptures or a (nearly) dead, severely moulded baby is delivered.

A primipara begins to have trouble when the cervix fails to dilate normally. An oxytocin infusion may speed up labour if the CPD is minimal or the lack of progress is caused by poor contractions, but cannot do so if CPD is gross. The result is that the labour usually obstructs before she is fully dilated, although she will usually reach full dilation eventually. The results, if the situation is not rapidly relieved, are dire:
(1) Asphyxia and infection in the foetus ensue, owing to prolonged uterine contractions reducing the placental blood flow.
(2) The foetal head is damaged, so that brain injury may result.
(3) Pressure necrosis and sloughing of the vaginal wall occur. As this slough separates, a fistula develops anteriorly between the vagina and the bladder (21.18), or posteriorly between the vagina and the rectum (21.19). The fistula may involve the proximal half of the urethra and/or the neck of the bladder, up to its ureteric orifices, or involve the rectal sphincter mechanism.

MPHO MOKETE (14yrs, para 0, gravida 1) became pregnant after her 1st period. She hid the pregnancy from her parents, and so received no antenatal care. She arrived at hospital exhausted, anxious, and febrile, with a fast pulse. Her contractions were strong and painful, with little relaxation between them. The head of the foetus, showing signs of foetal distress, was high, and overlapped the brim of the pelvis. The liquor had drained, so that the uterus was moulded around the foetus.
The vulva and cervix were oedematous, and although the foetal head could be felt just inside the cervix, this was not because it had descended, but because it was severely elongated. Abdominal examination showed that most of it was still above the pelvic brim. The vagina was dry and 'hot', and the cervix not fully dilated.
The bladder was distended. Catheterizing it was difficult, and the foetal head had to be dislodged by putting two fingers into the vagina and pushing it up. The bladder was drawn up so high that the catheter had to be passed a long way before any urine flowed; when it did so, it was blood-stained. The foetus was alive, and the foetal head was 4/5 above the brim, so neither symphysiotomy nor vacuum delivery were suitable. She was therefore resuscitated with IV fluids, treated with antibiotics, and delivered by Caesarean section. The baby survived with a neurological handicap, but the wound became infected, and she developed a pelvic abscess, which was drained. She was in hospital a month, and was lucky not to develop a fistula.
LESSONS (1) The decision to perform a Caesarean section was correct. (2) She was only 14, and so the pelvis will continue to grow. (3) She is at risk of a ruptured uterus in future, so she must deliver in hospital.

Later, as the ring of necrosis in the vagina heals and contracts, it stenoses. If the mother does not die, she delivers an injured, severely moulded dead baby.

She is also at risk from septicaemia, peritonitis, peritoneal abscess, atonic postpartum haemorrhage (22.11), and foot drop from the pressure of the foetal head on the pelvic nerves. Even if the fistula can be repaired, she may be infertile, and the vagina may be so stenosed that sexual intercourse is difficult. There are of course also serious psychological consequences.

If the fistula is successfully repaired, and she becomes pregnant again, she must have a Caesarean section to prevent the repair breaking down. If the fistula is not repaired (in which case she is less likely to become pregnant), stenosis of the vagina is likely to prevent vaginal delivery.

A multipara may show the same failure to dilate as a primipara, or the cervix may dilate normally to begin with, and then slow during the active phase, only to dilate finally if she is left untreated. Meanwhile, the presenting part fails to descend.

MAPULESA (35yrs, para 8, gravida 10) arrived at hospital just before the uterus ruptured. She too was anxious, distressed, and febrile. The cervix however was fully dilated. The lower segment had continued to retract and thin, so that the junction between the upper and lower segments had risen in the uterus as far as the umbilicus. She had a 'three-tumour abdomen' when viewed from the side: an oedematous distended bladder, a distended, tender lower segment, and a tonically contracted upper segment. A (Bandl's) ring could be felt through the abdominal wall between the upper and lower segments (21-5G). The round ligaments (21-18) stood out on either side of the ballooned lower segment, like the guy ropes of a tent. A brow presentation was felt on vaginal examination. She was resuscitated with IV fluids, and had a Caesarean section. At operation the uterus was found to have ruptured into the abdominal cavity. The foetus was alive, but was asphyxiated, and died within 1h. The uterus was repaired, the tubes ligated, and she recovered uneventfully.
LESSONS (1) Even a patient who has had many normal deliveries may get an obstructed labour from a malpresentation, malposition, or just because babies tend to become larger with higher parity and males are also on average larger and have larger heads than females. (2) A partograph would have given earlier warning of the impending obstruction. (3) Tubal ligation was essential.

(f) Rupture of the uterus is the critical event, in a patient like this. It usually starts in the thin lower segment, and extends downwards on one side into the vagina, as well as upwards towards the fundus. Several things can then happen:
(1) The presenting part may remain jammed in the pelvis.
(2) The foetus may be expelled through the rupture into the peritoneal cavity.
(3) Bleeding from the ruptured uterus may be seen in the vagina.
(4) Occasionally, the bladder also ruptures, especially if it is stuck to the scar of a previous lower-segment Caesarean section.

Before rupture, the signs that it is imminent are:
(1) Failure of labour to progress. Lack of progress should therefore alert you to the possibility that rupture might be imminent.
(2) Bandl's ring.
(3) A distended bladder which is difficult to catheterize.
(4) Frequent strong uterine contractions, with little or no pause between them.
(5) Persistent pain between contractions in a restless, anxious patient.

After rupture, a woman may have little or no pain. Typically, contractions were strong, but then suddenly stopped, and were replaced by a lesser continuous pain, or no pain at all. She may be alert and even talkative, or quite obviously collapsed in severe hypovolaemic shock, with cold, sweaty skin, and a weak or absent radial pulse. She might survive only 10mins or 72h. You feel no uterine contractions, but you can usually feel the foetus through the abdominal wall lying free in the abdomen. Often the presenting part, when examined vaginally, has disappeared and cervical dilation has decreased (21.17).

A multipara who is not steadily progressing in the 2nd stage (when the cervix has dilated fully) and has started to push but has not delivered after 60mins is in great danger.

N.B. Most fistulae occurring after uterine rupture are complications of the surgery, not the labour.

21.5 Managing obstructed labour

If a woman with obstructed labour is admitted from home, she may have been in labour for days, and tried many home remedies. Her stomach is likely to be full, and she can inhale its contents only too easily if she vomits. She is thus a major anaesthetic risk. There are several ways in which you can deliver her foetus, but *you should never use mid-cavity or rotational forceps, such as Kielland's* (21.1).

Vaginal delivery is often possible, but try to predict when it is going to be difficult, so that you can avoid a 'failed vacuum', and perform a symphysiotomy (21.7) or a Caesarean section (21.9) from the start, especially when there is foetal distress. However, judging whether vaginal delivery will be successful by clinical vaginal examination can never be perfect. If you are prepared to perform vacuum extraction combined, in case of failure, with a symphysiotomy, you will be able to avoid around 25 subsequent Caesarean sections if your judgement is only wrong in 10% of cases. *This statistic is only invalid if you perform a tubal ligation at the same time as the Caesarean section.* The side-effects of one symphysiotomy are different but certainly *not* more dangerous than the side-effects of one Caesarean section, and the side-effects of 25 Caesarean sections are naturally overwhelmingly more serious and frequent than those of one symphysiotomy.

N.B. Of course a symphysiotomy is nearly absolutely contraindicated if the foetus has died.

An assisted vaginal delivery (apart from shoulder dystocia which needs a combined vaginal and abdominal approach) *is absolutely contraindicated if the uterus has already ruptured:* perform a laparotomy. Often, you will not know whether the uterus has ruptured, nor whether it will rupture in the next 30mins: so perform *all* vaginal procedures for the relief of neglected obstructed labour in the operating theatre, with equipment and staff available instantly for a laparotomy.

Caesarean section has a limited role in the management of obstructed labour, *especially if neglected*, and is likely to be a serious risk, *so don't undertake it lightly*. It is mainly indicated when the foetus is alive and the mother is in reasonable condition. However, make sure that the foetal heartbeat is still present just before you start the incision!

A destructive operation (21.8) is indicated when the foetus is dead, the cervix is fully dilated or nearly so, the presenting part is fixed in the pelvis, and the uterus has not ruptured, and is in no danger of doing so. If you are in any doubt, perform a mini-laparotomy as above. Occasionally, when a destructive operation for foetal death would be difficult, because the foetal head is mobile and ≥3/5 above the pelvic brim, you might have to resort to a hysterotomy (21.5).

(a) Symptoms

A woman in obstructed labour is in great pain, anxiety, and distress. In the bustle of treating her, *don't forget to comfort and reassure her*.

If the foetus is already dead, inform her. *If you don't, she may blame you for its death*, and not come to hospital when she is pregnant next time.

(b) Diagnosis

Suspect obstructed labour or neglected obstructed labour if you find:

(1) No cervical dilation despite what appear to be good contractions.
(2) Increasing moulding (21.5) and caput (21.3), but without descent of the foetal head.
(3) Anxiety and restlessness.
(4) Hypertonic uterine contractions, with poor relaxation in between.
(5) A stretched lower segment with a Bandl's ring.
(6) Bloody urine.
(7) Unexpectedly easy dislodgement of the presenting part followed by a gush of vaginal bleeding: then abandon vaginal delivery and open the abdomen.
(8) Variable or poor application of the cervix to the head.
(9) Offensive discharge or fever.
(10) A vesico- or recto-vaginal fistula (usually found 2-3 days after delivery).

N.B. Now determine if the uterus is ruptured.

If you are in doubt about uterine rupture, make a mini-laparotomy to check the uterus: if it is intact, proceed to a vaginal delivery while the incision is covered with sterile towels, then recheck the uterus and perhaps, when indicated and desired, tie the tubes.

This is far less risky as an operation than a Caesarean section, because the contaminated fluids in the uterus don't enter the peritoneal cavity and don't soil the uterine and abdominal wall incisions.

Otherwise, with ultrasound guidance, you can try aspiration with a long needle lateral to the uterus to reveal meconium-stained fluid (more common than bright red blood). This confirms the diagnosis of rupture.

N.B. Ultrasound, on its own, is unreliable unless the rupture is obvious.

(c) Differential diagnosis

There is, in the main, a prolonged latent phase. If the patient was made to push during the latent phase, she may be distressed and dehydrated, and the vulva and cervix may be oedematous.

The cervix will however not be dilated, or only slightly so, the membranes are likely to be intact, and there will be no Bandl's ring. Reassurance, observation, analgesics, sedatives and IV fluid replacement may be all that is needed.

(d) Emergency care
Hypovolaemic and/or septic shock are very common. Resuscitation must be rapid, because delivery is urgent. Admit the patient directly to whatever high-risk area you have, usually the labour ward or the theatre, and resuscitate her there. This will allow you to operate as soon as she is in an optimal condition.
Correct the dehydration, the electrolyte deficit, and the acidosis with warmed Ringer's lactate; there is usually no need to use bicarbonate. She may rarely need blood, preferably red cells only. *If the haematocrit is raised as a result of dehydration, a transfusion, even of safe blood, may be harmful:* her primary need is IV fluids.
Administer IV chloramphenicol, benzylpenicillin and rectal metronidazole. If, in spite of adequate resuscitation, shock persists, try a titrated infusion of dopamine. This will cause peripheral dilation, so correct it immediately with more IV fluids. *Don't however waste time with sophisticated methods if your team is not familiar with them;* proceed simultaneously with delivery.

(e) Monitoring. Record the pulse and blood pressure every 5mins. Monitor the urine output hourly.
If the presenting part stops descending, the cervix usually stops dilating, although this may not be so in multipara. On the partograph, the progress line will have crossed the action line.

(f) Management
(1) Assess the height of the foetal head. *Don't assess this by vaginal examination only*. There will be much caput (21.3), and this will mislead you. It's the descent of the skull that matters, not the descent of the foetal scalp swelling!
(2) Calculate the foetal moulding score:

0	**Bones still separate**
1	**Bones touching**
2	**Bones overlapping, but separate when you press with a finger**
3	**Bones overlapping but not separable**

Feel where the foetal parietal and occipital bones touch one another. Overlapping at both the sagittal and the lambdoid (between the parietal and the occipital bones) sutures is more serious than at the lambdoid suture alone. An easy way to document moulding is to add the moulding between the parietal bones (PP) and the moulding between the occipital and one parietal bone (OP). So if PP 2+ and OP+, total moulding is 3+.
(3) Watch for foetal distress. Count the foetal heart rate for 30secs, before, during and after a contraction. Foetal distress is shown by a rate of <120 or >160/min or slowing which persists *after* a contraction (slowing during it is normal).
CAUTION!
(1) *Don't use an oxytocin infusion if there are signs of obstruction.* On the correct indications (see later), you can use it for delay.
(2) *Don't use Kielland's forceps, or try internal version.*
(3) *Never attempt an operative vaginal delivery if the uterus has already ruptured*.
N.B. You may not know if the uterus has ruptured, so do all vaginal procedures for the relief of obstructed labour in the theatre, with a set of laparotomy instruments ready for instant use. Alternatively, perform a mini-laparotomy.
(4) Choose an appropriate method of delivery:

(g) Episiotomy. This is sometimes all that a primipara needs, especially if the foetal vertex is in an occipito-posterior position. Putting her into the lithotomy position may make delivery easier. *Beware of 'routine' use: the cut may become bigger, be painful, get infected and cause excessive blood loss.*

(h) Vacuum extraction (21.6)
INDICATIONS
(1) A live foetus with $<^2/_5$ of the foetal head above the brim: the more moulding there is, the lower the head should be for a successful vacuum extraction.
(2) An occipito-transverse or occipito-posterior position, without CPD, or with only mild CPD.
(3) Definite CPD combined with a symphysiotomy (21.7).
(4) For an unconscious patient, *e.g.* with eclampsia, perhaps combined with fundal pressure to make up for the lack of straining.

CONTRAINDICATIONS
(1) A live baby with $>^2/_5$ of the foetal head above the brim.
(2) Severe moulding.
(3) Definite CPD *unless combined with a symphysiotomy*.
CAUTION!
(1) Delivery with a vacuum extractor or outlet forceps should never be a difficult operation. If foetal asphyxia is already present, it should merely be a 'lift-out'.
(2) Use a systematic technique of pulling.
Don't be afraid to use an old machine which works even if you don't have a modern Kiwi device!

If it has taken >3h for the cervix to dilate from 7-10cm on the partograph, or the fundal height is >40cm, suggesting a large baby, perform the vacuum extraction in the operating theatre, and prepare for symphysiotomy or Caesarean section.

(i) Fundal pressure may save the day in such situations as: foetal distress, or an exhausted mother where her straining is just not forceful enough to deliver, and where a little bit of extra force will deliver the baby. Or else, even without foetal distress, when there is no working vacuum extractor present and transport to a suitable venue with better facilities will take too much time, or there is the risk of delivery in an unsuitable vehicle (or oxcart) during transport. Even in a sophisticated hospital, when a vacuum extraction is almost successful but that little extra force needed pulls off the cup, fundal pressure may solve the problem.

(j) Symphysiotomy (21.7)
INDICATIONS
A live foetus, of fundal height <40cm (2·5-4kg), in a cephalic presentation, with these scores:

Head above the brim	**Total foetal moulding score** (PP+OP)
$\leq^2/_5$	<6 if the head is on the pelvic floor
$\leq^3/_5$	≤3

(k) Destructive operations (21.8)
INDICATIONS. Make sure that *all* these conditions are met:
(1) Foetal death or severe foetal malformation incompatible with life.
(2) An impacted foetal head with $\leq^{2}/_{5}$ above the pelvic brim, or a transverse lie.
(3) Cervical dilation at least 7cm, and preferably full.
(4) *No uterine rupture, nor imminent danger thereof.*

CAUTION! A multipara who has been in labour for a long time will have a lower segment which will be very thin. If it is also tender and distended, it is certainly *very* thin and on the point of rupture.
Don't push an instrument against the foetal head without somebody supporting the fundus; otherwise your pushing might rupture the uterus if the head dislodges upwards.

N.B. When the lower segment is paper-thin, any destructive operation will rupture the uterus, unless you are simply decompressing a hydrocephalic head with a needle.

(l) Caesarean section (21.10)
INDICATIONS
(1) A live foetus whose head is too high for vacuum extraction or symphysiotomy.
(2) A dead foetus $<^{2}/_{5}$ above the pelvic brim, whose head cannot be pushed down into the pelvis to perform a destructive operation safely.

CONTRAINDICATIONS
(1) A head which is deeply engaged in the pelvis ($\leq^{2}/_{5}$ above the brim).
(2) A dead foetus which can be delivered by a destructive operation.

Here we are mostly concerned with a vertex presentation and a few curiosities. See elsewhere for a breech presentation (22.7), a transverse lie, and a brow or a face presentation (22.8).

VERTEX CEPHALIC PRESENTATION

If uterine rupture is suspected but uncertain, perform a mini-laparotomy for inspection (see above).
If the foetus is alive and the cervix is not fully dilated, perform a Caesarean section.
If the foetus is alive and the cervix is fully dilated, management depends on:
(1) the height of the foetal head,
(2) the degree of moulding,
(3) signs of foetal distress,
(4) the risks associated with needing further Caesarean sections,
(5) the estimated size of the foetus.

If the foetal head is:

$^{0}/_{5}$ above the brim, with minimal moulding, apply the vacuum extractor and see, in the course of delivery, whether an episiotomy is needed or not.

$^{1}/_{5}$ above the brim, with a total moulding score (PP+OP) 0-1 and foetal distress, perform a trial of vacuum extraction in the operating theatre, with everything ready for symphysiotomy.

$^{1}/_{5}$ above the brim, with a total moulding score 2-3 and foetal distress, combine vacuum extraction with a symphysiotomy, or perform a Caesarean section.

$^{2}/_{5}$ above the brim, with a total moulding score of 0-2, maybe 3, perform a trial of vacuum extraction in the operating theatre, with everything ready for symphysiotomy or Caesarean section.

$^{2}/_{5}$ above the brim, with a total moulding score of 4 or possibly 5 and foetal distress, perform a trial of vacuum extraction in the operating theatre, with everything ready for symphysiotomy or Caesarean section.

$^{3}/_{5}$ above the brim, with a total moulding score of 0-2, perform a trial of vacuum extraction. If necessary and if the pelvis is big enough (you can get your finger between the head and the symphysis), perform a symphysiotomy.

$^{3}/_{5}$ above the brim, with a total moulding score of >2, perform a Caesarean section, unless you can get a finger between the head and the pelvic wall, indicating that a symphysiotomy might be possible.

If the foetus is dead, **with an impacted head $\leq^{2}/_{5}$ above the pelvic brim and the cervix is ≥7cm dilated**, perform a destructive craniotomy, provided you can get a finger between the foetal head and the pelvis.

If the foetus is dead and its head is mobile or $\geq^{3}/_{5}$ above the brim, get an assistant to push the head into the pelvis as you perform a destructive craniotomy. If this fails, and the foetus is mobile enough in the uterus, see if you can insert a balloon catheter into the foetal rectum and apply traction for a breech delivery (22.7). Only as a last resort, perform a hysterotomy.

MENTO-POSTERIOR PRESENTATION

If the foetus is alive and the cervix is fully dilated, perform a Caesarean section.

If the foetus is dead and the cervix is fully dilated, perform a destructive craniotomy.

(m) A congenital vaginal septum (rare) seldom causes trouble, because it is usually quite thin, pushes to one side, and may never even be diagnosed during labour. If it does cause trouble, but is thin, you may be able to divide it. If it is thick, perform a Caesarean section, and excise it later making sure the patient is not pregnant.

(n) A vaginal stricture (quite common) caused by scar tissue from a previous delivery, or of uncertain cause, feels quite different from a cervix. If it is thin, incise it latero-posteriorly on both sides (at the 4 and 8o'clock positions), and let vaginal delivery proceed, and afterwards suture the incisions if they bleed significantly. If it is wide and fibrous, perform a Caesarean section.

(o) An ovarian tumour or a fibroid obstructing labour (21-6). Perform a Caesarean section. If there is an ovarian cyst or tumour, you can remove it at the same time as the Caesarean section. If there is a fibroid, leave it unless it has a thin pedicle, and remove it subsequently if necessary.

N.B. Never try to remove a non-pedunculated fibroid at Caesarean section, as it will bleed copiously.

(p) Post-operatively after a difficult vaginal delivery
An over-stretched infected uterus will often not contract properly after delivery. So there is an indication for active management of placental delivery (the 3rd stage), as well as rubbing the uterus and the continuous use of IV oxytocin after the placenta is delivered.

Keep the patient in hospital for 3-4 days (7-10 days for a symphysiotomy). Observe her carefully. Before she goes home, make sure that she understands what operation she has had, and why it was done. This will be important when she becomes pregnant again.

(q) Obstetric paralysis
If there is a history of neglected obstructed labour, examine her early in the puerperium for signs of peripheral nerve injury. She may fail to complain about sensory changes and weakness, so you will have to look for them. They are present in obstetric paralysis, and may vary from mild footdrop to extensive paralysis of her legs, including her gluteal and quadriceps muscles. If you are not careful, she may develop contractures. So put her joints through their full range of passive movements regularly, and encourage her relatives to do the same. If she has a foot drop, use a posterior plaster splint to keep her ankle fully dorsiflexed at night (32.11). During the day, typical high basketball shoes make walking much easier. She is almost certain to recover, but this may take 2yrs.

If despite infusion of large amounts of fluids, only <400ml urine is passed in 24h, she is in renal failure. Put her on an accurate fluid balance. This is serious, but potentially curable. Early treatment will improve her prognosis, so watch for it. Try furosemide 40-320mg IV. If this fails, try dopamine if obtainable. Start with 2-5μg/kg/min. Increase in steps till an effect has been attained; this is usually at <20μg/kg/min.

If labour was obstructed with cephalic presentation for a long time (active labour for ≥6h), insert a catheter for 14 days: it might just prevent a fistula. If one does develop, keep the catheter *in situ* for at least 6wks.

N.B. The neonate has a greater chance of brain damage in a difficult delivery. This may be caused by:
(1) Direct trauma from the procedure itself.
(2) Lack of oxygen.
(3) Poor foetal blood flow.

Watch carefully for signs of twitching, irritability, or fever.

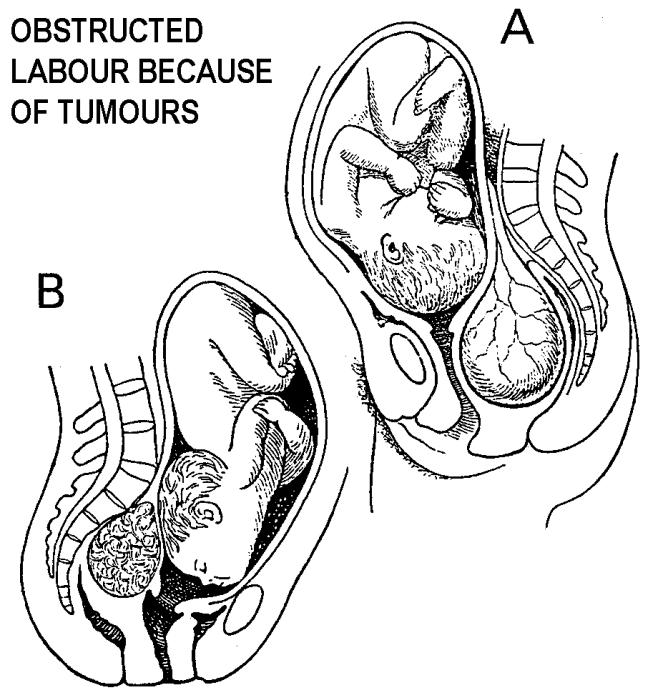


Fig. 21-6 TWO TUMOURS OBSTRUCTING LABOUR.
A, an ovarian cyst. B, a cervical fibroid. If a patient has an ovarian cyst or tumour, you can remove it at Caesarean section. If she has a fibroid, leave it unless it has a thin pedicle and remove it later if necessary.
After Young J. A Textbook of Gynaecology A&C Black 5th ed 1939 Figs 125,168.

(r) Oxytocin
Oxytocin is an invaluable drug for making the uterus contract:
(1) To induce labour.
(2) To accelerate labour.
(3) To stop bleeding after abortion or delivery.
Always have a secret cache of this drug so that you never run out.

The main dangers are that:
(1) *The uterus may rupture if you administer too much too fast to a multipara late in labour*. The sensitivity of the uterus to oxytocin varies greatly. Early in pregnancy it is comparatively insensitive; it becomes much more sensitive later, especially in a multipara. So in a pregnant patient always use an IV infusion, starting with a small dose. If you don't get the effect you want, use more in incremental doses. After delivery, or during an abortion, this rule does not apply, and you can safely use a bolus injection IV or IM.

(2) *The supply of oxygen and nutrients to the foetus* via *the placenta will be inadequate if uterine contractions take too long or are too frequent*. The foetus might not cope, even if there is a normal placenta. If, however, there is already marginal placental function, *e.g.* in the presence of high blood pressure and/or growth retardation, the effect on the foetus may be catastrophic.
Remember: *oxytocin might kill a foetus if you don't monitor its use properly. Even normal contractions may be too much for the foetus if placental function is very poor!*

(3) *You can add too much fluid* at the same time when infusing oxytocin IV, especially when you use oxytocin to induce labour early in pregnancy, when you may need high doses. So, when you use escalating doses, avoid the danger of water intoxication by using 0.9% saline or Ringer's lactate, *not 5% dextrose*.
The primigravid uterus is sufficiently insensitive for oxytocin to be safe enough for midwives to give routinely to accelerate labour. *But, using oxytocin to accelerate labour in multipara can be dangerous*, so only use it if you are experienced, and *don't let your midwifery team use it unsupervised.*
In Africa, the head is often high through much of the 1st stage. Speeding its descent with oxytocin is dangerous for the inexperienced. If labour in a multipara is slow, and her previous deliveries were normal, she will probably deliver the present foetus eventually, provided he has a cephalic presentation. So it is likely to be safer to leave her, after examining carefully to exclude a brow presentation, than risk rupturing the uterus by using oxytocin unnecessarily.
Oxytocin is used otherwise in many situations: evacuation of incomplete or delayed miscarriage (20.2), and retained miscarriage (20.4), inducing labour (22.2), breech presentation (22.7), multiple pregnancy (22.10), and post-partum haemorrhage (22.11).
Always use the 'protocol on the wall'

AVOID OXYTOCIN IN A MULTIPARA

21.6 Vacuum extraction (Ventouse) (GRADE 1.3)

You will find a vacuum extractor invaluable, so if you are not already using one, you must! A properly functioning one will save many mothers and babies. It has many advantages in the confined space of the commonly small pelvis of many women seen in many low-income communities.
Unlike forceps, the vacuum cup takes up no extra space beside the head in the birth canal, and it is difficult to injure the mother seriously. The foetal head can rotate spontaneously at the optimum level, and if it is not flexed enough, vacuum extraction will often flex it. *Most importantly, a vacuum extractor is less likely to damage the foetal brain than forceps.* The indications for its use in a hospital are somewhat broader than those in a health centre.

N.B. Make it a habit to clean and grease your (manual) vacuum apparatus weekly. Machines which don't function at the critical moment might kill babies and even mothers.
If somebody offers donations to your hospital, ask for extra cups (preferably the model Bird type, including one posterior cup) and tubing so that you have a few autoclaved packs ready, particularly over a long weekend. Invest in the best quality: *there are very poor-quality sets on the market*. Before you start applying the cup to the head, try it on your gloved hand first to see if there are any leaks.

INDICATIONS
(1) Delay in the 2nd stage of >1h in a primigravida, and 30mins in a multigravida, especially delay caused by malrotation of the occiput.
(2) To reduce maternal effort if a woman has cardiac failure, gestational hypertension, or exhaustion.
(3) To minimize the strain on a scarred uterus.
(4) Relative CPD due to deflexion and malrotation of the head.

N.B. If there is absolute CPD, don't use a vacuum extractor; it will be ineffective and potentially dangerous unless you combine it with a symphysiotomy (21.7).
(5) A need for haste because of foetal distress without or with only mild CPD.
(6) In an unconscious patient, *e.g.* with eclampsia, with near (8cm) or full cervical dilation and a moderate-sized foetus.
(7) Failure to progress, or exhaustion in a 2nd twin with a cephalic presentation, when the cervix is closing down even if the membranes have been ruptured and an oxytocin infusion is in progress. The height of the head does not matter in this situation, provided you can get the cup on the occiput. (*This is not easy because the head is often high and the flabby cervix and the cord of the 1st foetus tend to find their way into the cup.*)
(8) Prolapse of the cord in a multipara.
(9) In combination with a symphysiotomy in order to control the delivery better and keep the head away from the temporarily poorly supported urethra.

CONTRAINDICATIONS
(1) Prematurity <36wks, because of the risk of intracerebral haemorrhage.
(2) A brow or face presentation.
(3) CPD, unless you combine vacuum extraction with symphysiotomy (21.7).
(4) Foetal death.

CAUTION! Don't apply a vacuum extractor before full or nearly full (8cm) cervical dilation because it is usually dangerous: the only exceptions are (5), (6) and (7) above. *Don't use it for delay late in the 1st stage* (21.3). (If this does not respond to oxytocin, it is likely to be due to CPD.)

If (a) it has taken >3h to dilate from 7-10cm on the partograph,
or **(b) the fundal height is >40cm** (suggesting a large baby), expect difficulty. Perform the vacuum extraction in theatre, and prepare for Caesarean section.

REQUIREMENTS
(1) A cephalic presentation.
(2) The foetal head must be $\leq 1/5$ above the pelvic brim.

N.B. Always determine the position of the head in relation to the pelvic brim, and *not to the ischial spines*; if the pelvis is shallow and there is much caput, you may be able to feel it below the spines before it is engaged.
(3) The head must descend, or at least move somewhat, with contractions and bearing-down efforts.
(4) You should preferably know where the occiput is, because traction will be more effective if you can put the cup there. This is often not that easy and sometimes you are forced to put the cup at the lowest point. An ultrasound might help, if you are not sure, to locate the back of the occiput (21-7).

If a bimanual examination indicates that the head is wedged solidly in the pelvis, and you are unable to rock it up and down at all, then a vacuum alone is unlikely to be successful. It there is severe moulding >4, you will need to add a symphysiotomy at least. However, seeing the foetal head in the *introitus* especially between contractions is a good reason for optimism.

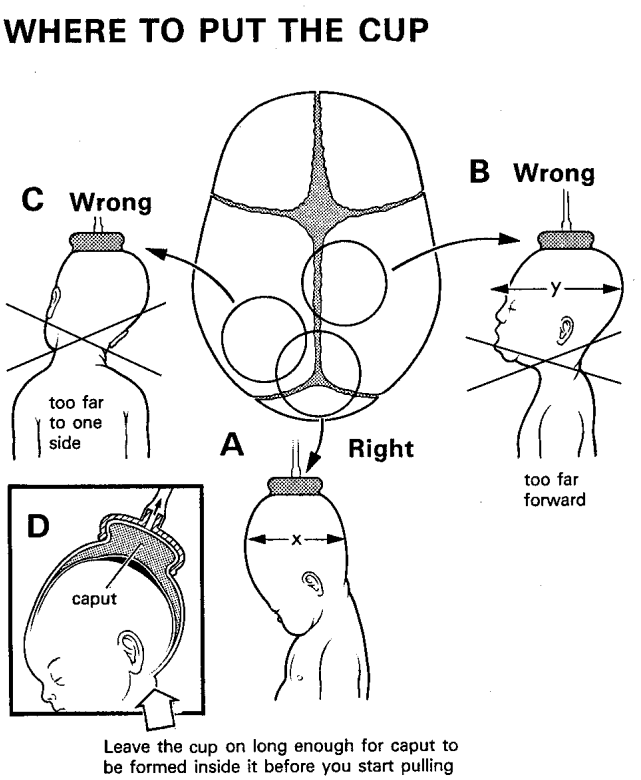


Fig. 21-7 WHERE TO PUT THE VACUUM EXTRACTOR CUP. You will find a vacuum extractor invaluable. Attach the cup as nearly as you can over the posterior fontanelle or just a little in front of it.

METHOD

Cooperation from a woman who is fully conscious is desirable, but not essential. Good uterine contractions, which mean 3-4 every 10mins lasting >40secs, are the norm, but not essential (as in the case of an unconscious patient). With an uncooperative patient, use ketamine.

N.B. An oxytocin infusion in the absence of good contractions may cause foetal distress.

Use 3 pulls, the first to dislodge the foetal head from its arrested position, the second to bring the head to the pelvic floor, and the third to deliver the foetus, or at least make the vertex visible at the *introitus*. Pull during contractions combined with maximum straining and keep traction maintained in between to prevent the head from retreating between contractions/pushing efforts.

N.B. There are situations where you use vacuum extraction to prevent strenuous pushing, e.g. when there is a scar on the uterus, maternal cardiac abnormality, hypertension or fear of maternal cerebral haemorrhage.

Sometimes you know that pulling a little harder could deliver the baby, but you also know that the cup will then come off. If you think that is the case, it is quite legitimate to get an assistant to push on the fundus. Contraindications for this fundal pressure are a scar in the uterus and perhaps an extremely enlarged spleen.

It might be advisable to insert the local anaesthetic for a symphysiotomy (21.7) before the vacuum extraction, in case it fails.

21.7 Symphysiotomy (Pelvic release)

Cutting the symphysis allows the two halves of the pelvis to separate 2-3cm. This increases its diameter by 0·6-0·8cm, which is enough to overcome mild or moderate CPD, and so avoid Caesarean section. After delivery, its circumference remains wider by about 1·5cm, and its diameter by about 0·5cm, so that the next deliveries may well be normal. Symphysiotomy is thus particularly valuable if the mother expects a large family. Moreover, having a scar on the uterus is hazardous if the next delivery is not guaranteed to happen in a well-equipped and -staffed hospital.

This is an invaluable operation which needs to be reinstated and given its proper place in obstetric practice in poorly resourced centres. The reasons are clear:

(1) Unlike Caesarean section, especially with unskilled anaesthesia, it is never fatal, and seldom produces complications, particularly serious ones.

(2) It does not leave a woman with a scar in the uterus which may rupture if she does not deliver in hospital when she is pregnant the next time.

(3) It may save her life if she delivers in a health centre and cannot be speedily referred.

(4) Retrospective studies show quite clearly that symphysiotomies are less dangerous for the women involved and very probably the baby also.

We encourage you to investigate this, since, like the destructive operations, it is one of the few practical procedures which might really alleviate maternal mortality from obstructed labour.

Symphysiotomy has fallen into disrepute in rich countries of the world, where CPD is uncommon, where trends are set and where most textbooks are written, because gynaecologists stopped performing the operation. There it is rarely used and then mainly for shoulder dystocia. But, in countries where CPD is common, symphysiotomy is excellent when used properly (as is the case for any procedure) for obstructed labour.

There is no doubt that, if CPD is marked, a woman needs a Caesarean section. The skill you need is to recognize when this is mandatory. You will not need to make a symphysiotomy very often, but there are hospitals in Nigeria where it is performed more often than a Caesarean section and the patients prefer a symphysiotomy if given a choice. You will find that deciding when to perform one needs more judgement than deciding when to perform a Caesarean section. If a symphysiotomy fails, you can still perform a Caesarean section: but you should look upon this as an error of judgement, and try to do better next time.

In many countries there is resistance from the medical/gynaecological/midwifery/political establishment to symphysiotomies. If you have to prove your point, it is therefore better to start with a very solid indication, *viz.* a woman who is feverish, has been in obstructed labour for hours, and has a live foetus whose head is low. In these circumstances, a Caesarean section is very dangerous and might be impossible to organize within the 30mins before the foetus dies.

On the other hand, with the stuck aftercoming head in a breech presentation, a shoulder dystocia or a failed vacuum extraction, it will take only 15mins to execute your first symphysiotomy slowly and methodically. If all goes well, that might be a watershed for introducing the value of this procedure.
If you have sufficient helpers, ask your staff to prepare for a Caesarean section, but get on with the symphysiotomy in the meantime!

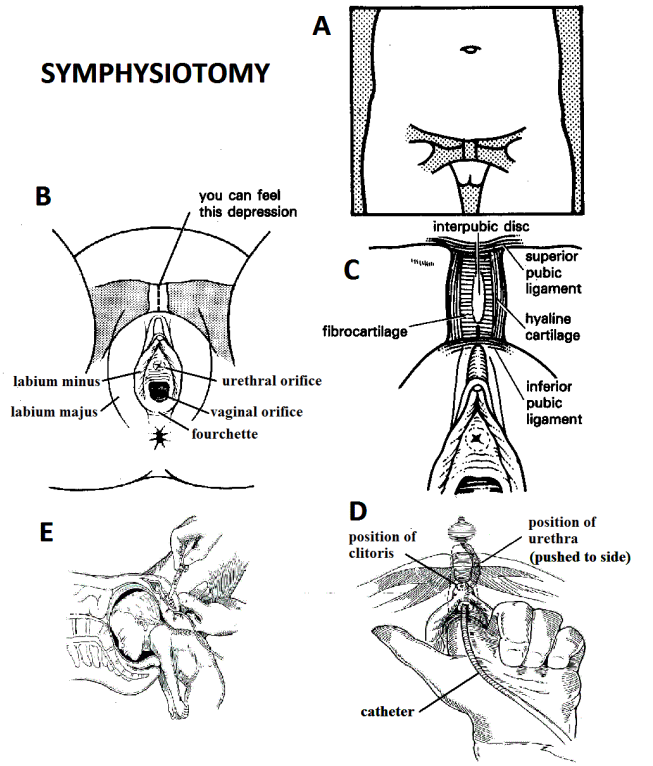


Fig. 21-8 SYMPHYSIOTOMY. A, position of the symphysis on the anterior abdominal wall. B, position of the symphysis in relation to the clitoris. C, structures to cut. D, incision in relation to the urethra pushed laterally, and the bladder. E, situation in shoulder dystocia.
After Pust RE, Hirschler RA, Lennox CE. Emergency Symphysiotomy for trapped head, Tropical Doctor 1992;22(2):71-75.

SYMPHYSIOTOMY (GRADE 2.1)

INDICATIONS

Mild or moderate CPD associated with any of these problems, most of which are interrelated:
(1) A failed trial of vacuum extraction even after applying fundal pressure. This is the most common indication. It is difficult to be sure that vacuum extraction won't work without having a try! A difficult vacuum extraction may succeed, but only after prolonged traction and the risk of damaging the baby. Symphysiotomy will make delivery easier and safer. At the strategic moment, preferably before there are any signs of foetal distress, it is ideal. If the indications are right, it is better than Caesarean section, and it avoids a difficult vaginal delivery.
(2) Neglected obstructed labour with a live foetus. If the foetal head is deeply jammed into the pelvis, perhaps with caput visible at the vulva, symphysiotomy will be safer. If you try to perform a Caesarean section, the foetal head will be difficult to deliver, and cause a laceration of the lower segment into the large vessels or deep in the cervix or even into the bladder.
Morover, there will often be an infection at this stage and by opening the uterus, infected material can contaminate the operative field including the peritoneal cavity. The wound in the uterus and the abdominal wall is likely to become infected. An infected uterine incision is likely to result in a weak scar.
(3) A woman living in an isolated location, who comes late to hospital, and who is likely again to present late with the next delivery, and especially whose foetal outcome is likely to be poor. She may subsequently be tempted to deliver at home with fatal consequences, about which you will hear nothing. If she has a symphysiotomy, her pelvis will be somewhat larger and there will be no uterine scar.
(4) Mild or moderate CPD with a live foetus, particularly in a primigravida, when the foetal head is $^{1}/_{5}$ or $^{2}/_{5}$ above the brim, and is too tightly held for vacuum or low forceps alone.
(5) To deliver the arrested aftercoming head of a breech presentation (if you are quick!) or a shoulder dystocia.
N.B. Never do this as your 1st ever symphysiotomy.
(6) Extreme anaemia when there is no blood available or the patient is a Jehovah's Witness who refuses transfusion.
(7) When there is a long lag time between taking the decision to perform a Caesarean section and actually doing it, especially if this delay might kill mother and/or child. Moreover, the longer the waiting list is for Caesarean sections, the more likely it is that somebody else will die who is even later in the queue.
(8) In a health centre, where a symphysiotomy is an emergency delivery method, thus securing a live baby, when referral is impossible.

CONTRAINDICATIONS

(1) Severe CPD.
(2) Malpresentations, with the exception of the aftercoming head of a breech (22.7).
(3) Foetal death; if there is minor CPD and a symphysiotomy would be feasible, a craniotomy will likewise be possible; if there is no CPD, a symphysiotomy is unnecessary. Only in very exceptional circumstances would a symphysiotomy be an option with CPD and foetal death: when a woman is highly likely to have CPD in the next pregnancy and she is very unlikely then to deliver in hospital next time. A larger pelvis after the symphysiotomy for a dead baby could help with the next delivery.
(4) A complete family when the patient would like a sterilization. The chances are that there will be fewer combined obstetrical problems now and in the future if a tubal ligation is performed together with a Caesarean section.
(5) Abnormalities of the maternal pelvis or legs.
(6) A large foetus >4kg as estimated by the fundal height being >40cm (who is too big to deliver by symphysiotomy), or <2·5kg (who does not need one).
(7) A foetal head which remains $>^{3}/_{5}$ above the pelvic brim after rupture of the membranes, *e.g.* in hydrocephalus (22.6).

Relative contraindications are:
(8) Severe obesity,
(9) Previous symphysiotomy, or
(10) Previous VVF repair.

If you have the opportunity, see what happens to a cohort of women who delivered by Caesarean section and who could have had a symphysiotomy instead. Record the complications, not only those around the index delivery

but also from subsequent deliveries, say over the next 10yrs. Such a study in your region will probably show that many women who had a Caesarean section, but could have had a symphysiotomy for failed vacuum extraction, or for failure to progress or foetal distress in the 2nd stage, die of complications of a subsequent delivery or become infertile because of post-operative peritonitis. There will then be evidence to challenge inappropriate use of Caesarean section in your region.

METHOD

Check the cervical dilation, the descent and position of the foetal head. At this point, decide if symphysiotomy is indicated or not. (Symphysiotomy is normally done at full dilation, but you can do it when there is still a 1 or 2cm ring of cervix.)

If the foetal head is $^1/_5$ above the pelvic brim, a symphysiotomy is unnecessary, unless combined with a failed vacuum extraction.

If the foetal head is $\geq^2/_5$ above the pelvic brim, symphysiotomy may be indicated.

If the foetal head is $\geq^3/_5$ above the pelvic brim, try to insert a finger vaginally between the foetal head and the pelvis. *If your finger passes too easily, symphysiotomy is unnecessary*. If it passes with difficulty, symphysiotomy is indicated. If it does not pass at all, CPD is too great, so proceed to Caesarean section.

Listen to the foetal heart to make sure that the foetus is alive. Place the patient in the lithotomy position.

CAUTION! Find 2 assistants and ask them to support each of the legs, so that the abduction angle between the two upper legs is at maximum 90° (each upper leg 45º angle with the horizontal). This must be their only job; they must do nothing else. If they allow the legs to flop apart, the fibres of the sacroiliac joint may rupture, and the patient will have much post-operative pain, and maybe pelvic instability. You will value these assistants anyway, even if you have reliable lithotomy poles, to prevent too much abduction.

Palpate the bony margins of the symphysis pubis. Infiltrate the skin and subcutaneous tissue over the symphysis and the 'joint' between the pubic bones with 12-15ml of 1% lignocaine with adrenaline. Infiltrate also the area for an episiotomy with the remaining LA in your syringe. Although normally a vacuum extraction does not automatically mean an episiotomy, it does with a symphysiotomy. By controlling the descent of the head and keeping it away from the urethra, you will prevent a tear.

Clean the skin and vulva well with betadine or chlorhexidine. Insert a stiff urinary catheter. You may need to push the foetal head a little to pass the catheter. Apply the vacuum extractor. Place the index finger (*don't use two fingers, because the urethra tends to slip between them*) of your non-dominant hand in the vagina, to displace the catheter in the urethra away from the midline, and place behind the cartilaginous groove between the pelvic bones, *so you can feel the scalpel move!*

CAUTION! You *must* displace the urethra, or you may cut it. This could be a major disaster!

Use a large blade, preferably a solid knife, so that the blade does not come off or break. Make a 2-3cm incision in the skin and subcutaneous tissue 1cm below the upper edge of the symphysis pubis exactly in the midline. Then hold the knife vertical, and find the exact position of the 'joint'. (This you should have located when you injected LA.)

Push the point of the still vertically held blade about 1cm deep in the space between the pubic bones (in the mid-point between the superior and inferior margins) with the cutting edge of the knife facing downwards. In order to avoid damaging the vagina or urethra (which you are pushing to the side with the index finger of your non-dominant hand), stop cutting downwards, but swing the blade towards you, so that you are now cutting upwards (away from the vagina). The fibres of the upper part of the 'joint' will then act as a fulcrum for the blunt part of the end of the blade. The sharp part of the blade will cut the fibres of the lower part of the 'joint'; you will feel them giving way as they part under tension.

N.B. If you feel the scalpel against bone, re-position it!

The skin is usually so flexible in this area that the skin incision will only be enlarged a little because of this manoeuvre. Once you have cut the fibres of the lower part of the 'joint', *taking care never to go beyond the arcuate ligament* (which arches across the inferior aspect of the symphysis), withdraw the knife, hold it again vertical and rotate it in such a way that the belly of the blade is now facing upwards. Re-insert the knife through the stab wound and now swing the blade away from you, thereby cutting the fibres near the superior margin of the 'joint'. When the 'joint' is almost divided, it will begin to open, sometimes audibly: 2cm is ideal, it should never open >4cm. Often, though, you will not have cut enough fibres and will have to divide some more. The best way to do this, keeping your non-dominant finger in the vagina pushing the urethra away from the midline, is to insert the blade again under the skin in the 'joint' but keeping it nearly horizontal, and push the belly of the blade (*not the point though*) against the tense fibres. *Don't make a sawing motion*, but you can move the handle up and down somewhat in a 20º rocking action against pressure of your finger in the vagina. Gentle pressure usually suffices.

If you cut 90% of the fibres, that is enough; *don't be persuaded to cut more in the superior part of the 'joint'* because it is more difficult to dislodge the proximal urethra and bladder base in this area. Alternatively, when nearly through the fibres, use the top of your thumb through the wound as a wedge to force open the 'joint' so that the knife stays far away from the vagina. (This works well, although your thumb will be somewhat tender for a few days!)

N.B. Sometimes the 'joint' is not completely vertical and you have to hold the blade aiming somewhat out of the midline (therefore not completely vertically down). *Be careful not to exert too much rotating force on the handle (clockwise or anticlockwise)* because you could wrench the blade out of the handle or break it. However, it is usually easy to retrieve a large blade.

N.B. Often the 'joint' opens suddenly and the head comes down very fast and it is a struggle to apply the vacuum and perform an episiotomy. *So it is better to apply the vacuum before the symphysiotomy. You may well need to perform an episiotomy as well.*

You will find anyway that most of your symphysiotomies will be done after a failed vacuum. It is very difficult to be sure, where there is an indication for symphysiotomy, that an extraction will fail. It is better to perform the episiotomy as late as possible because there might be significant loss of blood with greater chance of HIV exposure.

CAUTION!
(1) *Always keep the urethra to one side with your non-dominant hand.*
(2) *Don't cut above the symphysis pubis*, because the uterus or bladder may be protruding there.
(3) *Don't cut below the symphysis pubis*, in case you damage the clitoris.
(4) *Never perform a symphysiotomy without an episiotomy*.
(5) *Make sure the legs of the patient are held firmly in position and strapped together before moving her from the delivery bed.*

If you have operated on the right indications, the mother will deliver easily, usually after bearing down with 1-2 contractions.

CAUTION! Don't apply delivery forceps after symphysiotomy; they may stretch the sacroiliac joint too much or damage the initially poorly supported urethra.

If the incision continues to bleed, suture the subcutaneous tissue and skin with 1-2 vertical mattress sutures. Lie the patient on her side, and press on the wound for 5mins.

Leave a self-retaining catheter in place for as long as the patient is immobile because lifting the buttocks on a bedpan will be painful. Normally after 2 days when she is able to sit on the side of the bed, remove the catheter, provided the urine is not blood-stained. The usual cause of this is obstructed labour or a balloon blown up still partly in the urethra because the foetal head prevented it being passed all the way in the bladder.

If the indication was for prolonged obstructed labour (>6h), the patient is at risk of developing a fistula; so leave the catheter *in situ* for 10 days to try to prevent this from developing or to help a tiny fistula close completely.

Apply a stretch bandage around the knees for 2 days. Allow walking with the help of a chair, frame or trolley as soon as the legs can move independently without pain (usually 72h). Some patients can do this easily, others, especially the heavier ones, fail to walk until the 5th-7th day. Remove sutures on the 7th day. Most patients walk well, and are fit for discharge, on the 10th day. *There is no need to bind the pelvis:* the symphysis will heal leaving the pelvis larger than it was before.

Difficulties with symphysiotomy
If spontaneous delivery does not occur, pull with the vacuum extractor. Use oxytocin with the birth of the anterior shoulder or very soon after, because an infected, exhausted uterus tends to contract poorly, so there is then a risk of PPH.

If the wound shows signs of local infection, use ampicillin or chloramphenicol, which are not passed through breast milk as are tetracycline or sulphonamides.

If there is post-operative fever, suspect urinary or puerperal infection because of prolonged labour, or both.

If urine does not pass when the catheter is removed on the 3rd day, replace it and try again on the 5th day.

If there is incontinence of urine, it may be:
(1) stress incontinence which happens often in the first weeks after operation but will usually recover spontaneously within 3 months, or
(2) evidence of a fistula. In this case, keep the catheter *in situ*: it might close or at least the hole might become smaller. However, check that there is not in fact a large fistula kept open by the catheter balloon!

If, later, the patient develops chronic pain and discharge, she has chronic pubic osteitis. This is rare, and treatment is difficult; use NSAIDs, and rarely antibiotics. It probably only occurs when the incision involves bone, so keep strictly to the midline in the fibrocartilage of the joint.

If the patient has difficulty walking with an uneven gait, this may well be because her hips were not held firmly, and the sacral ligaments were stretched or disrupted. A corset is rarely required, but active physiotherapy is necessary.

If you injure the urethra, see 21.18.

21.8 Destructive operations

For an obstructed labour with a dead baby, a destructive operation is usually better than a Caesarean section. These operations are sometimes said to be old-fashioned, and to have no place in modern obstetrics. Old-fashioned and unpleasant perhaps, but they have some useful features:
(1) They need few instruments and only simple anaesthesia, so that they can be done in a health centre where a woman is first seen. If she cannot be referred, they may save her life. If referral is difficult, they avoid the risks and delays of a long journey.
(2) They leave the mother with an intact uterus, which will be less likely to rupture if she has a home delivery the next time.
(3) If there is already infection present, they are less likely than Caesarean section to spread the infection into the peritoneum.
(4) Hospital stay is shorter.

The case for destructive operations is strongest in communities where a woman is married very young. She may not be fully grown when she first becomes pregnant, so that the pelvis is small and the first labour obstructs. The pelvis will continue to enlarge until she is 19yrs, so, if she delivers vaginally with the first pregnancy, the later deliveries may be possible vaginally without the risks of scarring the uterus.

Besides their distasteful messiness, the main problems with these operations are:
(1) overlooking uterine rupture
(2) damage to the vagina from instruments or ragged foetal parts.

We insist that as long as there are fistulae resulting from neglected obstructed labour, symphysiotomy and destructive operations are *not* obsolete. If your maternity staff are not familiar with these procedures, explain their indication and benefit carefully.

There are several types of destructive procedures, each with its own indication:
(1) Craniotomy,
(2) Decapitation,
(3) Cleidotomy (cutting the clavicles),
(4) Thoraco-abdominal evisceration (or embryotomy).
A transverse lie requires decapitation, and often evisceration also, which is more difficult than craniotomy; but even so, it is often wiser than Caesarean section, which is particularly dangerous for an infected neglected transverse lie.

INTRA-UTERINE FOETAL CRANIOTOMY (GRADE 2.3)

INDICATIONS

(1) For a cephalic presentation, all the following conditions must hold:
(a) Foetal death.
(b) $\leq {}^{2}/_{5}$ ($^{3}/_{5}$ if you are experienced) of the foetal head must be above the pelvic brim. The foetal head must be impacted.
(c) ≥7cm cervical dilation, preferably full.
(d) *No uterine rupture, present or imminent*.

N.B. If a multigravida has been in labour for a long time, the lower segment will be very thin, and if it is tender and distended, it is *extremely* thin. Any destructive operation, except pushing a needle into a hydrocephalic head, will rupture the uterus.
(2) For a breech presentation (22.7) when a normal or hydrocephalic aftercoming head has 'stuck'.

PREPARATION. Always perform a destructive operation in the theatre with a laparotomy set ready for immediate use. You need this, either immediately instead of a destructive operation if you find that the indications are unsuitable, or immediately afterwards if you discover that the uterus has ruptured.
Confirm foetal death. Set up an IV infusion, take blood for cross-matching, and administer pethidine 50mg and diazepam 10mg IV with chloramphenicol 1g, benzylpenicillin 5MU.
Use the lithotomy position, and clean and drape the vulva and perineum.
Infiltrate the perineum with 0.5% or 1% lignocaine.
Catheterize the bladder. Ask your assistant to hold 1 or 2 Sims' specula in the vagina so that you can see the foetal head well.

CAUTION! Ask another assistant, standing on a footstool if necessary, to steady the foetal head by fundal pressure so that the foetus is not pushed upwards when you operate on the head.

METHOD

N.B. For a hydrocephalic head, you only need a large bore cannula to drain off the fluid for the head to collapse. (You can do this through the mother's abdominal wall if guided by ultrasound, if the presentation is not cephalic.)

Put your fingers through the cervix to rest against the foetal skull. Check that the foetal head is wedged in the pelvis, or stabilized by your assistant.

Push a closed pair of strong pointed forceps (*e.g.* Kocher's) or, better, Simpson's perforator into the skull through the cranium.

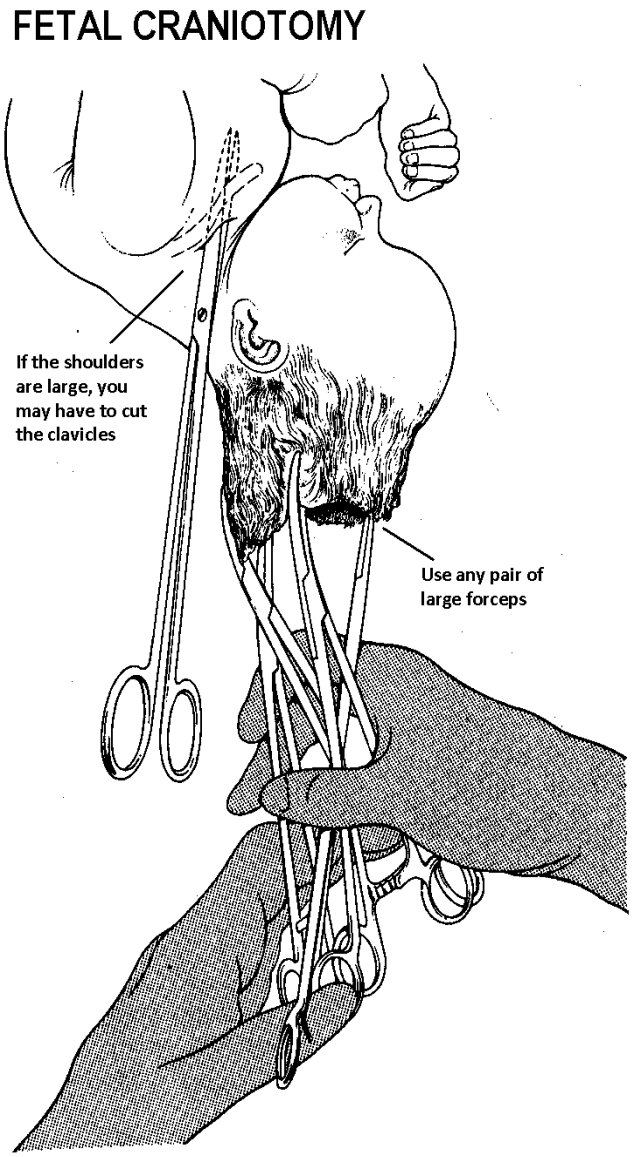


Fig. 21-9 CRANIOTOMY AND CLEIDOTOMY.
For an obstructed labour with a dead baby, a destructive operation is usually better than a Caesarean section or symphysiotomy.
Kindly contributed by John Lawson.

For a face presentation, perforate the foetal hard palate or the foetal orbit.
Ensure that the tip of the instrument is at the centre of the foetal skull. You may have to withdraw it a little to be sure of this.
Stabilize the instrument well so that it does not slip off the skull and damage the mother. When you are 5-10cm inside the skull, open the instrument and rotate it 360º to break down the septa.
Remove the instrument while still partly opened to enlarge the hole. Brain tissue will flow out from the perforation; the foetal skull will now collapse.

Remove any loose pieces of sharp bone. Attach 3 or 4 strong forceps to the foetal scalp and the remains of the foetal skull. Pull on them and try to bring the foetal posterior fontanelle under the symphysis. If sharp edges of bone stick out, protect the vagina with a Sims' speculum.

Wait until there is a contraction. Hold the 3 pairs of forceps together, and pull and twist. The collapsed foetal head should now deliver. Sometimes descent does not go very fast; instead of struggling impatiently and panicking, it is better to connect a strong traction rope to the handles of the instruments and connect that to a weight of 2-3kg over the foot end of the bed and observe for 20mins. The foetal body will follow. If a piece of the foetal skull pulls off, reattach the forceps taking a deeper bite of skull closer to its base. Make an episiotomy if indicated and deliver the remains of the foetal head.

CAUTION!
(1) *Don't include folds of the vaginal wall or cervix*.
(2) Use a good light and a large Sims' speculum, to make sure you grasp only the foetal skull.

If delivering the foetal shoulders is difficult, put a hand behind the foetus and try turning it through 90º or 180º. Then try delivering the foetal shoulders again.

If you cannot bring down the foetal shoulders by turning, bring down the foetal arms one by one. Put a hand behind the foetus in the vagina and feel for the foetal posterior arm. Gently pull it down. *Don't worry if the arm breaks, but don't damage the vagina*.

Then turn the foetus through 180º and deliver the other arm in the same way. Delivery should now be easy. Alternatively, cut the foetal clavicles (cleidotomy, 21-9).

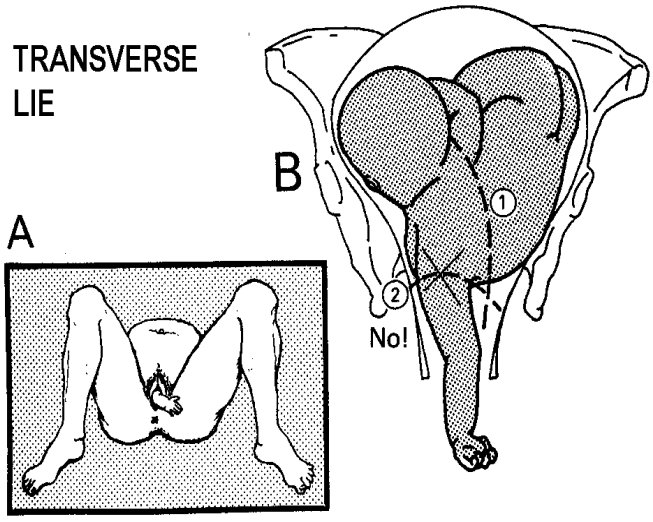


Fig. 21-10 A TRANSVERSE LIE.
A, if a community health worker meets this, advise her to refer the patient to you urgently! B, a shoulder presentation with a prolapsed arm. (1) The incision for decapitation, for a dead foetus, leaving the head attached to an arm. (2) *Don't try to remove an arm*, leave it attached to the head or the body, to help you to bring these down.
A, after Werner D. Where there is no Doctor. Hesperian Foundation 1978; B, after Howie B, High Risk Obstetrics, Macmillan, 1986 with kind permission.

INTRA-UTERINE FOETAL DECAPITATION
(GRADE 2.5)

INDICATIONS
For a transverse lie, all the following conditions must hold:
(1) Foetal death.
(2) A transverse lie.
(3) Cervical dilation ≥8cm.
(4) *No uterine rupture, present or imminent.*

PREPARATION. As for craniotomy above.

METHOD
Put one hand into the vagina and support the fundus with the other. Measure the cervical dilation. Examine the condition of the lower segment; explore it as far as you can without using force. Assess the exact position of the foetus. Which of the foetal arms have prolapsed? Where exactly are the foetal head and neck, chest, abdomen, and back? Can you reach the foetal neck easily?

CAUTION!
(1) *Don't try an internal version without doing an evisceration first*: you will rupture the uterus.
(2) *Don't attempt decapitation, or evisceration, through the vagina if the foetus is still high in the birth canal*; you will not be able to protect the vaginal wall and cervix adequately.

Bring a foetal arm down or pull on a prolapsed arm with one hand, and apply a weight connected to the arm; if you can bring the foetus down even a little, the operation will be much easier and if it is a premature foetus, this will often achieve spontaneous delivery! It also prevents the foetus being pushed upwards by your hand in the uterus, prevents the distended lower uterine segment being stretched, and brings the foetal neck lower, which you can now feel with your other hand. Feel how large it is, and how easy it is to put a finger around it. If the foetus is small and macerated, you can usually cut the foetal neck with strong scissors.

If it is larger, use the Blond-Heidler or Gigli saw (or equivalent, 21-11C). If you do have to use a saw, fix the thimble (21-11A) to it and put this on your right index finger (21-11A). Pass the thimble over the foetal neck, and down the other side (21-11B). If this is difficult, because there is little room between the foetal neck, head, and chest, try putting the saw over the foetal neck and under the arm. (21-10B: this has the advantage of making delivery of the head easier, simply by pulling on the arm.) Or improvise a smaller thimble by fixing something else, such as a piece of wire, to the end of the saw. Remove the thimble, and fix handles to each end of the saw, and cover the saw with rubber sleeves (21-11C). Keep the handles close together, so that the vagina is not injured. Protect it with the speculum. Cut the foetal neck with a few firm strokes (21-11D).

CAUTION! Hold the handles close together, otherwise you might lacerate the uterus or vagina.

To deliver the foetal body, pull on the foetal prolapsed arm, protecting the vagina from any jagged pieces of bone in the foetal neck.

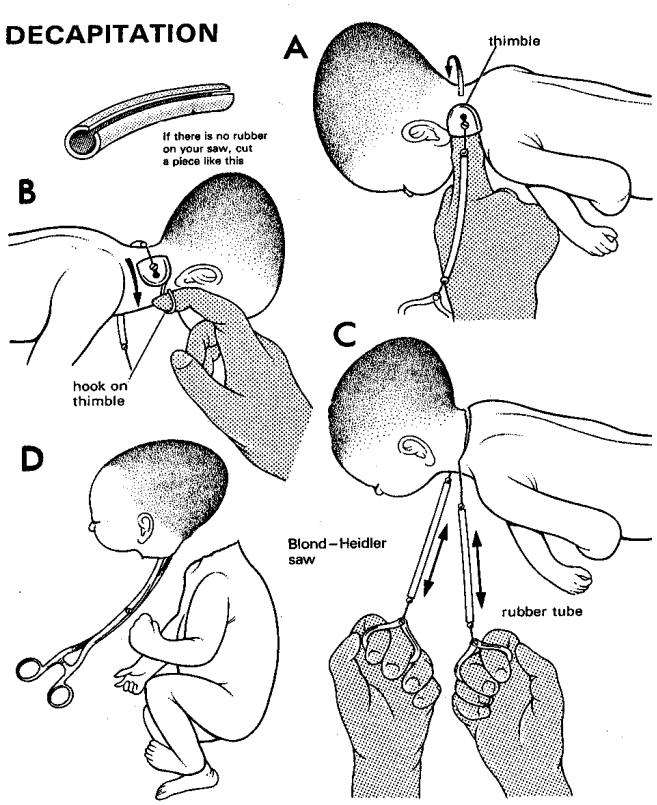


Fig. 21-11 DECAPITATION FOR A DEAD FOETUS will leave a woman with an intact uterus, which will be less likely to rupture if she delivers at home next time. Cut through the foetal neck with a Blond-Heidler saw. A, push the thimble round the foetal neck. B, pull the loop of the thimble down the other side of the foetal neck. C, saw through it. D, remove the foetal head with forceps. Pieces of rubber tube cover the outer ⅓ of each end of the saw.
Kindly contributed by John Lawson.

To deliver the foetal head, put a hand in the vagina, and turn the foetal head so that the neck points downwards. Grasp the stump of the foetal neck with large forceps, and put a finger in the foetal mouth. Then deliver the foetal head, as if it were the aftercoming head of a breech. This will prevent the stump from injuring the birth canal. If the foetal head is very large, you may need to perform a craniotomy. If you delivered the foetal head first, deliver the foetal body by pulling on the other arm. *Don't try version*: the cut foetal neck might lacerate the uterus.

If you are using scissors, hook one or two fingers round the foetal neck and pull it down. Ask an assistant to protect the vaginal wall with 2 specula. Gently pull the foetal arm. When you do this, you will feel the foetal neck. Try to see what you are cutting with each cut, because you could otherwise easily cut the uterus or bladder. Cut the foetal neck a little at a time, then deliver the foetus as before.

CAUTION! Don't cut if you cannot see the foetal neck. After each cut, pull on it. It will come a little further down with each cut until you have cut right through.

INTRA-UTERINE FOETAL EVISCERATION (GRADE 2.5)

INDICATIONS

For a transverse lie when the foetal neck is difficult to reach, but the foetal body is well down, or after decapitation if this fails to effect delivery.

PREPARATION. As for craniotomy above.

METHOD

Ask your assistant to pull on the foetal prolapsed arm, and find the foetal axilla. Protect the vaginal wall with 2 specula. Grasp the foetal abdominal wall with strong long forceps, and with strong scissors make a large opening in the foetal abdomen. Put one or two fingers into the opening and remove all the internal organs. Make sure you remove the liver, heart, and lungs. If necessary, perforate the foetal diaphragm with the scissors. Now reassess the situation, and try whichever of these manoeuvres seems best:

(1) Put two fingers behind the foetal pelvis and hook the foetal breech down.

(2) Inflate the balloon of a Foley catheter placed in the foetal pelvis with 50-60ml and apply traction (as in a breech presentation, 22.7).

(3) Grasp a leg or foot and bring that down.

(4) Try to bring the foetal neck down for decapitation by pulling on the foetal arm.

(5) Divide both clavicles (cleidotomy) to reduce the width of the shoulders of a large dead baby. Use long scissors to make a small cut in the skin of the foetal neck. Through this, guided by the fingers of your other hand, feel inside the foetal skin, until you can divide a clavicle between the tips of the opened blades of the scissors. The ends of the foetal clavicle will then overlap and narrow the foetal shoulders. *Be sure it is the foetal clavicle and not the spine of the foetal scapula that you are cutting*.

Or, finally,

(6) Separate the foetal prolapsed arm at the shoulder. Push the embryotomy scissors (19.2) through the foetal axilla and divide the internal structures from inside the foetal skin, while keeping your other hand between the foetal body and the uterus, as a constant guide. Finally, divide the foetal skin and superficial tissues under direct vision, and deliver the foetus in 2 halves.

DIFFICULTY WITH DESTRUCTIVE OPERATIONS

If you have difficulty delivering the foetus, try to visualize carefully the foetal lie and identify its anatomy. Ultrasound may help. Remember, there is usually no rush to perform this procedure.

POST-OPERATIVELY AFTER A DESTRUCTIVE OPERATION

Remove the placenta manually, and immediately feel for tears of the uterus and lower segment. Administer oxytocin IV as the foetus is delivered. Check the uterus by feeling inside it to make sure it has not ruptured. If it has, perform a laparotomy and repair it (21.17). Check the cervix, vagina, and vulva for tears, and repair them (21.15).

If the uterus is not well contracted, set up an IV oxytocin infusion with 5-20U in 500ml. Continue the saline infusion for 24h as well as perioperative antibiotics.
Monitor for:
(1) Postpartum haemorrhage in the first 24h.
(2) Acute urinary retention in the first 24h.
(3) Infection of the genital tract after 24h.
(4) Infection of the urinary tract at 7-10 days.
(5) A fistula (21.18).

If the foetal head has been impacted in the pelvis for many days, leave a urinary catheter *in situ* for 14 days. This will help to prevent a fistula. However, obstructed labour with a transverse lie does *not* cause pressure necrosis of the vagina.

CAUTION! After any destructive operation, be sure your assistant wraps up the foetus immediately on delivery. Try wrapping it in such a way that the mother can still see the face if she wishes, without exposing the debris of the operation. This will help her in the grieving process more than simply disposing of the dead foetus. In some cultures, the family may wish to bury the foetus with all due ceremony. *Be careful that you respect such traditions.*

AVOID CAESAREAN SECTION
WHEN THE FOETUS HAS DIED

21.9 Which kind of Caesarean section?

Caesarean section is the commonest emergency procedure after episiotomy and evacuation of retained products of conception carried out in a district hospital. If you are inexperienced, it will also be the one which you will be most frightened of doing. In unskilled hands, it is often fatal, as a result of:
(1) anaesthetic-related problems such as obstruction of the upper airways and the inhalation of gastric contents, spinal shock or ventilatory paralysis and overdose,
(2) haemorrhage, often related to inexperience or technical problems after earlier operations,
(3) sepsis.

There are several methods of uterotomy (opening the uterus):
(a) Lower segment, through a transverse incision (21.10). This has long been the standard operation because:
(1) a scar here ruptures 10 times less often than the scar from a classical incision,
(2) when it does rupture, it does so less dangerously,
(3) the incision in the uterus heals better,
(4) the danger of spreading infection is reduced,
(5) the placenta is less often directly underneath the uterine incision,
(6) the bowel is less likely to stick to the scar in the uterus,
(7) there are fewer post-operative complications.

There are still some problems, though:
(1) A lower segment operation needs more skill.
(2) It is still dangerous if there is intra-uterine infection, although less so than the classical incision.
(3) You may injure the bladder.
(4) Bleeding from the ends of the incision is more difficult to control, especially if there are lateral extension tears, as may happen if the lower segment is thin and distended, or the foetus is in an awkward position, as in a transverse lie. These tears may bleed severely, and in trying to control bleeding you may tie or cut the ureters.
(5) You may find it difficult to extract a distorted presenting part through a lower segment incision, and tear the uterus as you do. This tear will be dangerous, and the only way to avoid such, is to extend it. If you can do this curving the incision upwards at the edges, well and good; otherwise you may have to resort to a T incision, which does not heal well, and is a very bad incision to have to make. So only make the standard transverse incision if it is safe.
(6) The risk of spontaneous rupture is low, but not insignificant, at 1%.

Because of these dangers, we describe 5 other methods:
(b) Transverse classical, through a transverse incision low in the upper segment. This is occasionally needed if there is:
(1) a transverse lie,
(2) a contraction (Bandl's) ring,
(3) a very vascular lower segment, with many thick veins,
(4) a placenta praevia (20.11),
(5) a poorly developed lower segment especially if it is a premature delivery, or
(6) a uterus which is a mass of fibroids in its lower part.

(c) Midline classical, through a vertical incision in the upper segment (21.12). This is really only rarely indicated if:
(1) the lower ½ of the upper segment is very vascular, or inaccessible as a result of adhesions from previous surgery,
(2) a previous classical incision has healed poorly,
(3) you will proceed to hysterectomy anyway,
(4) in the rare case of postmortem delivery.
N.B. You should combine a classical incision with a tubal ligation unless there is a very good reason not to, because uterine rupture may occur spontaneously in pregnancy and occurs in up to 10% of all cases.

(d) De Lee (**Kronig or Cornell**), through a vertical incision, ⅔ in the lower segment, and ⅓ in the upper. Use this if:
(1) a lateral tear is likely, as can happen if the lower segment is very thin,
(2) the foetus is in an abnormal position, *e.g.* transverse lie, especially if the foetal back is presenting.
N.B. It is often difficult to know where the lower segment ends and the upper segment starts, so a de Lee may actually become a midline classical incision. Risk of spontaneous rupture is up to 5%.

(e) With hysterectomy. Indicated occasionally for:
(1) rupture of the uterus (21.17),
(2) severe bruising of the lower segment,
(3) laceration of the uterine artery,
(4) established uterine infection,
(5) severe placenta accreta after previous Caesarean sections,
(6) early *proven* cervical carcinoma
(*N.B. You can easily confuse this macroscopically with cervical schistosomiasis or TB.*)

(f) Extraperitoneal, by excluding the peritoneal cavity from passage of the foetus. This greatly reduces the impact of infection as septic fluids are prevented from entering the peritoneal cavity.

21.10 Lower segment Caesarean section

A quick, effective, and anatomically correct approach is the Joel-Cohen abdominal wall incision (11.2) for a Caesarean section. This is especially justified if it is combined with a tubal ligation. Adhesions behind a lower abdominal incision, likely especially after infection or keloid formation, may be very difficult. Occasionally, a lower midline incision is safer if future circumstances are uncertain, but the Joel-Cohen incision is still preferable in most instances, even if there has been a previous midline incision. It is easier to learn and perform, quicker, causes much less bleeding, heals stronger, risks less infection, and is more versatile.

(a) Pre-operative counselling
Always think of discussing with the patient the advisability of performing a simultaneous tubal ligation. Indications are:
(1) >1 previous Caesarean section.
(2) Parity >2.
(3) >30yrs of age.
(4) Hypertension, diabetes, sickle cell disease, or heart disease.
(5) Advanced or untreated HIV infection.
(6) Previous classical incision.

It is always a good idea, especially in resource-poor countries, to discuss, preferably halfway during pregnancy, whether a parous pregnant woman would like a tubal ligation in case she needs a Caesarean section. She then has time to think about it and discuss it with the relevant persons, and is not rushed into making a decision in an emergency. *A pre-printed questionnaire on her antenatal record will help remind staff and also make it routine.* This is important as patients must not feel the question is specifically directed at them, but that it is the routine to ask.

(b) Antibiotics halve the incidence of wound infection after Caesarean section: use prophylactic pre-operative benzylpenicillin, chloramphenicol IV and metronidazole PR stat as soon as you make the decision to perform surgery.

If you wait till the cord is clamped, it is too late to help prevent foetal sepsis.

If you have difficulty catheterizing the bladder before operating, raise the foetal head with your hands. If you fail to pass a catheter on the first occasion, try again after the patient is anaesthetized, when pushing up the foetal head will be easier. If you have to operate with a full bladder, *be very careful as you open the peritoneum*. Open it as far cranially as you can, and empty the bladder with a long needle *via* the abdomen.

Clip the hair where you intend to make the incision. *(Avoid shaving, which increases the risk of infection.)* Prepare the skin of the lower abdomen (21-12B), drape the abdomen with 4 plain towels, and cover these with a towel with a slit (21-12C).

EQUIPMENT. Make sure you have, if possible, at least 2 Caesar laparotomy sets (4.12) ready on stand-by at all times. Have sutures and oxytocin ready also.

(c) Anaesthesia. You can use GA, spinal anaesthesia, ketamine or LA, or a combination. *Don't let the lack of an anaesthetist inhibit you in performing an emergency Caesarean section.*

(d) Method (GRADE 3.1)
POSITION. Stand on the right side of the patient if you are right-handed (on the left if left-handed). Prevent the supine hypotensive syndrome (pressure on the vena cava) by tilting her about 5º to the left. Do this either by tilting the table, or by putting a pillow or sandbag under the right buttock. Find some way of preventing her slipping off the table. A moderate head-down (Trendelenburg) position, after the spinal anaesthetic is fixed at the correct level, will give you better access to the lower segment. It will make delivering the foetal head easier if there is a vertex presentation. It will also be an additional safeguard against vomiting.

INCISION
Pinch 3 marks in the skin crease c.3cm below the level of the anterior superior iliac spines, at both ends of your planned incision and in the midline (21-12D). Stretch the skin sideways to enable you to make a straight incision *through the superficial skin layer only (not the subcutaneous tissue).* At the centre of this incision, deepen a 2-3cm cut down to the rectus sheath (21-12F), because there are no large blood vessels here.

Now make a small transverse incision in the middle portion of the *rectus* sheath and gently separate the fascia from the sheath under it upwards and downwards (21-12H); this exposes the *rectus* muscle and makes an oval opening. Then hook 3 fingers of each hand through the midline that separates the *recti* muscles, so you get right beneath the muscle, and pull firmly laterally (21-12I). (You may even find yourself lifting a light patient slightly off the bed!) This will tuck any blood vessels in the fat out of the way.

At this point, the peritoneum is attached to the *transversalis fascia.* Stretch this layer laterally till you make a small hole in its middle, and then open it by stretching it vertically with both index fingers to just above the reflection of the bladder (21-12J). *Don't use a blade:* there is then less danger to bladder and bowel!

CAUTION! If the patient has had a previous operation, including a previous Caesarean section (21.14), omentum or bowel may be adherent to the abdominal wall and *can be easily damaged*. If you cut the bowel by mistake, clamp it and close it later (11-5). If there have been several previous Caesarean sections, the anatomy will be much distorted by adhesions.

LOWER SEGMENT CAESAREAN SECTION

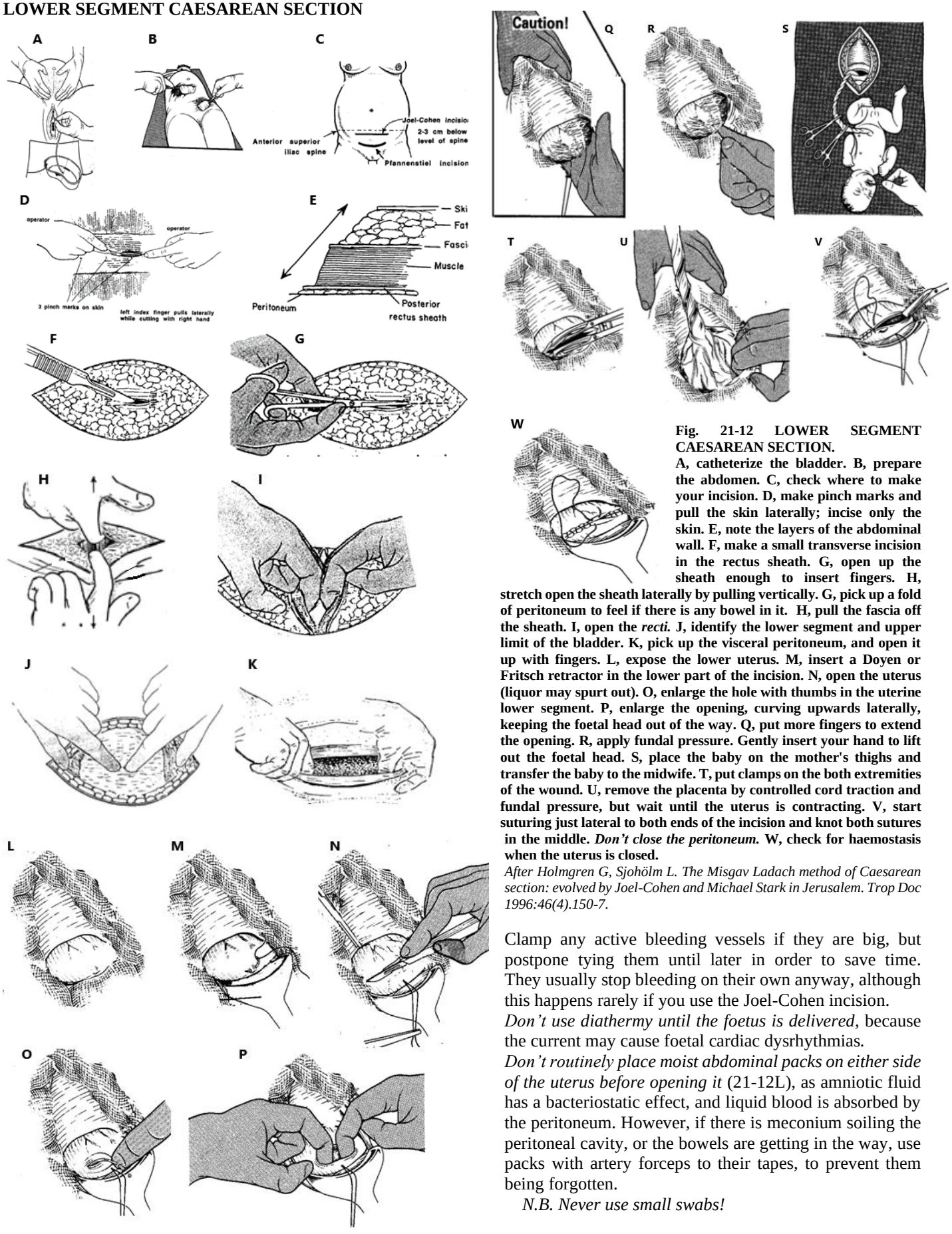


Fig. 21-12 LOWER SEGMENT CAESAREAN SECTION.
A, catheterize the bladder. B, prepare the abdomen. C, check where to make your incision. D, make pinch marks and pull the skin laterally; incise only the skin. E, note the layers of the abdominal wall. F, make a small transverse incision in the rectus sheath. G, open up the sheath enough to insert fingers. H, stretch open the sheath laterally by pulling vertically. G, pick up a fold of peritoneum to feel if there is any bowel in it. H, pull the fascia off the sheath. I, open the *recti.* J, identify the lower segment and upper limit of the bladder. K, pick up the visceral peritoneum, and open it up with fingers. L, expose the lower uterus. M, insert a Doyen or Fritsch retractor in the lower part of the incision. N, open the uterus (liquor may spurt out). O, enlarge the hole with thumbs in the uterine lower segment. P, enlarge the opening, curving upwards laterally, keeping the foetal head out of the way. Q, put more fingers to extend the opening. R, apply fundal pressure. Gently insert your hand to lift out the foetal head. S, place the baby on the mother's thighs and transfer the baby to the midwife. T, put clamps on the both extremities of the wound. U, remove the placenta by controlled cord traction and fundal pressure, but wait until the uterus is contracting. V, start suturing just lateral to both ends of the incision and knot both sutures in the middle. *Don't close the peritoneum.* W, check for haemostasis when the uterus is closed.
After Holmgren G, Sjohölm L. The Misgav Ladach method of Caesarean section: evolved by Joel-Cohen and Michael Stark in Jerusalem. Trop Doc 1996:46(4).150-7.

Clamp any active bleeding vessels if they are big, but postpone tying them until later in order to save time. They usually stop bleeding on their own anyway, although this happens rarely if you use the Joel-Cohen incision.

Don't use diathermy until the foetus is delivered, because the current may cause foetal cardiac dysrhythmias.

Don't routinely place moist abdominal packs on either side of the uterus before opening it (21-12L), as amniotic fluid has a bacteriostatic effect, and liquid blood is absorbed by the peritoneum. However, if there is meconium soiling the peritoneal cavity, or the bowels are getting in the way, use packs with artery forceps to their tapes, to prevent them being forgotten.

N.B. Never use small swabs!

You may find it helpful to place a stay suture held with a haemostat, into the lower segment (21-12L), either in the middle or at the edges, *but don't do this if you are in a hurry!*

If the foetal head is impacted in the pelvis and needs to be disimpacted from below, ask yourself if a symphysiotomy (21.7) would not have been better, and remember this next time! Ask an assistant, with sterile gloves, to put his hand into the vagina, and to disimpact the foetal head. *He should do this before you incise the uterus*. If he waits until after you have incised it, the foetal shoulders may prolapse into the wound, and make delivery difficult. Unfortunately, it is difficult to predict that the head needs disimpaction, until after you have opened the uterus. If the disimpaction fails, perform a symphysiotomy (21.7) nonetheless.

Push the bladder downwards with your index finger or a swab. Pick up the loose peritoneum of the vesico-uterine pouch with dissecting forceps (21-12J). Make a 1cm transverse cut just below the point where the loose peritoneum becomes firmly attached to the anterior uterine wall. This is the abdominal marking of the lower segment. *Don't make this opening too low, or you may not give enough room to extract the foetal head.* Open this laterally with fingers, and push the visceral peritoneum down off the bladder. Feel and observe the uterus to find how it is rotated, and identify the presenting part. It is usually rotated to the right, so that the left round ligament is usually more anterior and closer to the midline than the right. If the uterus is markedly rotated, turn it towards the midline. *If you don't allow for rotation, you may cut the left uterine artery,* because your incision will be too far to the left.

Insert a Doyen or Fritsch retractor over the bladder, to protect it for the rest of the operation.

Cut a small opening in the lower uterine segment to introduce your index finger. The membranes may bulge out or break at this point. Stretch the uterine opening laterally (21-12I) with your thumbs usually slightly more to the right than the left (as the uterus is normally rotated a little to the right, as above). For a large head, curve the incision slightly upwards with scissors, keeping the foetal head away with your fingers. The not-so-neat edges of the arteries cut by fingers bleed less and the extent of the opening is easy to control.

N.B. If you avoid using a knife, you cannot in this way cut into the foetus and transfer HIV, HBV, or HCV.

As you reach the edges of the uterus, aim your fingers in a more cephalic direction, so that the 'smile' opening in the peritoneum is c.10cm long, curved (21-12J) with its ends curving gently upwards directed to the attachments of the round ligaments. This should be ≤2cm below the peritoneal reflection, and at least ≥2cm above the detached bladder.

N.B. Don't make your transverse opening too low in the lower segment. Place it where normally the empty bladder reflects (*not where a bladder pulled up by adhesions reflects*) or 1-2cm higher.

CAUTION!

(1) *Take great care to avoid injuring the bladder*, especially if this is pulled up high and is oedematous.

(2) *Don't denude the lower segment of peritoneum for >5cm*: if the cervix is effaced and dilated, you may enter the vagina by mistake.

If the patient has had previous Caesarean sections, and the uterus is very fibrotic, you may have to extend the incision with scissors, curving it upwards laterally.

CAUTION!

(1) The lower segment varies considerably in thickness. It is thick before labour and becomes thinner during labour, so *be careful not to cut the foetus!* This will not happen if you open the uterine wall with your fingers.

(2) *Don't make the opening too small*, or the uterus will tear as you remove the foetal head.

(3) If you decide to enlarge the opening, curve it upwards at its ends in a U-shape, so as to avoid the uterine vessels. Also, when you suture it, you will be less likely to suture the ureters.

If there is a scar in the lower segment from a previous Caesarean section, make a shallow cut along it, where you want to open the rest of the uterus.

If you can feel the foetal vertex through the uterine wall, the placenta is probably lying in the fundus or posteriorly, so you can expect a delivery without difficulty.

If you tear the placenta as you open the uterus, try to detach it, and deliver the foetus around it. *Only tear through the placenta if you have to*. There can be severe bleeding from a lacerated placenta, so clamp the umbilical cord quickly (21.11).

If the ends of the opening in the lower segment bleed severely, before the foetus has been delivered, quickly proceed with delivery, and then control bleeding as described below.

If there are large veins over the lower segment, place your uterine incision towards or in the upper segment, or place clamps across the veins and incise the uterus precisely and carefully between the clamps, and deliver the foetus rapidly. The veins will probably stop bleeding soon after. If necessary, ligate the veins.

DELIVERING THE BABY

Remove the retractor. Put 2 fingers (only) into the uterus under the baby's head to decompress a vacuum, and make it easier for the foetal head to rise in the incision. Get your assistant to push downwards on the fundus of the uterus. Then put your hand outside the lower flap of the incision, and lift the foetal head up (21-12Q).

If the incision is not long enough to deliver the foetus without a lateral tear, extend its ends upwards and laterally with your fingers, so as to make a U-shaped flap.

Now ask your assistant (or the anaesthetist by groping under the drapes) to press on the fundus to assist delivery. This may mean pressing firmly, but carefully and without hurrying.

It may be necessary also to pull up the upper margin of the uterine opening to make delivery of the baby easier.

CAUTION! Don't try to aspirate the nose especially with a big Yankauer sucker: it may push maternal blood into the nose and/or traumatize the nasal passages.

ERGOMETRINE OR OXYTOCIN

If there is hypertension, or eclampsia, or you are operating under LA, avoid ergometrine, and administer 5U oxytocin IV or IM. Ergometrine occasionally makes a conscious patient sick, and may raise the blood pressure. Because you are manipulating the uterus and can easily make sure it is empty (leaving placenta behind is embarrassing), the uterus is usually well contracted during closure. But after that, you will often not notice relaxation because bleeding will not be overt. An oxytocin infusion at the end of the operation and the first post-operative hours can save lives.

If there is placenta praevia, clamp the cord quickly, because blood loss from the injured sinuses of the placenta may be significant. Otherwise delayed cord clamping, placing the baby below table level and milking the cord all serve to increase the Hb level of a neonate, especially if pre-term.

CONTROLLING BLEEDING

Now deliver the uterus by lifting the fundus out of the abdomen; it is easier then to see what you are doing.

REMOVING THE PLACENTA

When the uterus is contracting firmly, remove the placenta by a combination of controlled cord traction and fundal pressure (21-12U). If necessary, help it to contract by massaging the fundus from inside the abdomen. Pull gently on the cord, and press the uterus back with your left hand. This should deliver the placenta easily.

If there is a placenta praevia grown into a previous Caesarean section scar, there is serious danger of serious blood loss at this stage.

If you cannot remove the placenta manually (the practical definition of placenta accreta), then you may be forced to proceed to hysterectomy. While deciding or organizing blood, apply a tourniquet round the cervix (22.11).

When the placenta is delivered:

(1) Inspect the uterine cavity to make sure it is empty. Remove pieces of membrane.

(2) Make sure that the placenta is complete.

CAUTION! Don't probe the cervix to improve drainage: keep out of the contaminated vagina!

CLOSING THE UTERUS

If you are a quick operator, apply one Green-Armytage clamp (or sponge-holding forceps) at one angle of the uterine opening (21-12V), and start suturing at the opposite end.

If you are a slow operator, apply several Green-Armytage clamps (or sponge-holders) all round the cut edges of the uterus, particularly at the angles. *Make sure they don't grasp the posterior wall of the empty uterus, as it lies on the promontory of the sacrum*. You can easily do this by mistake if bleeding has been brisk. The difficulty in applying many clamps is that they will get in your way.

Don't pull on the clamps during suturing as this will result in an asymmetric closure.

Close in one layer using #1 long-acting absorbable sutures on a large round-bodied Mayo's needle. *Don't use non-absorbable sutures, particularly not on the inner wall*. Ask your assistant to hold the lower edge of the uterus forwards, while you suture from the angles inwards (21-12T). Start the 1st sutures just beyond the lateral extremity of the wound. Knot with good tension and protect the point of your needle with the needle holder; then start with a different suture and needle holder just beyond the other lateral extremity, make a similar knot and work towards the first suture, and tie this 2nd suture to the 1st in the middle of the uterine opening (21-12U). In this way, you secure the angles first. You will, if you use 90cm sutures, have at least enough material left to tie the tubes (if desired), and close the rectus sheath.

Unless the sutures are tight, they will not stop the bleeding.

CAUTION!

(1) Start suturing just lateral to the wound extremity with an adequate bite through the whole uterine wall.

(2) *Don't suture the lower edge above the upper one*, because this may advance the bladder up the uterus.

(3) *Don't include the bladder in your sutures*. If you find, at the end of your closure, that you have included it, you will probably be wise to leave a catheter in for a few days, rather than removing the sutures and starting again, which can cause severe bleeding.

(4) *Don't suture too deeply with a large needle at the angles of incision;* you may obstruct the ureters.

(5) *Don't suture the front and back walls of the uterus together*. So, before the first layer of sutures is completed, put 2 fingers into the uterine cavity, to make sure that its walls are free. If necessary, release the sutures and start again.

(6) *Don't suture the bowel to the back of the broad ligament*. Suture only the uterus, and not too deeply downwards towards the vault of the vagina. If you are in any doubt, put your fingers down behind the uterus before you start to close the lateral extremities of the opening so that the peritoneum stays out of your suture.

When the uterus is no longer bleeding, close the peritoneum of the vesico-uterine pouch with continuous sutures of non-absorbable (21-12V). *Again, avoid including the bladder with the lower edge of the peritoneum*.

COMPLETING THE REPAIR

Look carefully at your completed repair. If there is still bleeding, put in some more 'figure of 8' sutures at the bleeding points. *Don't close the peritoneum until you have controlled all bleeding*, but minimal oozing is allowed.

If you find an ovarian cyst >5cm diameter, perforate it, unless it is very large (23.9).

Now is the time to tie the tubes (19.4). The Fallopian tubes in pregnancy are swollen; *don't use rings or Filshie clips on them:* they tend to slip off or break and are costly!

CLOSING THE ABDOMEN

Clean all blood and debris from the peritoneal cavity, and especially from the paracolic gutters. They will be much cleaner if you have previously inserted abdominal packs beside the uterus (21-12K). Wash out the abdominal cavity with copious warm water if there was soiling present. Replace the uterus if you have exteriorized it. Place the greater omentum over the uterus: it will usually reach the bladder. *Don't close the peritoneum.* Close the anterior rectus sheath and bury the knots. *Don't put sutures in the fat layer. Don't insert a drain*. Leave the skin wound open if there is much sepsis.

(e) Bonding

As soon as the mother has delivered, after any necessary resuscitation, give her the baby to hold. This close early contact is important in developing the bond between them. If she has had a GA, place the baby in a cot beside her, so she can see her child when she wakes up.

(f) Post-operative care after Caesarean section

Estimate the blood loss: it will probably be more than you think, but if the membranes were intact, some of the liquid will be liquor. The average loss is 1L. Unless you have expert staff, check the vital signs yourself. Check and chart the pulse, temperature, and respiration ½hrly, until she is awake, and then, when the condition is satisfactory, hourly for 12-24h. Continue an IV infusion for 24h; start oral fluids as soon as she is fully awake. *Don't forget analgesia.* It is wise to administer oxytocin 20U in 1L IV over 6h post-operatively to ensure good contraction of the uterus. It is worthwhile making a postpartum vaginal examination: you will instantly diagnose a urinary leak, if present.

If bleeding was excessive, make sure there are 2 cannulae *in situ* and IV saline or Ringer's lactate fluid replacement is flowing nicely. Review the patient yourself in 1h. You will be surprised how often a patient who left the theatre in reasonable condition is now collapsed.

CAUTION! Look for signs of infection:
(1) Fever.
(2) A large, soft, tender uterus.
(3) Tender thickening in the lateral fornices.

If the membranes were ruptured for >24h before the operation, or there are other reasons for suspecting infection, continue antibiotics for 3 days.

N.B. Don't use antibiotics routinely post-op.

If vomiting ensues with abdominal distension, insert a nasogastric tube.

CAUTION! Before the patient goes home, make sure that she and her relatives know that future deliveries must occur in hospital: *this is ESSENTIAL!* When next pregnant, she must attend regularly for antenatal care. Give her a card which explains why Caesarean section was done, or add these details to the baby's birth card.

If there was obstructed labour and the urine is blood-stained, leave a catheter in the bladder for 10 days.

21.11 Difficulties with Caesarean section

Many difficulties attend Caesarean section, and many disasters can follow it, so the list below is long. *Torrential bleeding when you cut through a placenta praevia can kill a woman quickly. Disasters with the urinary tract are usually the result of very poor technique.* Fortunately, most of the other problems are rare. Some of the many difficulties are only seen in poor-resource settings, where inexpert operators find themselves working under difficult circumstances.

(a) Difficulties with the incision

If a patient has had a previous Caesarean section, dense adhesions may have formed between the uterus and the abdominal wall.

Don't excise a keloid scar: the keloid will probably get bigger. Just go through the middle of it. Excise redundant skin if you intend simultaneously to repair an incisional hernia (18.13). If the sides of the abdominal wall might prove difficult to line up accurately, mark a transverse line before your incision and align the points later.

Open the parietal peritoneum beyond the end of the previous scar. If you find a plane of loose connective tissue, free it with a finger. Cut fibrous bands. If dissecting the adhesions is very difficult (unusual), give up and make an upper segment incision.

CAUTION!
(1) Stay close to the uterus to avoid the bladder.
(2) If you find it helpful, open the uterus between stay sutures.

If the patient has had a previous classical Caesarean section, you would probably be wiser to perform a lower segment operation this time.

If the bladder has stuck to the lower segment, separate them with a finger, incise the peritoneum on the uterus about 2cm above the bladder. Lift the lower edge in forceps to stretch the adhesions between the bladder and the uterus. Cut them close to the uterus, keeping the points of the scissors directed at it. If this is difficult, give up and make an incision c.3cm above the area where the bladder and the uterus have stuck together.

If the incision in the uterus tears as you extract the foetal head, there will probably be a vertical tear in the corner which will run down behind the bladder, often with heavy bleeding. If you are alone with the scrub nurse, ask for an extra assistant. Exteriorize the uterus by drawing it out of the abdomen. Identify the edges of the incision and the tear. Mobilize the bladder further downwards if necessary.

If you cannot define the extent of the tear, carefully open the broad ligament by cutting the round ligament (21-18). This will let you feel the ureter, so that you can avoid it before you apply any clamps. Now apply Green-Armytage forceps to the edges of the tear, and draw its angle into view.

Apply direct pressure with a dry pack, find the bleeding vessels, and tie them. Use interrupted sutures in the area of the tear. These will be easier to unpick if you find you have caught the bladder or the ureter by mistake.

CAUTION! After repairing a tear, check visually that the ureter has not been caught in a stitch by mistake. If you have a doubt, inject dye into the ureter to see if it leaks.

If these measures fail, the only way to control bleeding may be to tie both the uterine arteries, just after they have entered or branched into the uterus or cervix.

If you are unable to repair the uterus, perform a subtotal hysterectomy (21.17).

(b) Difficulties with particular presentations

If there is obstructed labour with a cephalic presentation, enter the abdomen just below the umbilicus so as to avoid the bladder. If catheterization before the operation was impossible, empty the bladder now with a needle and syringe. Much of the swelling will be oedema, which will not go away. Mobilize the bladder free from the lower segment as usual. If necessary, get an assistant now to push the foetal head up from below through the vagina, before you open the uterus. Otherwise, the foetal shoulder may prolapse into the incision and make delivery more difficult. Make a transverse incision in the lower segment. Choose its level carefully. If it is too high, delivery will be difficult; if it is too low, you may have technical difficulties closing the vagina or you may even incise the vagina.

If delivering the head is difficult, don't panic. Everyone finds this a problem, especially when the uterus is tight around it. Take time to push the uterine wall back from around the head, by inserting 2 fingers all round. If you still have difficulty, enlarge the wound curving upwards laterally in a U-shape.

CAUTION!

(1) *Don't lever the head out with your whole hand*, because this can cause vertical downward tears in the lower segment.

(2) If the liquor was purulent or infected, clean the abdomen carefully, and wash out the pelvis with warm saline. Administer antibiotics for at least 5 days.

If there is a breech presentation, feel for a leg or, better, both legs, and deliver the foetus breech-first as if you were delivering the head. Then deliver the head slowly, or you may damage it.

If, by mistake, you take hold of an arm, replace it. Then feel for a leg; recognize it by feeling for the heel.

If an arm comes out and will not go back, you are in trouble: you may have to make an inverted 'T' incision to get the foetus out.

When necessary, deliver the head (22.7) by a modified Mauriceau-Smellie-Veit manoeuvre, and the arms by pressure on the upper arms which you feel by palpating the back, or by a modified Løvset manoeuvre.

If there is a transverse lie, the choice of incision is important (21.9).

(1) **If the mother is in early labour, and the lower segment is poorly developed**, with most of the baby in the upper segment, make a transverse incision in the upper segment and deliver the foetus by breech extraction (22.7).

(2) **If early labour has begun, the lower segment is well developed, and the membranes are still intact**, make a transverse incision in the lower segment, and deliver the foetus by breech extraction.

(3) **If labour is obstructed, and most of the foetus is in the overdistended lower segment**, simple delivery through a transverse incision in the lower segment will cause large tears. So, if the foetus is alive, make a vertical incision in the lower segment, and extend the incision into the upper segment until it is big enough to effect delivery.

(4) **If the foetus is dead**, try to avoid opening the uterus. Only make a transverse incision in the lower segment as a last resort and decapitate or eviscerate the dead foetus in order to deliver it in any convenient way (21.8).

CAUTION! Don't try to deliver a dead impacted foetus intact, because this will tear the lower segment severely. *Don't make a classical or inverted 'T' incision for a dead foetus*.

(c) Difficulties with the placenta

If you anticipate placenta praevia, expect difficulty, and get help if you can. You can usually use the ordinary transverse lower segment incision. *This is, however, contraindicated if*:

(1) there is a poorly developed lower segment, which would not allow a transverse incision of adequate length.

(2) there is a very vascular lower segment with large veins on it: this might be a placenta accreta.

(3) the presenting part is high, and the foetus is lying transversely, indicating that the placenta praevia is probably central. If so, mobilize the utero-vesical fold, as for a lower segment operation. Make a low vertical midline or a transverse incision in the upper segment.

If you find placenta in the incision:

(1) Peel it away from the uterine wall and enter the uterus from above it.

(2) When the edge of the placenta is too far away to allow this, push your finger through it quickly, and deliver the foetus without delay through the hole that you have just made.

If you meet the cord, clamp it before you deliver the foetus, but *don't waste time looking for it*: you can clamp it immediately afterwards. Remember that a baby can easily bleed from an injured placenta. The mother can also bleed, so if you see a large bleeding vessel in the placental bed, control it with a figure-of-8 suture.

Beware of a placenta praevia after a previous Caesarean section: it might very well be a placenta accreta, increta or percreta. If you can't get the placenta off the uterine wall in this situation, you have confirmed the diagnosis and should proceed directly to a hysterectomy.

N.B. If, beforehand, a referral is possible to specialists where there is a reliable blood bank, that would be best.

If there is post-operative bleeding (not uncommon with the common type of placenta praevia), it is probably arising from the lower segment at the site of the attachment of the placenta. Administer oxytocin, and if necessary transfuse blood. In desperation, pack the uterus (22.11) using a condom in the uterus, attached to an infusion to fill it as a balloon.

(d) Secondary PPH

If there is severe vaginal bleeding 8-14 days after delivery, the operation site is infected (common after an obstructed labour with sloughing of the tissues) or a piece of placenta was left behind, under perioperative antibiotic cover. Take the patient back to theatre, and examine her under GA. Put a gloved finger into the uterus through the external os and feel for a piece of retained placenta, and for the inner wall of the uterine scar. If this feels weak, or has broken down, re-open the abdominal incision. You may find, especially in the presence of HIV infection, a soft necrotic bleeding uterus, with blood and spreading infection in the peritoneal cavity. What was the scar may now be an infected hole in the uterus. Under such circumstances, perform a subtotal hysterectomy (21.17). *If you don't attempt this, she will die*. Expect to find that the parametrium is acutely infected and swollen, so that it feels like cheese. In this situation, treat the patient for septic shock with much IV fluids and broad-spectrum antibiotics.

(e) Difficulties with the urinary tract

If you notice that you have opened the bladder, which is not often detected during the operation, identify the hole carefully, hold its edges with Allis forceps, mobilize the surrounding tissues if necessary, and bring its edges together with continuous inverting sutures of 2/0 or 3/0 long-acting absorbable. Drain the bladder continuously with an indwelling catheter for 14 days.

If you have injured the ureter at operation, first check that the other ureter is intact. Either, repair the damage if you can with 4/0 interrupted long-acting absorbable. Or, insert a fine T-shaped tube into the ureter, bringing out the 'leg of the T' to the abdominal wall, and close the abdomen. Later, refer the patient for expert help. *Don't fashion an external ureterostomy,* as this will stenose.

If there is anuria:

(1) This may be the result of severe hypotension while the patient was in obstructed labour (not uncommon) or the result of pre-eclampsia. Hydrate the patient well and add IV furosemide 40mg.

(2) Both ureters may have been tied or damaged. This you will find out usually the day after the operation. Perform an ultrasound to look for hydronephrosis. If you think you have tied one ureter or both, re-open the abdomen. For each side, check if the ureters are patent by injecting blue dye proximally. If no dye comes out in a urinary catheter, remove the uterine sutures one by one. Then close the uterus again. Otherwise refer the patient for reconstruction; if you cannot, except after considerable delay, fashion bilateral temporary nephrostomies (27.14).

If there is a severe dull pain in one loin post-operatively, you may have tied one of the ureters. Proceed as above. Otherwise refer the patient as before.

N.B. Sometimes, when the ureter is damaged, neither the patient nor you are aware of it: the kidney merely stops functioning.

If urine discharges from the vagina after 2-5 days, there might be:

(1) Overflow incontinence. Check if the bladder is distended. This can happen if there is bladder atony due to stretching of the detrusor muscle fibres. Keep in an indwelling *(not a condom)* catheter for 4wks. Expect complete recovery.

(2) Bladder or urethral stress incontinence. Ask the patient to cough: you will see urine spurting out of the urethra. If it is disabling, refer her for a colpo-suspension.

(3) A fistula. Pass a Foley catheter and perform a dye-test by instilling methylene blue in the bladder.

N.B. Not all urinary leakage postpartum is due to a fistula!

If the test is +ve, the dye comes out from the anterior vaginal wall (juxta-urethral, 21-20A or mid-vaginal VVF, 21-20C) or the cervix (juxta- or intra-cervical VVF, 21-20E,F). The first is due to pressure from the foetal head during prolonged obstructed labour. The last is the result of cutting the bladder or stitching the bladder together with the lower uterine segment during the Caesarean section.

Don't prescribe antibiotics (they are of no use) *and don't send the patient away for evaluation in 3 months' time!*

Contrary to general belief, a great deal can be and should be done immediately by very simple measures under any conditions, however deprived of resources, by whoever is involved in postpartum care. The principle is to decompress the bladder totally for a sufficiently long time to give it the opportunity to heal spontaneously as the fresh wound edges lie against each other. The earlier you do this, the better the chance. Some 25-30% of fistulae will heal spontaneously in this way and larger fistulae will reduce to half the size.

It is still worthwhile trying with fistula presenting up to 3 months postpartum. Open drainage of the catheter into a pot or a plastic bowl is often better than closed drainage into a urine bag, which is often carried on the head or shoulder! Therefore it is good to fix an IV giving set to the catheter to allow the patient free mobility.

There should be free drainage at all times: instruct the patient to take care not to block the catheter or to lie upon it when she sleeps.

In all cases keep a sufficiently large (Ch18) indwelling catheter *in situ* for at least 4-6wks, filling its balloon with a *maximum* of 10ml water. Encourage the patient to drink at least 5L fluids daily in order to produce a minimum of 4L urine/day: the urine should be completely colourless and odourless like clear water. If she does not drink enough and the catheter gets blocked, flush it or change it for another. *Don't allow it to get blocked or infected!*

Early on, sitz baths bd are useful, to prevent leaking urine irritating the skin. Remove any necrotic tissue from the vagina.

Support the patient with haematinics, and a high-protein diet. Insist on immediate mobilization: *it is a catastrophe to develop contractures and bedsores because of a fistula.*

For the first few days the patient should stay in the hospital under close supervision to monitor her and to instruct her properly in catheter care and drinking. Then when her general condition is satisfactory she can be treated as an outpatient. She should come every week to report on the leak and to be instructed again to drink as much as 5L/day.

After removal of the catheter, instruct the patient to continue drinking and to pass urine frequently. She has to refrain from sexual intercourse for 4-6 months. She must attend antenatal care at subsequent pregnancies and deliver in a hospital at all subsequent deliveries.

If after 6wks of catheterization, the urine still leaks all the time *(i.e. not dry at night)*, and upon vaginal examination the fistula is too big or the balloon is inside the fistula, remove the catheter and try to refer the patient to a hospital where fistula repair is done regularly: *the first attempt gives the best result.* If referral is impossible, you may be justified to attempt repair of simple fistulae (21.18).

If the dye test is -ve, there might be a leak from a uretero-vaginal fistula. To confirm this, do the 3-swab test: put 3 pieces of cotton wool in the vagina, instil methylene blue through the catheter and let the patient walk around for 20mins. After that, remove the cotton swabs one by one.
(1) If the first swab is blue, there is urethral incompetence or a urethro-vaginal fistula.
(2) If the middle gauze is blue, there is a vesico-vaginal fistula.
(3) If the last gauze is blue, there is a vesico-cervical or vesico-uterine fistula.
(4) If the last gauze is wet but not blue, there is a ureteric fistula. This type of fistula is probably caused by damage to the ureter at Caesarean section or hysterectomy by:
(a) clamping it in error, not recognizing this, and leaving the clamp on for more than a few minutes, or
(b) including the ureter in a suture whilst closing the uterine opening. An IVU will tell you which side is involved.
The kidney on the affected side will show some degree of hydronephrosis. The left side is almost twice as often damaged as the right side. There may or may not be pain in the loin. A uretero-vaginal fistula has a better prognosis and is less urgent than tied ureters, because it means that the kidneys will not stop functioning. Try to refer for elective repair: the ureter may need re-implanting into the bladder, or repair end to end. To maintain good kidney function, refer without delay.

If there is a contraction (Bandl's) **ring** in the lower segment, or between the lower and the upper segments, deal with it like this: If the foetus is entirely above the ring, make a transverse incision entirely above it. If it is round the neck, make a vertical incision across it.

If there are uterine fibroids, leave them unless they are pedunculated and removal is very easy. In this case, make a V-shaped incision, *not a straight transection,* so that closing the uterine surface is possible without tension. Otherwise, leave them: they may settle and atrophy. Removing a fibroid at delivery, from within the wall of the uterus, causes severe bleeding.

If there are ovarian cysts or tumours, remove them if they are >5cm diameter. Ovarian cystectomy is possible, but removing the ovary and tube will be quicker and safer. Smaller functional luteal cysts will have usually disappeared spontaneously by the end of pregnancy.

If there are dense adhesions, you will have to separate them sufficiently to get good access to the uterus. *Don't try to remove them from around the tubes and ovaries*; they will ooze and form again.

21.12 Alternative methods of Caesarean section

(a) Transverse classical in the upper segment
Check that the uterus is wide enough. Incise the peritoneum over the lower part of its upper segment with a scalpel. Mobilize it away from the incision with scissors, and incise the uterus transversely in the midline. Enlarge the incision to the right and left, by stretching it with your fingers (it is usually too thick to be cut with scissors), and deliver the baby by breech extraction.

(b) Midline classical in the upper segment
The midline classical Caesarean section is seldom done by experienced obstetricians. Because rupture of the uterus is such a danger with subsequent pregnancies, perhaps as early as 28wks, you must close the uterus with particular care, and do all you can to persuade the patient to consent to a simultaneous tubal ligation. Many steps are similar to those for a lower segment operation. However, the uterus is much thicker in the upper than in the lower segment, so you will have difficulty opening it using your fingers only. Make a 12cm vertical midline incision: *check carefully that the uterus is not rotated*, before cutting!

MIDLINE CLASSICAL CAESAREAN SECTION

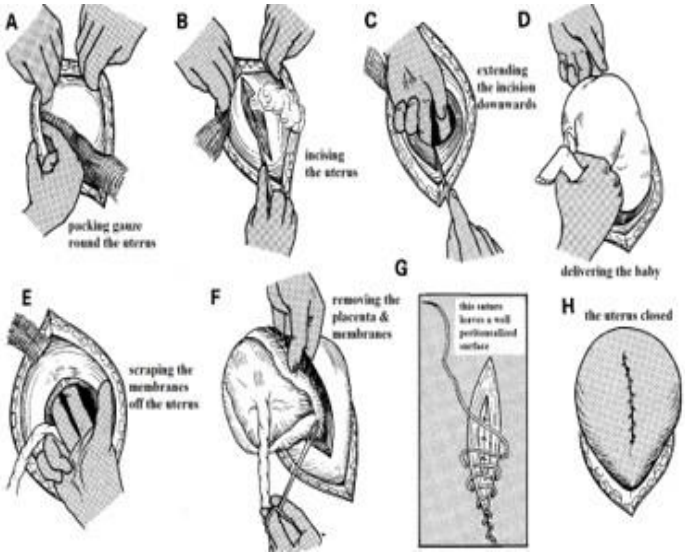


Fig. 21-13 MIDLINE CLASSICAL CAESAREAN SECTION.
A, packing gauze round the uterus. B, incising the uterus. C, extending the incision downwards. D, delivering the baby. E, emptying the uterus. F, removing the placenta and membranes. G, an anchor stitch has been inserted, and the wound is being closed by an inverting suture, which pierces each wound edge from within outwards. H, this buries the peritoneal surface of the wound, and minimizes the formation of adhesions.
After Bonney V. Gynaecological Surgery. Baillière, 1971 Figs 331-7 with kind permission.

(c) De Lee (Kronig/Cornell) vertical
In ⅔ of the lower and ⅓ of the upper segment
To make a de Lee incision, incise the visceral peritoneum transversely, as described below but high on the lower segment. Mobilize the peritoneum and the bladder well down. Find the midline of the uterus. Insert a small transverse suture where the bottom end of your incision is going to be, to prevent it extending downwards behind the bladder. Make a longitudinal incision, two-thirds of it in the lower segment, and one-third in the upper segment.

Later, repair a de Lee incision with two layers of continuous #1 or #2 absorbable. *Don't catch the full thickness of the uterine wall in the first layer*: it is often too thick. Make sure you include the uterine fascia in the second layer, or it will continue to bleed. Repair the peritoneum and pull it up high, so that the top of the incision is covered, preferably with a locking suture (4-8G).

If you have made a long cut in the upper segment, tie the tubes on the same indications as in a classical Caesarean section.

MODIFIED EXTRAPERITONEAL CAESAREAN SECTION

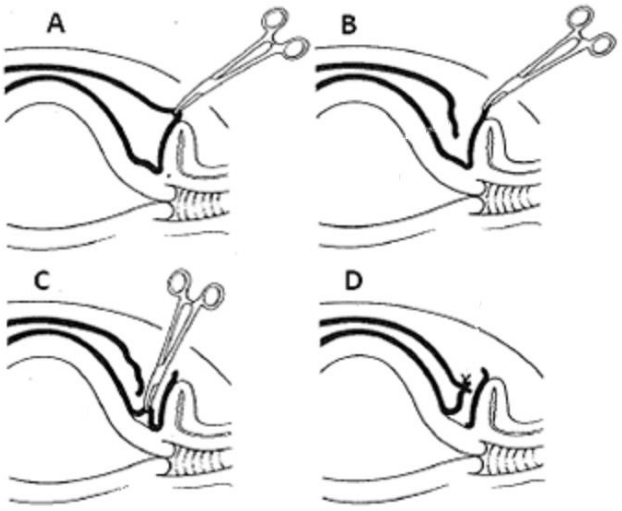


Fig. 21-14 MODIFIED EXTRAPERITONEAL CAESAREAN SECTION. A, holding the parietal peritoneum, B, separating it from the *rectus* muscle, and cutting it transversely. C, picking up the utero-vesical fold. D, suturing this to the parietal peritoneum to leave a passage for the foetus to pass through.

(d) Modified extraperitoneal
This method has undergone a renaissance, being recommended not so much where antibiotics are absent, but where infection is rife. The idea is that the foetus, on its way out of the uterus, does not pass through and so does not contaminate the peritoneal cavity (21-14).

Separate the parietal peritoneum from the *rectus* muscle down to the level of the dome of the bladder, and cut it open transversely to its lateral limits. Similarly cut open transversely the utero-vesical fold.

Then suture the superior leaves of the parietal peritoneum and of the utero-vesical fold together, thus sealing off the peritoneal cavity from the lower uterine segment, before you actually open this. *Don't exteriorize the uterus!*

21.13 Infection following Caesarean section

Peritonitis (10.1) may follow any obstructed labour, or an infected Caesarean section, and is common after rupture of the uterus. Death is a real risk when:
(1) labour is abnormally long: the longer, the greater the risk,
(2) the foetus is dead,
(3) membranes rupture early and liquor becomes infected,
(4) HIV infection is present,
(5) sterile procedures are poor.
Use pre-operative antibiotics and proper sterile practice to try to prevent sepsis (2.7).

Infection may take the following forms:
(1) Wound infection (11.13) may discharge through the scar into the cavity of the uterus. Infection may resolve, or you may need to drain pus suprapubically. If fever recurs with signs of more pus collecting, do another drainage operation.
(2) Pelvic abscess (10.3), which will need draining suprapubically or *via* a gridiron incision.
(3) Peritonitis (10.1): You will probably find that the uterus is totally disrupted, so it is hopeless to try to repair it. A subtotal hysterectomy (21.17) will usually be enough, commonly with the removal of both adnexa, but retain one if you can.
(4) Subphrenic abscess (10.10), which is a common late complication, and is likely to kill the patient if you don't drain it; as may multiple abscesses between loops of the bowel (10.3).
(5) Secondary postpartum haemorrhage (22.11,12) may occur with a retained segment of placenta, needing evacuation, or an infected uterine scar, which will mean a hysterectomy (21.17).
(6) Burst abdomen (11.14) occurs especially after several Caesarean sections, and needs repair.
(7) Infertility (19.3) is a late complication of infection.
(8) Bowel obstruction and low-grade peritonitis will occur if packs or swabs have been left behind in the abdomen.

21.14 Elective Caesarean section, 'trial of scar', or Caesarean section early in labour?

If a woman has had one Caesarean section, the alternatives for the next pregnancy are:
(1) an elective Caesarean section, before labour ensues.
(2) a Caesarean section in early labour.
(3) an attempt at vaginal delivery (a 'trial of scar' (TOS), or 'vaginal birth after Caesar' (VBAC)).

A lower segment Caesarean section is sometimes done for such conditions as foetal distress, placenta praevia, or the prolapse of the cord or an arm, which are unlikely to happen again in a later pregnancy. When a woman like this becomes pregnant again, there is every reason to expect that the labour will be normal, except for the scar that she now has in the uterus. This will almost always give some warning before it ruptures, so, with good supervision, you can safely let her have further attempts at delivering vaginally.

She can have as many trials (TOS or VBAC) as she likes, provided the previous one was successful, but she must have only had 1 previous Caesarean section. Sometimes, however, after several TOS there is only a very thin membrane, the peritoneum, separating the uterine and the abdominal cavity. A subsequent TOS is more likely to end in disaster in this case.

If a woman has had ≥2 Caesarean sections, you should nearly always repeat the Caesarean section.

N.B. It is a good idea with any multipara to discuss during the antenatal period whether, if she needs a Caesarean section in any case, she would also like a sterilization. Note on her medical card that it has been discussed and what the result of the discussion was. This is even more important where women have one scar from a Caesarean section.

TWO CAESARS OR MORE
ALWAYS A CAESAR AGAIN

When you try a trial of scar, admit the patient to hospital and monitor her closely. If the scar shows signs of rupturing, perform a Caesarean section immediately. These warning signs only last 1-2h before the uterus ruptures, *so you must monitor her with the greatest care*.

If CPD was the reason for the Caesarean section, it reduces the chances of a successful trial of scar in the current pregnancy, but *does not exclude it*, because: (1) the pelvis continues to grow up to the age of 19yrs, (2) uterine action is often poor in the under-16s.

NO TRIAL OF SCAR
IF THE PREVIOUS CAESAREAN SECTION
WAS CLASSICAL MIDLINE!

You can assume that no progress after 6cm cervical dilation in the 2nd stage of labour after a Caesarean section, with a vertex presentation and good (oxytocin-induced) contractions, means CPD. Caesarean section for foetal distress in developing countries is most often related to CPD.

ONE VERTICAL SCAR, ALWAYS ANOTHER

Good care during a trial of scar means that monitoring must be reliable, and you must be able to perform a Caesarean section within 1h, even if it is 3am at night.

If the organization and discipline of your hospital are not such that it can provide care of this quality, elective Caesarean section will give the mother a better chance of saving her foetus, as well as her own uterus, and perhaps her life.

If it takes several hours to find a driver, to fetch you, and to prepare the theatre, a trial of scar will be dangerous. Ideally, *uterine rupture should never occur during a trial of scar*.

Even when conditions are not ideal, a trial of scar may be justified, because the immediate and future risks of a further Caesarean section can be considerable. If a woman knows that she cannot have a trial of scar in hospital, she may try to have a dangerous trial by herself at home. A woman will usually understand if you say, "We will give you a trial, and if you have any difficulty, we will perform another Caesarean section."

Ask the clinics to refer all women who have had a previous Caesarean section, and who are sure of their dates, at 34wks, so that you can assess. If a trial of scar is not indicated, plan an elective Caesarean section at 38wks, or in early labour, if a woman is not sure of her dates.

Elective Caesarean sections are a way to avoid a trial of scar, but they are not the complete answer: you may perform a Caesarean section too early because the dates may be uncertain, but even if they are certain, they need to be confirmed by early ultrasound, and risk prematurity. Furthermore, you may become unpopular, so find out what the local women think.

The best indication that a uterine scar is going to rupture is a tachycardia, or foetal distress. Make observations ½hrly. If the foetal pulse rises >160/min, or drops persistently <100/min, or there is pain *between* contractions, the scar is probably rupturing, so proceed to Caesarean section. Other signs are described below.

HARBANS KAUR (38yrs, gravida 4, para 3) was admitted at 9am on a Saturday, for a trial of scar, having had one previous Caesarean section with the first pregnancy. She was 7cm dilated and had good contractions. At noon she was fully dilated and the foetal head was $^{3}/_{5}$ above the brim. During the next $^{1}/_{2}$h it remained there. The doctor on duty was called for another emergency Caesarean section, so the intern was advised to attempt vacuum extraction. He failed, but in doing so, he included the cervix under the cup, and tore it. At 3pm she developed pain, shock, and abdominal tenderness, and the foetal heartbeat disappeared. She was rushed to the theatre. The uterus had ruptured, and the tear had extended into the bladder. The superintendent was called. He found that the ureter had been caught in a hastily applied suture. The following day she was found to be leaking urine vaginally.
LESSONS These are many; they include: (1) In multiparas the second stage should not last longer than 20 minutes. (2) A vacuum extractor was applied when the head was 3/5 above the brim. It should be only $^{1}/_{5}$ above or less (except for a trial of vacuum or symphysiotomy). (3) When you apply the cup of a vacuum extractor, you should make sure that you don't include the cervix. (4) When a trial of scar is done, it must be possible to perform an immediate Caesarean section if the trial fails.

WARNING SIGNS MAY ONLY LAST 1-2H
BEFORE RUPTURE

TRIAL OF SCAR
INDICATIONS
(1) A woman who has had only one previous lower segment Caesarean section, and the indication for it is absent in the current pregnancy.
(2) A scar from a myomectomy (provided the uterine cavity was not opened during the operation), hysterotomy (21.5), or uterine perforation during a D&C.

N.B. A request for tubal ligation may induce you to perform an elective Caesarean section. On its own, it is not a reason to avoid vaginal delivery, because this followed by tubal ligation will still be safer for the patient whether she arrives in labour or not.

CONDITIONS
(1) Only ≤1 previous Caesarean section.
(2) In hospital.

(3) Caesarean section must be available within 1h any time of the day or night.
(4) Estimated foetal weight <4kg.
(5) A vertex presentation in the occipito-anterior position.

CONTRAINDICATIONS
(1) ≥2 previous lower segment Caesarean sections.
(2) 1 previous classical midline Caesarean section.
(3) Any degree of CPD, or suspected CPD in this pregnancy.
(4) An occipito-posterior presentation.
(5) Any other form of malpresentation (except perhaps a breech, if you are experienced) or obstetric complication.
(6) Foetal or maternal distress.

METHOD. Assess all pregnant women with a uterine scar in the antenatal clinic at 36wks. Take a careful history. Assess the pelvis clinically and assess the size of the foetus by measuring the height of the fundus; if it is >40cm, don't do a trial of scar. A previous successful vaginal delivery at term is a good omen.

Ask the patient to avoid heavy work during the last month of pregnancy, or to come in for rest. If possible, admit her to a waiting area at 36wks for rest and observation. When labour starts, restrict oral intake to fluids only. *Don't as a rule induce labour*. Unless your blood bank can be relied upon to have blood available within 1h, have it cross-matched, and ready to use if necessary. Record the pulse and the foetal heart rate carefully.

You may sometimes be able to feel the scar in the lower segment at vaginal examination. This will be easier if you are using epidural anaesthesia. If it bulges or feels weak, perform a Caesarean section immediately: you may be able to assess weakness of the scar on ultrasound. The tenderness of a scar is difficult to assess in labour, and is not on its own an indication for Caesarean section. Assist delivery with vacuum extraction, if necessary.

Abandon the trial if:
(1) The alert line is crossed on the partograph!
(2) The foetal pulse rises to 160.
(3) There is pain between contractions.
(4) Pain is generalized.
(5) There is unexplained vaginal bleeding or haematuria, *though this may be caused by the balloon of a Foley catheter being inflated in the urethra when the foetal head is low*.
(6) Uterine contractions cease.
(7) There is rectal or vaginal tenesmus.
(8) Restlessness ensues.
(9) Palpation of the uterine scar causes the patient to hit you on the hand!

Stay with the patient during labour so that you can examine the lower uterine segment vaginally immediately after delivery of the placenta, so as to be sure that it has not ruptured. This is uncomfortable, but does not need anaesthesia if you do it just after the placenta is delivered. *Don't delay because vaginal examination is an insufficient indication for a GA!* If you find a rupture, which may present as postpartum haemorrhage, *don't delay but proceed to laparotomy* (21.17).

21.15 Birth canal injuries

(a) 1st and 2nd degree perineal lacerations
The objective of your repair is to achieve haemostasis and to approximate any disrupted tissues to assist natural healing. Good anatomical alignment is essential. You need good lighting properly to identify the structures involved.

Ensure that your patient has adequate analgesia and is as comfortable as possible throughout the initial assessment (*i.e.* the systematic examination of the perineum, vagina and rectum) and also the subsequent operational procedure. For minor injuries LA might be sufficient, but if the patient is uncomfortable or distressed, a GA or spinal anaesthesia may be more appropriate.

METHOD
Insert a large swab or tampon made from sterile compresses tied together with a thick thread into the upper fornix of the vagina to absorb any bleeding. Leave the 'pull string' visible at the vulva to prevent you forgetting it when the procedure is over.

It is important to reach the upper edge of any wound. Put a stay suture at upper end, leaving the thread long. Place another suture c.1cm above the apex of the tear to avoid bleeding from retracted blood vessels.

Close the vaginal laceration from inside to out up till the hymenal margins, using a continuous (spiral) suture. *Leave no cavity underneath the vaginal wall* because this can produce a painful haematoma, which may become infected. *Take care at the same time not to suture too deeply to avoid injury to the rectum*. Perform a rectal examination after the procedure to ensure that no suture material is inserted into the rectal mucosa. Use the hymenal margin on both sides as a landmark for correct vaginal re-adaptation.

Repair the *bulbocavernosus* and *transverse perineal* muscle layers with 2-3 absorbable single sutures, and the perineal skin with rapidly absorbable continuous or interrupted sutures.

If there are multiple vaginal lacerations with friable tissue, insert a vaginal tampon and remove it after 24h; leave a catheter in the bladder while the pack is *in situ*.

(b) A 3rd degree tear (GRADE 1.3)
You can nearly always avoid 3rd degree (anal sphincter) tears by controlled pushing of the fourchette (21-8B) from both sides to the midline with your fingers if a tear is imminent. An episiotomy does not altogether prevent a 3rd degree tear. It is not proven but likely that massage and stretching of the perineum before the largest diameter of the head is delivered might help in preventing large tears. These large tears follow instrumental more often than simple vaginal deliveries. If a vacuum extraction is performed for foetal distress, marginal CPD or exhaustion, *it does not mean an episiotomy is also always needed* (a narrow pelvis can co-exist with a wide vulva).

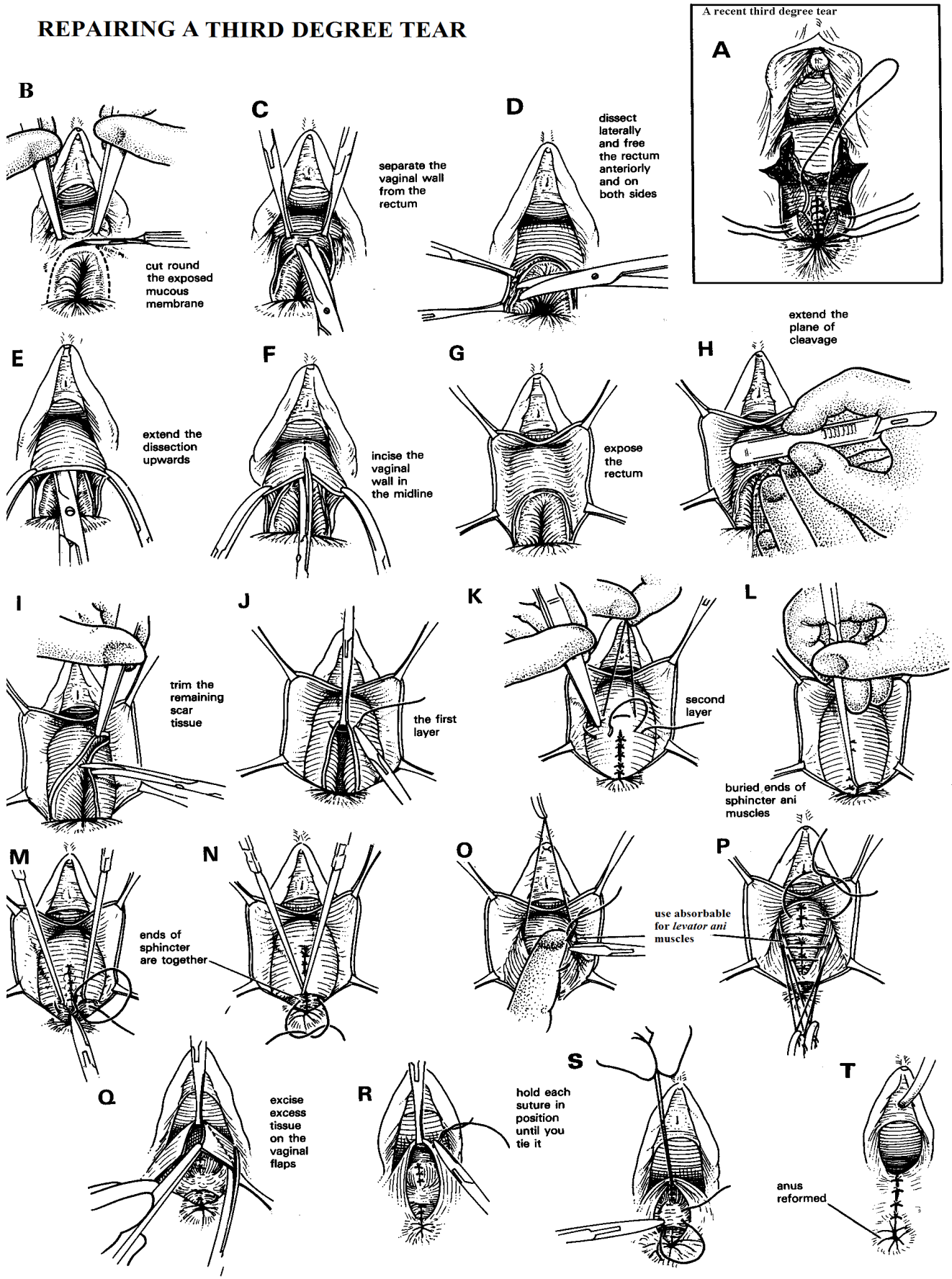


Fig. 21-15 REPAIRING A 3rd DEGREE TEAR. A, a recent tear. B-T, an old one. *After Parsons L, Ulfelder H Atlas of Gynaecological Surgery WB Saunders 2nd ed 1968 p.259,261 with kind permission.*

On the other hand, a fast delivery through the soft tissues of the vulva gives the tissues less time to stretch and, although a vacuum cup does not increase the diameter of the foetal head, a large tear becomes somewhat more likely, often because you are impatient or worried about the wellbeing of the foetus.

Don't blame the midwife. She will be upset anyway, and will be tempted to conceal such a tear if you are harsh. A common mistake is to support the perineum too vigorously, so as to force the head against the pubis, and tear the tissues. It is best to repair 2nd (perineal muscle) or 3rd degree tears within 24h of delivery, provided they are suitably clean, or delaying till they are so. The problem is that, if you wait, the tissues may become very distorted and stenosed (21.16).

With a recent 3rd degree tear:
(1) Start by suturing the edges of the patient's rectum together.
(2) Cover these sutures with a layer of fascia.
(3) Suture the anal sphincter with 2-3 interrupted sutures.
(4) Close the vaginal and perineal skin. If a tear is old, first incise and reflect the skin which has grown over it.

Repair this as soon as possible in theatre; you usually will need GA or spinal anaesthesia. *Don't consider this a trivial operation*.

The best chance of success is the 1st attempt. If you fail, the patient is condemned, at best, to some episodes of faecal incontinence.

METHOD
Use the lithotomy position, with the buttocks hanging well over the edge of the table. Shine a good light on the wound. Clean it and the skin round it thoroughly. Put a large gauze pack with a tape attached to it into the vagina. This will keep the tear free from blood, but *be careful that you don't obscure occult uterine bleeding.* Get a competent assistant to retract the vaginal wall while you survey the tear.

If the tear goes high up the rectum and vagina, you must repair these in separate layers, first dissecting them free from one another. More distally, the perineal body makes a clear separation between the two. Suture the rectal serosa with interrupted or continuous sutures on a round-bodied curved needle, starting at the apex of the tear from outside inwards, so that the knots end up on the outside of the rectum.

If the rectal tear is very extensive, pick up the pre-rectal fascia with a second row of sutures, reinforcing the first layer. To close the *external sphincter ani*, look for the torn ends of this muscle at the left and right postero-lateral positions (4 & 8o'clock), which may be deeply retracted and rolled up laterally. Search for these with hooks or baby Babcock forceps (*artery forceps will damage the muscle and tear it*). Pull on the ends of the muscle on both sides, and get your assistant to hold the forceps approximated.

Place your index finger in the rectal lumen: you should feel firm contraction over it. *If you do not, you will need to look again for the retracted end of the sphincter muscle*. Then insert 3 deep long-acting absorbable sutures through the muscle *and surrounding fascia* (if you pick up muscle alone, the sutures will cut out), making sure you exclude the forceps. *Don't tie the sutures until you have removed the forceps* (21-15A); try to approximate the 2 blocks of tissue accurately and tie them together without tension.

Check that you have not made the rectum too narrow. You should be able to insert 1 finger comfortably.

To close the vaginal skin, use a single layer of continuous absorbable sutures.

To close the *levator ani* muscles (25-15P), take deep bites with the needle each side, so as to take a good hold of the muscles and the fascia covering both their surfaces. These thick sheets of muscle and fascia lie deep on each side of the rectum. Begin at the anal end and approximate them (21-15P). Suture the anal skin with a few interrupted intracuticular absorbable sutures, doing the same with the perineal skin.

CAUTION! Don't close the skin and vaginal wall too tight; leave room for drainage, in case there is infection or oozing.

POST-OPERATIVELY, encourage sitz baths twice daily, mobilization and provide some laxatives. *Don't use an enema*: rough use may destroy your handiwork!

If the tear is very soiled with faeces, delay surgical repair. In untreated HIV+ve patients especially, make sure you have emptied the rectum beforehand with laxatives and enemas, and have administered gentamycin and metronidazole for 3-5 days. In severe cases, establish a defunctioning colostomy first (11.5).

If the cervix is torn, it may have a single tear, large enough to need suturing, or numerous small tears. Bleeding from small tears is most easily controlled by packing (22.11). A large laceration will need repair (22-11).

N.B. Bleeding is more likely to be arising from a poorly contracted uterus, which needs oxytocin.

If there is a haematoma of the vulva, incise it at its lowest point, and evacuate the clot. Insert a drain, and suture this in position. If it bleeds severely, pack the cavity for 24h. These haematomas are usually unilateral, cause great pain, and occasionally retention of urine.

If the clitoris is torn, it may bleed severely. Put in a figure-of-8 non-absorbable suture.

21.16 Old 3rd degree tears

Delay in anatomical repair of a 3rd degree tear will result in scarring, distortion and stenosis. Make sure any woman with a deep perineal wound gets sitz baths at least bd. *Don't be tempted to rush in to repair before you can identify all the structures.* This may mean considerable, difficult, intricate dissection, and it will mean a good understanding of the normal anatomy. Sometimes patients have minimal symptoms; remember >20% of patients still have some incontinence after a successful repair. Operate if your means for effective referral are very limited. If there is a minor tear in the *levator ani*, the patient may only have mild incontinence with loose stools: *don't make a tolerable situation worse!* Consider that this region is always primarily infected. Reconstruction is hazardous in untreated HIV+ve patients. Make sure the rectum is cleaned out with laxatives and enemas 2-3 days pre-operatively.

REPAIR OF AN OLD 3RD DEGREE TEAR (GRADE 2.4)

METHOD
Position the patient as before (21.15). Cut round the exposed mucous membrane for the full thickness of the vaginal skin. Apply tissue forceps, and use scissors to separate the vaginal wall from the rectum gently (21-15C). While you exert gentle tension on the vaginal wall, dissect laterally and free the rectum anteriorly and on both sides (21-15D). Apply clamps to the cut edges of the vaginal skin, and hold them downwards. Extend the dissection upwards in the plane of cleavage between the rectum and the vagina, holding your scissors against the posterior vaginal wall (21-15E).
Incise the vaginal wall in the midline (21-15F), to expose the rectum (21-15G). Hold the rectum medially, and use the handle of your scalpel to extend the plane of cleavage between the vaginal flap and the rectal wall (21-15H).
If you are able to mobilize the rectum, you will be able to close it without tension. Trim the remaining scar tissue from the edge of the rectal mucosa (21-15I). Hold the upper edge of the torn rectum in tissue forceps, and invert its mucosa with a row of fine atraumatic long-acting absorbable sutures (21-15J). Continue these until you reach the muco-cutaneous margin of the anal opening, so as to refashion a normal anus. Reinforce and bury the 1st layer of sutures with a 2nd layer (21-15K). This will reduce the size of the rectum, but only temporarily.
Search for the retracted ends of the *sphincter ani* muscles, which you will find buried in dimples at either side of the anus. This is essential, because *if you only freshen up the margins, you will not achieve continence*. Use hooks (21-15L), or baby Babcock forceps. Bring the hooks together to see if you have secured the sphincter (21-15M,N) and approximate them with at least 3 long-acting absorbable sutures.
Close the *levator ani* muscles with long-acting absorbable (21-15O,P) starting distally. Excise any excess tissue on the flaps of the vagina (21-15Q), *taking care not to remove too much*. Bring the raw edges of the vaginal wall together with interrupted absorbable sutures (21-15R,S).
Hold each one until the next is in position, and then cut it. When you have closed the vagina, close the perineal skin. The last 2-3 sutures should complete the formation of the anus, so that anal skin folds (*rugae*, 21-15T) radiate from it like the spokes of a wheel. If they don't, you have not done the operation as you should.
Manage the patient post-operatively as above (21.15).

21.17 Uterine rupture

The uterus can rupture before or during delivery, especially
(1) in multipara,
(2) after previous Caesarean section, especially with a vertical incision, and
(3) when oxytocin is used, or
(4) when version has been performed.
In only c.⅔ of cases do you make the diagnosis before delivery. In the rest you make it afterwards, usually after some difficult obstetric manoeuvre, such as a retained placenta (22.11), a destructive operation (21.8), or after a trial of scar (21.14). Here we are mostly concerned with rupture of the uterus before delivery, as a complication of obstructed labour.

If a woman, particularly a multipara, arrives late in obstructed labour, or you don't make this diagnosis, the uterus is likely to rupture. *This is a great obstetric disaster*. If primary care is really poor in your district, 50% of the women referred to you may need an operative delivery, and of these 5% may end up with a uterine rupture.
The usual story (21.11) is that a woman is brought from a rural setting in obstructed labour, having waited a long time for transport to hospital. She is often sufficiently clear-headed to be able to tell you that she had strong frequent pains which stopped suddenly.
When the uterus ruptures, it may do so intra-peritoneally (complete rupture), or extra-peritoneally (incomplete rupture, less common) when the foetus remains out of direct contact with the intestines.

If the membranes have ruptured some time before delivery, the contents of the uterus will become infected, and the uterine muscle bruised and in poor condition for repair.

N.B. Never try to deliver a woman with a ruptured uterus vaginally. Your aim is to:
(1) Resuscitate the patient and operate soon.
(2) Remove the baby and the placenta.
(3) Control bleeding.
(4) Repair or remove the uterus on the indications given below. Unless the rupture is extensive, and the tissues are particularly bruised and oedematous, repairing the uterus is likely to be easier than removing it, because distortion of the anatomy makes hysterectomy difficult. But even repair is not easy, because the edges of the tear will be ragged and not easy to bring together.
Hysterectomy takes longer than repair, and causes more bleeding. A subtotal, which leaves the cervix and perhaps part of the lower segment, is easier than a total hysterectomy; it causes less bleeding, and there is less danger to the ureters. If you have to remove the uterus, try to leave at least one ovary behind.

The secret of success is to exert continued compression on the uterus (22.12), to place a tourniquet around the cervix or base of the uterus (22.11), and to identify important structures and landmarks before you start to cut or suture them.

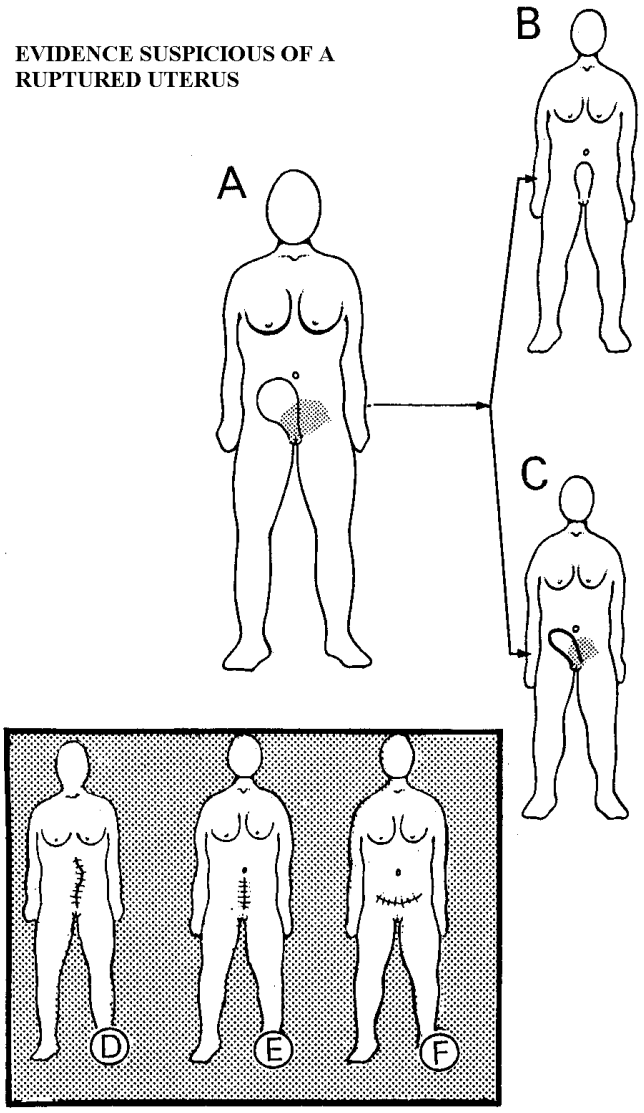


Fig. 21-16 EVIDENCE SUSPICIOUS OF UTERINE RUPTURE.
If a postpartum patient has a mass connected to the uterus (A), which does not disappear on catheterizing the bladder (B), but persists (C), it is probably a haematoma of the broad ligament due to rupture of the uterus. If a previous Caesarean section has left scar D, suspect strongly that it was the midline classical type. Scar E might be either. F is almost certainly a lower segment scar.
After Nasah BT, Drouin P. Review of 70 cases of ruptured uterus in Cameroun. Tropical Doctor 1978;8(3):127-31 with kind permission.

Speed is critical. Most time is lost getting the patient to theatre and in getting it ready, so make sure that it is *always* ready. If you are not familiar with the anatomy, study 21-18, 23-20,21! Be aware of impending rupture when labour is obstructed, especially in multipara, and try to prevent it by rapid intervention.

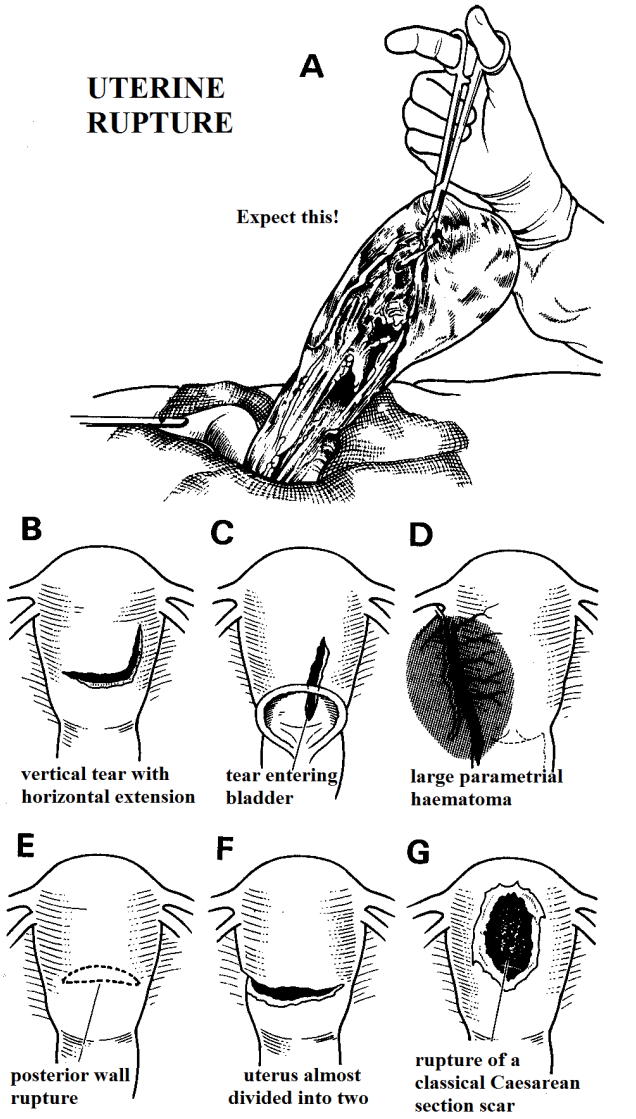


Fig. 21-17 UTERINE RUPTURE.
A, may look like this. B, a tear in the anterior wall with a vertical extension at one end. C, a tear involving the bladder. D, a tear in the lateral wall opening into the broad ligament. E, a transverse tear in the posterior wall. F, a tear which almost detaches the uterus. G, a tear from a classical vertical Caesarean section.

(a) Signs of impending rupture:
(1) The Bandl's ring between the upper and lower segments rises towards the umbilicus.
(2) The lower segment becomes stretched and painful to touch, even between contractions, which increase in strength and duration.
(3) The patient becomes anxious and restless with a tachycardia >100 and irregular respiration.

(b) Signs of actual rupture:
(1) Uterine contractions stop suddenly and are replaced commonly by no pain, less pain, or else, more rarely, by severe continuous pain.
(2) Shock and pallor without immediate response to blood transfusion (especially if the placenta is retained).
(3) Vaginal bleeding, usually quite severe, but not necessarily so.

N.B. **If the presenting part is jammed in the pelvis**, no blood can escape from the vagina. Perform an immediate ultrasound to see if the patient has a haemoperitoneum, or aspirate at the sides of the uterus.
(4) A tender uterus to palpation (it may feel soft, or be permanently tense), especially vaginally. Later, the entire abdomen may be tender.
(5) The foetus is usually abnormally difficult to feel, but may be abnormally easy to palpate! Sometimes, the shape of the uterus changes, and you may be able to feel the foetus outside it (usually the limbs are close under the abdominal wall, a certain sign of rupture).
(6) The foetal head which was previously low in the pelvis, has now risen higher and may now no longer be palpable vaginally.
(7) Blood-stained urine.
(8) An absent foetal heartbeat usually, unless the rupture is early and small.
(9) Rarely, the appearance of the placenta at the vulva before delivery.
(10) Rarely, prolapse of loops of bowel into the vagina.

N.B. Shock or severe vaginal bleeding may dominate the picture. The patient is usually lucid, and may even be talkative, which may delude you into thinking she is less critical than she really is. Dramatic symptoms of rupture are uncommon *but death within a very short time is not*.

(c) Differential diagnosis
Suggesting placental abruption: the cervix is closed or nearly so.

N.B. Beware making this diagnosis in a patient with a previous Caesarean section scar. The cervix may still be closed in rupture of a vertical Caesarean scar, or a corneal ectopic gestation.

Suggesting septicaemia: purulent discharge or other signs of infection, which may be subtle in the presence of HIV.

Suggesting severe dehydration: reduced skin elasticity, extreme thirst, dry mouth, no urine output.

(d) Management
Resuscitate the patient vigorously in theatre or the labour ward. Instil at least 1L normal saline or Ringer's lactate before anaesthesia starts. *Don't delay*; adequate resuscitation is impossible if bleeding continues internally. Continue the resuscitation while you operate.
Start 2 IV infusions: one for saline or Ringer's lactate run fast, and the other for blood. Prepare for an emergency laparotomy (11.1). Administer IV antibiotics.
Use the lithotomy position. Wash the abdomen, and introduce a urinary catheter: this will prevent you mistakenly opening a high full bladder.

If there is obstructed labour, and you are still not sure if there is uterine rupture or not, perform a vaginal examination. The presenting part may have disengaged, so that your hand passes through the rupture into the abdominal cavity, allowing you to feel the inner surface of the abdominal wall. You may find that the presenting part is unexpectedly easy to dislodge from the vagina, and there is then a gush of blood flowing out.

If the presenting part is not easy to dislodge, try pushing it up a little vaginally. If this fails, stop for fear of damaging the urethra. Pass your fingers anterior to the presenting part, into the uterus, and feel for a rupture. If there is one, you will feel the inner surface of the abdominal wall. If you are convinced there is no rupture, proceed to vaginal delivery if the foetus has died (21.8).

If the patient is sufficiently conscious to understand, explain that you would like to tie the tubes. If she is not fit enough to understand, speak to the relatives. It is seldom necessary to tie tubes without permission. As a general rule, no woman who has had a ruptured uterus should ever become pregnant again. The only exception is an extraperitoneal (partial) rupture through a lower segment scar.

CAUTION! Don't try to deliver the foetus before starting resuscitation, and beware of using any relaxational anaesthetic because this will remove the tamponade effect of the abdominal wall muscles, increase shock, and perhaps extend the uterine tear.

EXPLORATION
Make a low midline incision (11.2), and insert a self-retaining retractor. You may find a lot of bleeding, and an obvious uterine rupture with, commonly, a dead foetus. The placenta may be lying free in the abdomen, but if it is still attached to the uterus, the foetus may still, albeit rarely, be alive, even if lying free in the abdominal cavity. A detached placenta means foetal death, wherever it is.

If the foetus is lying free in the peritoneal cavity, uterine rupture is complete. Extract the foetus and check if it is still alive.

If the foetus is in the broad ligament, divide it and extract the foetus.

If the foetus is still in the uterus, as with a posterior rupture, deliver it through a transverse incision in the lower segment, as for a standard Caesarean section.

At delivery, administer oxytocin. Suck out blood, meconium and liquor from the abdomen. Lower the head of the table and pack off the bowels. Deliver the empty uterus into the wound and inspect it, especially its posterior wall: there may be another tear. Find the edges of the tear along its whole length. Divide the round ligament (21-18) if this makes the tear easier to see. The rupture may:
(1) be in the anterior wall of the uterus, often with a vertical extension at one end, making it L-shaped (21-17B).
(2) extend into the bladder (21-17C).
(3) extend longitudinally, along the lateral wall of the lower segment, from the fundus to the vagina, opening up the broad ligament and involving a uterine artery (21-17D), especially on the left.
(4) extend, rarely, transversely across the posterior wall of the uterus (21-17E).
(5) detach the uterus almost completely (21-17F).
(6) keep to the upper segment through the scar of an old classical vertical Caesarean section (21-17G).
(7) have torn one of the uterine pedicles right across.

Feel for the placenta and detach it from the uterus with your fingers. Try to get a tourniquet (a taut Foley catheter is the most readily available) around the base of the uterus, close to the cervix (22.11). Control bleeding from the uterus with #2 long-acting absorbable. Or, clamp the edges of the tear with several pairs of Green-Armytage forceps. Control bleeding from the broad ligament temporarily with pressure from a pack. If there is an extensive haematoma tracking up from the torn vessels on one side towards the kidney, evacuate the haematoma and ligate the vessels.

REPAIR OF A RUPTURED UTERUS (GRADE 3.1)

INDICATIONS
(1) A rupture which is not too large.
(2) A rupture with clean edges which are easy to see and are not too oedematous.
(3) Little or no infection.
(4) Relative inexperience.

CONTRAINDICATIONS
(1) Extensive or multiple tears.
(2) Edges which are very bruised and oedematous and not easy to define, especially a posterior rupture, or rupture extending down into the vagina.
(3) Gross uterine infection.
(4) Uncontrollable haemorrhage.
In these circumstances, a hysterectomy is preferable.

Start by defining the position of the uterine pedicles, the ovarian pedicles, and the round ligaments (21-18).

If the tear extends into the cervix or lower segment, reflect the bladder as for a lower segment Caesarean section. *Avoid the ureters*. Ask your assistant to pull the uterus forwards and to the opposite side. Lift the tube and ovary, to make the infundibulo-pelvic ligament, which carries the ovarian vessels, taut. Put your thumb and index finger on either side of this ligament, and slide them down.

Feel for the ureter as a hard round cord near the pelvic brim. From there trace it down to the injured area (23-20). Remove all clot. If there is a little bleeding, disregard it. If there is much, apply haemostats or transfixion sutures.

CAUTION!
(1) Be sure to keep the bladder well away from the edges of the tear.
(2) *Don't excise any tissue unless it is obviously dead*.

Start at the apex of the rupture; if convenient, hold it with a stay suture. Suture it as for Caesarean section, using 2 layers of continuous long-acting absorbable in a large (#2 or #3) half-circle round-bodied needle. You can suture a vertical tear going down to the cervix from below upwards, but sometimes the other way round is easier. Traction on the suture will help to bring the lower end into view. Don't worry if the inner layer has to be placed inside the uterus. Make the second layer an inverting continuous suture. If necessary, use extra sutures to close off the corners, or repair the vagina, usually anteriorly.

If the rupture is lateral and has extended into the broad ligament, open its peritoneal roof, and ligate the bleeding vessels. Control any oozing not stopped by compression with under-running sutures. *Avoid the ureter*. With one finger inside the broad ligament and another behind it, feel for the ureter; if necessary, pass a cloth tape under it to keep it out of the way. Start at the apex of the rupture and work downwards. Exert traction on the running suture to expose the depths of the tear. Stop before you reach the lower edge, so as to leave room for a drain from the broad ligament into the vagina. If there is much oozing, pack the broad ligament with a gauze bandage, bring it out of the vagina, and close the visceral peritoneum over it. Remove the pack 12h later.

N.B. Don't forget to perform a tubal ligation (unless you have repaired a lower segment rupture, and the patient wishes for more children).

PARTIAL HYSTERECTOMY FOR A RUPTURED UTERUS (GRADE 3.4)
Hysterectomy may be surprisingly easy when the tear is extensive and transverse, and the uterus almost completely detached.

INDICATIONS
(1) Complicated rupture of the uterus.
(2) Postpartum haemorrhage, which is not responding to treatment, and when a B-Lynch suture (22-13) or tying uterine arteries has failed to control bleeding.

METHOD
This is modified somewhat from elective hysterectomy (23.15).
Having delivered the uterus from the abdomen, maintain traction on it with one hand, or insert a traction suture.
Start by identifying: (1) the uterus and round ligaments, (2) the tubes and ovaries on both sides, (3) the infundibulo-pelvic ligaments (21-18), (4) the avascular area in each of the broad ligaments, (5) the lower segment, (6) the rectum, and especially (7) the ureters (23-21). You will find this difficult, because of the size of the uterus, and the disturbance to the normal anatomy caused by bruising and oedema, both near the tear and far from it.
Deflect the bladder, and trace the ureters over the whole length of the operative field (23-22G). Find where they are in relation to the tear, in the distal part of their course. Find the tear and clamp the obvious bleeding points. Pull the uterus to the left, and divide the right round ligament between clamps about 2cm from it. This will open the anterior peritoneal leaf of the broad ligament. Enlarge this opening down towards the bladder. Lift the right tube and ovary with one hand, and push a finger of your other hand from behind through the avascular area in the broad ligament.

CAUTION! Leave the ovary and tube in place on one or both sides.
On the side on which you will remove the ovary, clamp the infundibulo-pelvic ligament between two artery forceps and divide it. On the other side, to retain the tube and ovary, clamp and divide the tube and the ovarian ligament near the uterus. If they are very thick and vascular, you may have to clamp and divide them in 2 steps.
Transfix the pedicles of the round ligaments and infundibulo-pelvic ligaments with #2 multifilament or absorbable.

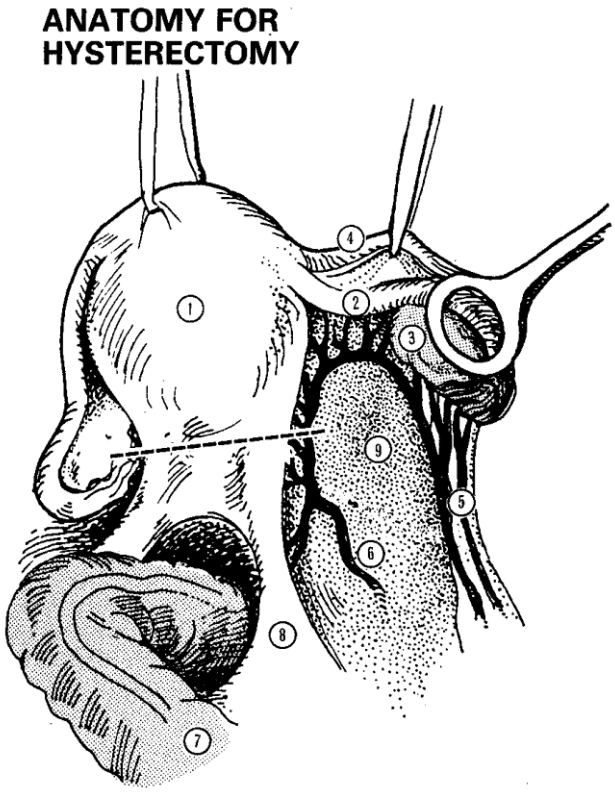


Fig. 21-18 ANATOMY FOR EMERGENCY HYSTERECTOMY. Posterior view of the unruptured uterus. It is tilted to the left and the adnexa (ovary and tube) have been lifted up to show them more clearly. (1) the body of the uterus. (2) the right Fallopian tube. (3) the ovary. (4) the round ligament. (5) the ovarian vessels running in the infundibulo-pelvic ligament which is being stretched. (6) the uterine vessels. (7) the rectum. (8) the sacrouterine ligament. (9) the avascular area in the broad ligament.
Kindly contributed by Frits Driessen.

Using the clamps that you have already applied, pull the uterus well up in the midline, and cut the peritoneum between the uterus and the bladder. Extend the incision laterally to meet the incisions you have made in the anterior leaves of the broad ligaments.

Push the bladder off the lower segment for 2-3cm with a swab on a holder. *Pushing it further down can cause bleeding*. If the rupture is anterior, put its edge on the stretch before you separate off the bladder.

Now expose the back of the lower segment by pulling the uterus forwards over the symphysis pubis. Divide the peritoneum over the back of the lower segment at the same level as you did anteriorly. Extend the incisions laterally to join the openings in the broad ligament. Push the lower peritoneum off the lower segment with a swab on a holder; or, if this is difficult, cut it loose with scissors.

On either side of the uterus there will now be a bundle of loose connective tissue containing the uterine vessels. If you need to see them more clearly, strip down the peritoneum off the broad ligaments. Pull the uterus to the right and clamp the uterine vessels with strong Kocher forceps, just above the level where the bladder is still attached to the lower segment.

CAUTION! Make sure the points of the forceps are close to the uterus or even a little in its wall.

Place a 2nd clamp inside the 1st, and cut the uterine vessels between them. Tie and transfix the pedicle. Use a double transfixion ligature because of its width, and then do the same thing on the other side.

Excise the uterus through its lower segment, just above the level of the cut uterine vessels. Have artery forceps ready to pick up the cut edge of the lower segment, before it disappears in the depth of the pelvis. Clamp any bleeding vessels.

If the tear extends across the lower segment, it will probably serve as the line of demarcation to remove the uterus. Examine the edge and remove any very oedematous and bruised tissue, again first checking the position of the ureters.

If there is a downward tear in the cervix, repair this now, after making sure that the bladder and ureters are well out of the way. Alternatively, perform a total hysterectomy, and remove the cervix.

Suture the anterior and posterior walls of the lower segment with figure-of-8 sutures, being sure to include the angles on each side, because these bleed. If there are signs of infection, leave the centre open so that you can insert a drain; otherwise close it. The pelvis should now be nearly dry. Tie any remaining bleeding vessels.

If the broad ligaments are oozing, apply compression and perhaps, if oozing persists, place a drain near them and bring it out through the vagina.

Close the pelvic peritoneum with a continuous suture. Start on the left at the pedicle of the infundibulo-pelvic ligament, and suture the anterior edge of the peritoneum to the posterior edge, placing all vascular pedicles under it. Let the remaining ovary and tube hang freely in the pelvis.

Clean and wash the peritoneum with at least 2L warm water. Close the abdominal wall (11.8).

(e) Difficulties with rupture of the uterus

If the bladder is torn, its wall near the opening is usually stuck to the lower segment, and needs mobilizing before you can repair it. You may find that the bladder is so torn that it lies flat like a handkerchief.

Use Allis forceps or Babcock clamps to stretch the wall of the bladder and the lower segment. Suck away the blood. Separate the bladder from the lower segment with a 'swab on a stick', or with scissors. Gently dissect it off the lower segment, taking care not to make the tear any bigger. Free the bladder wall round the opening for 1-2cm.

Close the opening in the bladder with 2 layers of 2/0 continuous long-acting absorbable. Put the first layer through the full thickness of the bladder wall, but just submucosal if possible. If this is difficult, include the mucosa. Use the 2nd layer to invert the 1st one. Insert an indwelling catheter and maintain open drainage for 10-14 days.

Unfortunately, complete closure of the bladder is often impossible; its edges are usually thin and necrotic, so that a fistula often follows. In this case, re-insert the catheter and wait 3 months.

If complete closure of the torn bladder is impossible, because there is much pressure necrosis, or the opening extends far down into the urethra, you may have to close the bladder over a wide-bore suprapubic tube. If a vesico-uterine fistula develops, arrange for its repair later.

If you think that you have caught the ureter in a suture, unpick it; usually there is no permanent harm.
It is better to be safe than sorry! A dye test (21.11) may help. Otherwise a nephrostomy will bail you out.
If there is anaemia after delivery with a boggy pelvic swelling and deviation of the uterus, there is probably a PELVIC HAEMATOMA. This is really a rupture of the uterus which has bled into the broad ligament instead of into the peritoneal cavity (21-16A,C). If the patient presents <24h after delivery, perform a laparotomy and explore and repair the tear.

21.18 Vesico-vaginal fistula (VVF)

Fistulae are usually the result of:
(1) obstructed labour in a young primigravida (21.4).
(2) unskilled Caesarean section.
(3) rupture of the uterus into the bladder, especially through the scar of a previous Caesarean section.
(4) traumatic vaginal delivery (especially with Kielland's forceps).
(5) vaginal trauma (by assault or FGM).

MECHANISM OF VESICO-VAGINAL FISTULA FORMATION

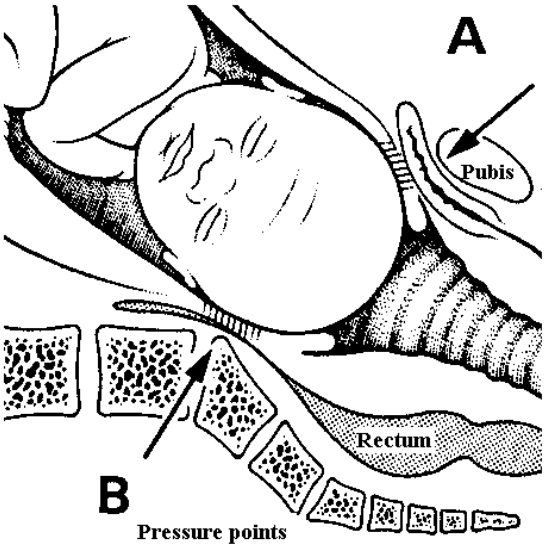


Fig. 21-19. THE MECHANISM OF FISTULA FORMATION DUE TO OBSTRUCTED LABOUR. A, pressure against the pubis causing necrosis of the vagina and posterior bladder wall. B, pressure against the sacrum causing necrosis of the vaginal and anterior rectal wall.

Fistulae result as the unrelieved obstructed foetal head impacts against the back of the pubis, especially in very young girls (<16yrs). The head cannot move further, resulting in pressure necrosis of the vaginal and adjacent bladder wall (21-19A). If the pressure of the head is exerted posteriorly (21-19B), the result may be a recto-vaginal fistula (RVF, 21.19).

A vesico-vaginal fistula (VVF) means uncontrollable urinary incontinence for the young woman, usually stillbirth, and often also infertility subsequently. After the catastrophe of losing her baby in childbirth, the young mother is often thrown out of the home because she cannot have sexual intercourse, is incontinent and smelly. She is rarely able to find a job, becomes depressed, lies curled up in the corner of a house where she can find lodging, develops contractures and pressure sores, and dies neglected.

There are estimated to be 3 million untreated VVF patients in Africa alone. In Ethiopia there are just under 10,000 new cases per year, and the Fistula Hospital in that country has gained a well-deserved worldwide reputation for the treatment of such fistulae. If you can get training there (or at another special centre), you will not regret it, but don't think that all VVF must be handled at a specialist centre: there are too many fistula victims for this to be feasible.

They have a reputation of being impossibly difficult to repair, but this is not so for at least 25% of cases. Certainly, though, your best chance of success is in the first attempt; so, don't perform this operation unless you are reasonably experienced, or have gone to a special training centre, preferably several times in between increasing your own experience. The important thing is to be able to recognize which is the easier fistula, and which the complicated. You are advised to leave the latter, and the second or subsequent repairs, to experts.

Even then, experts who tackle every type of case will only achieve 75% success, meaning total continence (*i.e.* absence of stress incontinence, which is often severe). Anyone who claims a 100% success rate either has not done many, has selected easier cases, or has not done a proper follow-up.

N.B. A *gracilis* flap is easy to learn and can help out in many difficulties: try to get an expert to show you how to do this!

Fistula surgery for easier cases does *not* need special instruments or equipment; good post-operative nursing care is, however, very important but *not* complicated and you can achieve success even when you have not done such surgery previously.

These cases are immensely rewarding, because you can transform the life of a young woman from existence as an outcast; indeed one of the most senior VVF surgeons in Addis Ababa started off as an outcast VVF patient!

(a) Site of the fistula

The area where pressure necrosis occurs will be where the fistula results. Most commonly this is the urethro-vesical junction, but other sites can occur together or individually: juxta-urethral, mid-vaginal, juxta-cervical and intra-cervical.

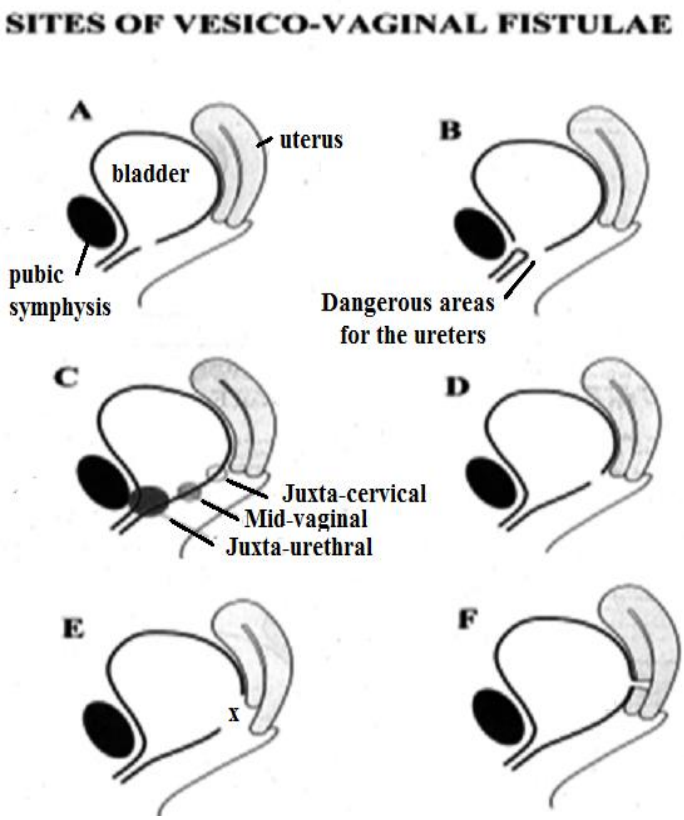


Fig. 21-20 SITES OF VESICO-VAGINAL FISTULAE.
A, simple juxta-urethral. B, extensive juxta-urethral with a gap between (stenosed) urethra and bladder. C, mid-vaginal. D, simple juxta-cervical. E, complex juxta-cervical involving the anterior cervix. F, intra-cervical. (x) is the site of the ureteric orifice.
After Hancock B, First Steps in Vesico-Vaginal Fistula Repair, RSM, London 2005, Figs 2-7 p.4-6.

The first is the most common: if ischaemia is mild, a small hole is produced (21-20A); if ischaemia is prolonged, the defect may be circumferential and result in separation of the urethra, which becomes closed off, from the bladder itself (21-20B). The mid-vaginal fistulae are less common but easy to repair, unless they extend to the cervix (21-20C). The juxta-cervical fistula (21-20D) usually follows a vertical tear in the lower segment at Caesarean section (21.10): the defect may include the anterior cervix, and encroach onto the ureteric orifice (21-20E). The intra-cervical fistula may be due to prolonged pushing against an incompletely dilated cervix, or due to iatrogenic injury at Caesarean section (21-20F).

(b) Features of the fistula
Apart from their position, take note of their **size**: tiny (admitting only a probe) to large (>3cm, usually involving most of the anterior vaginal wall and the complete vesico-urethral junction). Occasionally the fistula may be truly extensive, extending to the anterior bladder wall, and exposing the bare bone of the back of the pubis. The ureters may even be draining freely outside the bladder!

Take note of the amount of **scar tissue**: if you find extensive scarring, leave the case to an experienced surgeon.

Note the remaining **length of the urethra**: the shorter it is and the more scar tissue that needs to be removed, the bigger the risk of stress incontinence.

(c) Prophylaxis
At Caesarean section, take care to dissect the bladder well down off the lower segment prior to incising the uterus.
If the head is deeply impacted in the pelvis, get an assistant to push from below, rather than inserting your hand between the foetal head and the lower segment.

After Caesarean section, take care when suturing lower segment tears, and always ask yourself if you have not inadvertently picked up the bladder.
Leave a urinary catheter *in situ* at least 10 days after a Caesarean section performed for prolonged obstructed labour.

At follow-up, if urine is leaking, leave a Ch18 urinary catheter *in situ* for 4-6wks, making sure you inflate the balloon only with a maximum of 10ml, and the balloon is not inflated in the urethra. Let the urine drain freely. Recommend sitz baths to wash the perineum, a high-protein diet with haematinics, copious drinking of fluids to 5L/day, good mobilization, and abstinence from sexual intercourse for 3 months.

(d) History
(1) **Age and parity** (some patients still achieve pregnancy after developing a fistula after a Caesarean section).
(2) **Incontinence.** Check if the patient is wet all the time: if she is dry at night, a fistula is unlikely. Ask about faecal as well as urinary incontinence. They may be separate or combined.
(3) **Amenorrhoea.** Ask about menstruation: it often stops after traumatic childbirth, but may suggest a hysterectomy about which the patient was never informed.
(4) **Delivery.** Ask if labour was prolonged, delivery difficult and traumatic. Ask if it was at home, in a clinic or at hospital. Note whether a Caesarean section was performed, and if so, how many times.
(5) **Previous surgery.** Ask sympathetically if previous attempts at repair have been made: remember patients may try to hide this, fearing they may be turned away.
(6) **Neurological deficit.** Look especially for a foot drop, even if partially recovered.

(e) Examination
(1) **General.** Note malnutrition, state of hydration (many patients drink little in order to reduce their wetness), anaemia and the psychological state.
(2) **Dermatitis and pressure sores** (34.16)**.** Look for an obvious urinary leak and signs of dermatitis (discoloured darkened or reddened skin around the perineum) and examine pressure points. If you cannot see any wetness, ask the patient to drink plenty of water and re-examine her after 1h. Get the patient to cough if you still cannot visualize any wetness.
(3) **Urethral orifice.** Is it destroyed or stenosed?
(4) **Vagina.** Perform a gentle digital examination and then, if necessary, with a Sims' speculum. You may find it easier in theatre in the lithotomy position with a good light, but you *don't need a GA.* Is the vagina stenosed?

The lateral or posterior surfaces may feel fibrous, or the whole vagina narrowed. Is there a palpable defect anteriorly? If so, where is it in relation to the urethra and cervix? Are the margins of the fistula soft and supple, or firm, or even fixed to the pubic bone? Can you identify the cervix? Does it feel normal? Is the vagina shortened?
(5) **Ureter.** If you suspect ureteric damage after Caesarean section, ask the patient to empty her bladder and then insert a dry swab high in the vagina. Get her to drink water, walk about and then 30mins later, remove the swab, looking for wetness.
(6) **Ano-rectum.** Examine for anal sphincter function: this is often impaired after delivery. Look for evidence of urine flowing out of the anus.

(f) Dye test
Use *dilute* methylene blue or gentian violet: too concentrated dye will colour everything, making interpretation impossible. Introduce a urinary catheter and inflate the balloon. Place 3 moist swabs well up in the vagina; then insert 50ml slowly into the catheter. Remove the swabs gently one by one. A fistula is confirmed if the 2nd or 3rd but not the 1st swab is discoloured with dye.

If you don't see any swab stained blue, repeat the test with 200ml after leaving the dye 20mins in the bladder. If this 2nd test is -ve, suspect a uretero-vaginal fistula (see above). If the 3rd gauze is wet but not blue, suspect a ureteric fistula (21.11).

If, after you have removed the catheter, leaving the dye inside, the colour dribbles out, especially on coughing, this indicates stress incontinence, which you should treat with pelvic floor exercises, or 12wks of continuous catheter draining if there is an atonic bladder with overflow after delivery.

(g) Selection of cases
Select a straightforward case for your first attempts at repair; you will get a good reputation through success, so *don't attempt any difficult cases until you have considerable experience.* You will do your patients and your reputation no good by attempting difficult cases and failing. *Don't waste time with futile investigations and treatment:* if you cannot treat a patient, refer her early.

(h) Contraindications
Don't attempt:
(1) Complex fistulae, including those you have difficulty visualizing, and recto-vaginal fistulae, except in special circumstances (21.19).
(2) Cases where the urethra is completely detached.
(3) Cases where there is considerable scarring.
(4) Surgery in grossly malnourished, anaemic or untreated HIV+ve patients.
(5) A case where you are uncertain how to proceed.

(i) Preparation
Explain clearly what the patient is to expect, especially the length of time she will be in hospital and how long she may need a urinary catheter, and how long she may need to abstain from sexual intercourse.

Make sure the patient is well hydrated from the moment you take the decision to operate. She should be drinking 6-8L/day before operation. Make sure she gets a high-protein diet, iron and folate. Stop fluids only 4h before anaesthesia. *You should see urine dripping when she stands with legs apart.*
There is no indication for pre-operative antibiotics.
Make sure the rectum is empty by getting the patient to go to the toilet just before theatre, or administering an enema the evening before.

N.B. It is no good doing this just before theatre because the perineum will be contaminated.

EQUIPMENT
No special equipment is necessary: you will need a self-retaining weighted Auvard speculum, good-quality dissecting scissors, toothed dissecting forceps, Allis tissue forceps, artery forceps, a metal catheter, a small probe, towel clips and a #15 blade.
Sharply curved Thorek scissors, a sharp Deschamps aneurysm needle and long vaginal instruments are a great help.
Long-acting absorbable sutures are ideal: *don't use non-absorbable.* Half-circle 25mm round-bodied needles are best, though J-shaped needles are very useful in more difficult cases.
You will need an operating table which tilts and has shoulder rests for more complex cases. A headlight is very useful; if this is missing, turn the table to face the bright sunshine!

(j) Method of VVF repair (GRADE 3.3)
Place the patient, with the buttocks well over the end of the table, in the exaggerated lithotomy position with legs flexed and slightly abducted. Make sure *your* position is comfortable, that your seat is not too high or the operating table too low.

Suture the labia to the thighs, and cover the anus with a swab (21-21A), or insert a temporary purse-string suture to close it if faecal leakage is troublesome.

Record the size and position of the fistula. Estimate the distance from the external urethral orifice to the distal fistula margin (ideally 3-4cm), and that from the proximal fistula margin to the cervix (ideally >4cm) (21-21B). The nearer the fistula is to the cervix, the greater is the danger to the ureters.

Only operate juxta-cervical fistulae in multiparous women where you can easily pull the cervix downwards.

Pass artery forceps or a metal catheter through the urethra, holding it to point forwards to expose the fistula clearly (21-21C). Check if the urethra is detached from the bladder: in this case, leave it for an expert.

Administer gentamicin 160mg IV plus metronidazole 500mg if there has been faecal contamination. Check with ultrasound if there are stones within the bladder, and estimate the size.

Infiltrate the tissues between the vagina and bladder with dilute (1:200,000) adrenaline solution (21-21D).

Whilst steadying the anterior vaginal wall with the forceps passed through the urethral orifice, lift up the mucosa over the urethra with an Allis forceps and incise in the posterior margin of the fistula through the vagina (21-21E).

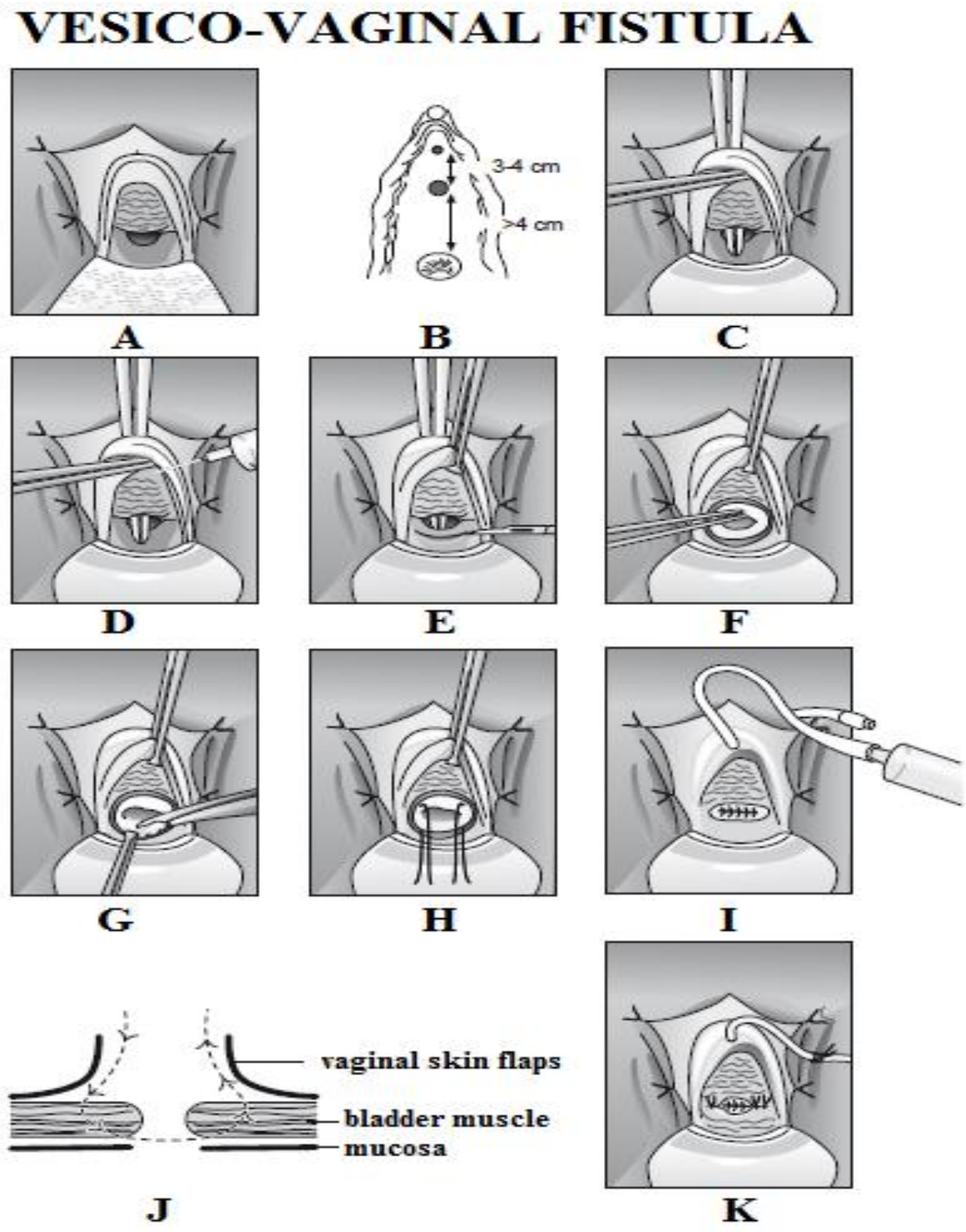


Fig. 21-21 SIMPLE VESICO-VAGINAL FISTULA REPAIR.
A, suture the labia to the thighs and cover the anus. B, record the size of the fistula and its distance from the external urethral orifice and cervix. C, insert artery forceps through the urethral orifice to expose the fistula. D, infiltrate around the fistula orifice with 1:200,000 adrenaline solution. E, steady the anterior vaginal wall with the forceps passed through the urethra and lift up the mucosa around the urethra with Allis forceps. Identify the correct plane between vagina and bladder, and mobilize the posterior margin, keeping at least 1cm from the fistula orifice. F, remove the artery forceps inside the urethra. Start the anterior dissection with a small incision vertically, and continue laterally. G, trim away any vaginal mucosa and scar tissue at the fistula margin. H, insert 2 corner sutures through the freshened margins of the fistula. I, complete the closure with 3-4 sutures, c.4mm apart, taking big bites of the full thickness of the bladder muscle, excluding the mucosa. Then insert a catheter in the urethra and introduce 50ml of dilute methylene blue dye. J, if there is no leak, close the vagina with interrupted everting mattress sutures. K, then fix the catheter to the labium, and pack the vagina with a betadinized swab.
After Hancock B, First Steps in Vesico-Vaginal Fistula Repair, RSM, London 2005, Figs 31,32,33a,c-h,k,o,q,t. p.25-28.

Identify the correct plane between vagina and bladder and continue to dissect round to the sides so that you mobilize at least 1cm beyond the fistula hole (21-21F). Start the anterior dissection with a little extension vertically towards the urethra and complete it right round; then tie the right and left antero-lateral flaps to the labia to improve the exposure of the fistula. *Don't use diathermy*. Trim away with scissors any vaginal mucosa and scar tissue (this should be minimal) at the fistula margin (21-21G).

Now you have freshened up and exposed the margins of the fistula, you can start closure from the corners (21-21H). Insert 2 sutures and complete the repair with interrupted big bites taking in the full thickness of the bladder muscle, but not the bladder mucosa (21-21I,J). Remove the forceps in the urethra and insert a catheter, and perform a dye test (21-21K) with 50ml of dilute solution. Press over the abdomen or ask the patient to cough to see if there is any discolouration. If there is a leak through the suture line, inset another one or two sutures, and re-test. Exclude the presence of a 2nd, unsuspected, fistula higher up.

Finally, close the vagina with interrupted everting mattress sutures, and fix the catheter *in situ* with a suture to the labium. Place a betadinized pack gently in the vagina.

(k) Difficulties with simple VVF repair

If exposure is poor, perform an episiotomy, on both sides, if necessary (and remember to repair them at the end of the operation).

If there is necrotic sloughy tissue, debride this adequately and review the situation when all the tissues are clean and fresh. This is likely, however, to become a complex fistula.

If the urethra is stenosed, try to dilate it with sounds. Remember you will need more generous exposure of the fistula, and need to excise scar tissue. You may find the stenosis recurs and needs regular dilation, so keep a careful follow-up.

If you find bladder stones, unless the stone is small, or comes out easily through the fistula, abandon the VVF repair, and proceed to cystolithotomy (27.16). Schedule the VVF repair at least 2wks later.

If you suspect the ureter to be damaged, you are likely to be dealing with a juxta-cervical fistula, which is a matter for an expert.

If your operation is taking >1h, you are either tackling too complex a fistula, or there is some difficulty with your technique. Consult this manual and get help.

(l) Post-operative care

You may have performed marvellous surgery, only for it to be ruined by poor nursing care; so make sure, before you embark on VVF repairs, that you involve all your nursing staff and explain what is needed and why. Keep a simple record of patients on their beds: *measuring urine output is unnecessary.*

(1) **The aim is that the patient is drinking freely, draining urine freely and free to mobilize without being wet.** *The catheter must never block*: if this happens, urine will emerge alongside the tube or even leak through your well-sutured repair and re-create the fistula. The problem about drainage bags is that they can fill up (quickly if the patient is drinking well) and fall on the floor, or cause traction on the catheter when the patient turns in bed, or overfill and cause back-pressure, or twist and become blocked. The easiest solution is connecting the catheter to a straight plastic tube that drains freely into a basin or bucket: this has the advantage that you can readily see if urine is dripping freely from the tube.

If the catheter blocks, *i.e. you no longer see urine dripping from the tube, and the patient is wet or has a full bladder*, act immediately: untwist the catheter, unkink the tubing (or replace it, if it is full of debris), gently irrigate the catheter with a bladder syringe. If this does not make urine flow, replace the catheter.

Urine will not flow if the patient is not drinking! So make sure she drinks 5L/day, because concentrated urine flows poorly and is susceptible to becoming infected. The urine should be almost colourless.

If the patient is wet, exclude a blocked catheter. Check if urine is leaking alongside the tube during bladder irrigation: this may suggest urethral dysfunction. Perform a dye test to check your repair or look for a second (missed) fistula.

(2) **Wash the perineum twice daily**, especially where the catheter emerges from the urethra.

(3) **Remove the vaginal pack** after 48h.

(4) **Mobilize the patient**, carrying a bucket to drain her urine.

(5) **Remove the catheter** after 12-14 days after you have confirmed that a dye test shows no leak. Encourage the patient to pass urine every 2h and then increase the interval gradually as bladder tone recovers. *Don't clamp and unclamp the catheter:* this all too frequently leads to disaster!

(6) **Before discharge:** wait 48h after removing the catheter. Check if the patient is dry; if not, perform a dye test. If it is +ve, all is not lost, though an early leak suggests a worse prognosis. Keep the catheter *in situ* a further 4wks if more urine drains through the catheter than the vagina. Monitor to see if the leak is reducing. Lying in the prone position allows the catheter tip to rest free from the fistula.

(7) **General advice.** Insist on abstinence from sexual intercourse for 3 months. Advise on tubal ligation or contraceptives. Further delivery must be by Caesarean section. Recommend a high fluid intake to prevent infection and development of urinary stones. Persuade patients to come for regular follow-up so you can check whether a late leak or urethral stenosis develops, or stress incontinence persists, and you can do an audit of your activity. In these latter cases, try to get help from an expert.

21.19 Recto-vaginal fistula (RVF)

Fistulae between the rectum and the vagina (RVF) are less common than those between the bladder and the vagina (VVF). If there is a large VVF, there may also be an RVF, also when there is pressure exerted posteriorly (21-19B) on account of unrelieved obstructed labour. An RVF may occur, rarely, without a VVF.

The diagnosis is obvious: faeces start to leak through the vagina.

Sometimes, especially in babies but also in adults, an RVF may occur spontaneously due to HIV disease (26.2). It may also occur as a result of syphilis, lymphogranuloma venereum and other venereal diseases; and also due to carcinoma, itself HIV-related.

To distinguish an RVF from a 3rd degree vaginal tear, clean away the faeces, and look at the perineum. If the site of the fistula is not obvious on inspection, digital palpation or proctoscopy, proceed to sigmoidoscopy. You might need to use ketamine to do this, remembering to position the patient *before* administering the drug. Note the position of the fistula, the degree of inflammation present, and its size.

Repair of an RVF is *not for the beginner*. Chances of success are better early rather than late, providing the initial inflammation has settled, and they are significantly improved if you can divert the faecal stream beforehand.

This will mean performing a defunctioning loop colostomy (11.6), which the patient may not tolerate well, and may think more debilitating than the RVF itself: so be cautious before inflicting this on a patient and make sure you explain carefully what it entails. It is only really worthwhile if you have arranged RVF repair beforehand, and you can fashion the colostomy as a staged procedure.

It is possible to treat low fistulae successfully with a seton (26.3), but be prepared for it to take a long time till healing results.

22 Other obstetric problems

22.1 Testing for foetal maturity

You can usually obtain the length of gestation from a pelvic examination early in pregnancy, or by an ultrasound scan (38.2,3).

If there is a clinical discrepancy or you have serious reasons to doubt your measurements, the surfactant test is a simple way of estimating the maturity of a foetus. However, if there are developments that suggest to you that the foetus in question will be born or delivered >28wks and <34wks, 2 doses 24h apart of betamethasone 12mg IM to the mother, within 1wk of delivery, is of proven benefit in preventing severe respiratory problems and hence cerebral problems. *N.B. If the mother is a diabetic on insulin, monitor the glucose and increase the dose accordingly.*

If delivery threatens again >1wk after the last injection and gestation is <34wks, you can repeat the treatment *once* more.

There might be situations where the mother is not much at risk but the foetus is (*e.g.* in not-well-controlled diabetes); then it might be useful to do a surfactant test. Unfortunately, the test is least reliable in cases of diabetes. There might be occasionally a reason to do the test if there is intra-uterine growth retardation (IUGR) without high blood pressure and/or proteinuria suggesting that the mother is also at risk.

N.B. **If there might be a chance the patient is HIV+ve**, then the test is dangerous both for the doctor (patients are known suddenly to push away your hands with the syringe if you penetrate the abdomen and you might get injured) and for the foetus, because:

(1) the needle with maternal blood in it might prick the foetus,

(2) the barrier between the 2 circulations in the placenta might become damaged if you hit the placenta,

(3) the foetal surface becomes exposed to maternal blood after her blood has contaminated the liquor, or

(4) the procedure ends in prolonged rupture of membranes.

The test is not infallible, *so don't rely on it alone*; use it in conjunction with an estimate of gestation by dates, and an estimate of the foetal size. It is a test for the surfactant which foetal alveolar cells secrete, and which is necessary for the expansion of the foetal lungs immediately after birth. If they don't expand, respiratory distress syndrome will ensue, so the test is a measure of the extent to which this risk exists.

The test normally becomes +ve at 36wks, so it is a good sign that the foetus is mature enough to ripen the cervix and induce labour. Obtaining amniotic fluid is easy and safe providing both foetus and mother are hepatitis B, C and HIV-ve; it is no more painful than an intramuscular injection.

(a) The surfactant test

Rare complications include rupture of the membranes and injury to the foetal head. If the mother is Rhesus-ve, putting a needle through the placenta increases the risk of rhesus immunization.

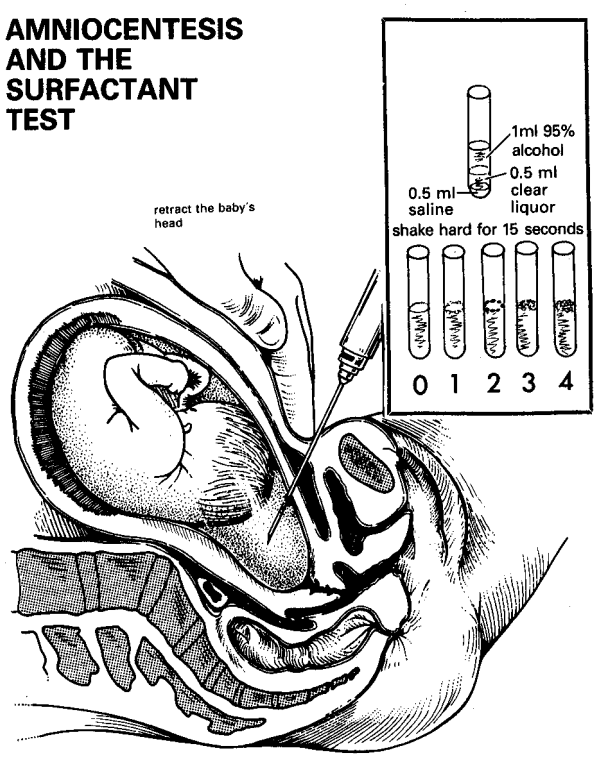

AMNIOCENTESIS AND THE SURFACTANT TEST
1ml 95% alcohol
0.5 ml clear liquor
0.5 ml saline
shake hard for 15 seconds
retract the baby's head
0 1 2 3 4

Fig. 22-1 THE SURFACTANT TEST normally becomes +ve at 36wks, so it is a good sign that a foetus is mature enough to deliver.

INDICATIONS

N.B. There should be a legitimate reason for induction, *but not if you would induce the patient anyway, such as in severe gestational hypertension*.

(1) An elective Caesarean section with uncertain dates.

(2) Suspected growth retardation (22.13).

You should be able to use ultrasound to localize the placenta, and you should be sure that the mother has a mobile presenting part, showing that she has enough liquor to aspirate. If there is not enough liquor, the foetus is probably mature enough anyway.

ASPIRATION

Take a sterile 10ml syringe and a 10cm long, 1mm diameter needle. You may need a longer one if the mother is obese. Have a 2nd syringe ready in case the 1st sample is blood-stained. Make sure the bladder is empty, so that you don't aspirate urine. There is no need for LA. Prepare the skin over the lower abdomen, preferably with iodine.

In the supine position, the lowest part of the foetus is usually the head: feel it, lift it up out of the pelvis as far as you can, and then hold it there with your left hand. This will allow liquor to swirl around it, and fill the lower segment.

If possible, use ultrasound as a guide. While retracting the foetal head upwards, plunge the needle attached to the syringe into the uterus at right angles to the plane of the lower segment, as near to the head as is reasonable, remembering that you don't want to hit it.

Remember also that the commonest complication is rupture of the membranes due to inserting the needle too low, too close to the cervix.
Alternatively, aspirate at the level of the umbilicus on the side of the foetal limbs. You need to be able to feel the position clearly. There is usually a good pool of liquor there. Injuring the foetus is very unusual.
Withdraw 5-10ml of fluid. Record it as being clear or blood-stained (indicating a traumatic tap), and the vernix, the white cheesy substance that covers a neonate's skin, as being absent, scanty, or plentiful. If you see vernix easily, this is in itself more or less proof of foetal maturity.
If, at an ultrasound scan (38.3)**, you see vernix floating clearly**, a shake test is not needed because the foetus is mature enough. *Don't try the procedure more than twice.*

EQUIPMENT. You need:
(1) 1ml of clear liquor, uncontaminated by meconium or blood. Only the faintest blood-staining is acceptable. If you cannot avoid blood contamination, centrifuge the liquor hard for 5mins and test the supernatant.
(2) 95% alcohol.
(3) Some completely clean glass test-tubes with an internal diameter 8-14mm.
(4) 'Parafilm' to cover the tubes. Otherwise, use new corks or rubber stoppers. If you don't have these either, a very carefully washed, and even more carefully rinsed, gloved finger is probably better than a used cork or stopper.

METHOD. Take exactly 0·5ml liquor, 0·5ml saline, and 1ml 95% alcohol. Shake the mixture vigorously for exactly 15secs. Then don't move the tube. Wait 15mins before examining it in a good light against a dark background (22-1).

0	No foam
1	An incomplete ring of bubbles peripherally round the meniscus
2	A complete ring of bubbles round the meniscus
3	As above, but foam just covering the whole meniscus
4	Plentiful foam covering the whole meniscus thickly

A score of ≥1 means that the lungs are mature.

CAUTION!
(1) *Avoid contamination with anything greasy*.
(2) Meconium contaminates and produces a false +ve result, so *don't do the test if there is meconium in the fluid*.
(3) *Don't shake the tubes a second time.*

(b) Difficulties with the surfactant test
If you aspirate nothing, has your needle entered the amniotic sac? Try again, pushing the needle a little deeper.

If you aspirate blood, it may be foetal or maternal blood. Check the foetal heart ½hrly for 4h. If the rate rises steadily, the foetus is bleeding. Perform a Caesarean section immediately.

If the uterus becomes hard, and there are other signs of placental abruption, there is bleeding behind the placenta (20.12).

22.2 Inducing labour at term

If labour does not start when you would like it to, you may be able to get it going. If it is progressing, but too slowly, you can speed it up. Distinguish between:
(1) Priming the cervix which might also result in labour without further interference,
(2) The attempt to start labour when the cervix is ripe (induction),
(3) The acceleration of labour (augmentation of contractions) in the active phase with the cervix >3cm dilated.
Here we are concerned with (1) and (2).

If the continuation of pregnancy would be harmful to a mother or to her foetus, and especially if either of them is in danger of death, the logical solution might seem to be to start labour and deliver the foetus. Unfortunately, this action has its own risks for both, so there are *very few* indications for doing it in a district hospital. The commonest one is probably proven rupture of the membranes (22.4) lasting >24h, when the mother is near term (>37wks). This indication is pressing if there are signs of infection (elevated temperature, tachycardia in the foetus).

Prostaglandins given orally or vaginally normally without artificial rupture of the membranes (ARM), or oxytocin IV with ARM, are the most powerful ways of starting labour.

The effects of induction/priming with prostaglandin can be more powerful than anticipated. Whilst you can easily reduce the speed of an IV oxytocin infusion, and immediately reduce its action, you cannot so easily stop the effects of administered prostaglandins. Nonetheless, poorly supervised or inappropriate use of oxytocin might also cause foetal death and uterine rupture. In general, the main disadvantage of the use of oxytocin is that it works best when the membranes have ruptured (artificially or spontaneously) and then you are committed: if it does not work in a reasonable time, you will have to perform a Caesarean section. Apart from that, *oxytocin does not work well with an unripe cervix and early in pregnancy*, even if the membranes have ruptured.

Don't use these methods for minor indications, because:
(1) priming might result in too strong contractions with foetal distress, damage or death and uterine rupture.
(2) with ARM you may introduce infection. If labour starts soon, the risk is small, but if it is delayed or prolonged, the risk is large, especially if the foetus is dead. Minimize infection by taking the most careful aseptic precautions if you choose ARM.
N.B. Intra-uterine death is a contraindication to ARM except in cases of abruption when blood loss, not infection, is the overwhelming risk.
(3) if you try to induce labour too soon:
(a) the foetus will be immature and have less chance of surviving.
(b) labour is unlikely to start, and if it does start, it may be so slow that you have to perform a Caesarean section.

So only induce labour when the balance of risks favours it, and Bishop's inducibility test (see table below) shows that the cervix is ripe, and ready for labour.
(4) inducing labour increases your Caesarean section rate, with all the disadvantages this has (21.1).
(5) rupturing the membranes may cause the cord to prolapse.
(6) the placenta may separate (abruption).

So never induce labour to suit your convenience or the mother's, but only for the soundest of obstetric reasons.

If the cervix is unfavourable, you can also try ripening it, but only of course if the membranes are intact, by inserting the balloon of a Foley catheter into the extra-amniotic space.

(a) Indications for starting labour

(1) Proven rupture of the membranes lasting >24h when the foetus is near term (>37wks), or when associated with a fever which cannot be explained otherwise (malaria, HIV/TB) even <36wks.
(2) Severe pre-eclampsia or the HELLP syndrome (severe liver pain associated with haemolysis, elevated liver enzymes and low platelets). Of course, the nearer the gestation approaches 40wks, the less is to be gained by potential further growth of the foetus. Therefore, continuing the pregnancy while the mother is at some risk from the pre-eclampsia has less merit the further the pregnancy is advanced. On the other hand, an induction which involves ARM increases the chance of Caesarean section, which also is quite a risk then and in future pregnancies.
Low-dose misoprostol (half a tablet, *i.e.* 100µg, dissolved in 20ml water) giving 4ml (= 20µg) orally 3hrly, might be the solution. No ARM is needed. The dose/frequency can be increased if the urgency is high and decreased if the parity is high.
(3) Diabetes.
(4) Abruption (20.12).
(5) Postmaturity (22.5) is an uncertain indication, because the diagnosis is rarely made in district hospital practice. There should be, apart from the gestation of >42wks, very little liquor or a sudden decrease in foetal movements or a bad obstetric history before you try to induce labour for this indication.

If the indication for induction is rupture of membranes, then ARM is of course not needed provided you are sure. Try to abstain from entering the cervix during examinations. **If there is a lot of liquor pouring out**, placing 200µg misoprostol in the posterior fornix will probably not help because the active ingredient will be washed out. Use oral, sublingual, or buccal misoprostol or oxytocin IV instead.

BISHOP'S INDUCIBILITY SCORE. Assess the dilatation of the cervix, its length, its consistency, its position in relation to the axis of the vagina, and the height of the foetal head. Add up each individual item: the higher the total score is, the more likely that induction will succeed. The highest score is 13; a score of ≥7 is favourable for induction.

SCORE	**0**	**1**	**2**	**3**
Dilation (cm)	0	1-2	2-3	3-4
Length (cm)	0	3	2	1
Station of the head	5/5	4/5	3/5	2/5
Consistency	Firm	Medium	Soft	
Position of cervix	Posterior	Middle	Anterior	

Don't confuse the position of the cervix with the position of the presenting part (occipito-anterior OA, occipito-posterior OP, etc.).
Make a poster of this score system and keep it on the Maternity wall.

(b) Ripening the cervix

INDICATIONS
When the cervix is not sufficiently ripe to enable you to rupture the membranes to induce labour. After ripening, labour will often start without any need to rupture the membranes (22.4).

METHODS. There are several ways of ripening a cervix:
(1) **A dinoprostone vaginal tablet** (3mg) **in the posterior fornix** on the evening before you induce labour. Follow this by another 3mg 6-8h later if labour is not established, and then, if necessary, a further one, to a maximum of 3.
CAUTION!
(i) The tablet must be close to the cervix in the posterior fornix; merely slipping one into the introitus does not work.
(ii) Avoid prostaglandins in multipara ≥5.
N.B. There may be hyperstimulation, so:
(iii) Insist on close overnight observation of mother *and* foetus.
(2) **A dinoprostone** 0·5mg (prostaglandin PGE_2) **oral tablet in the cervical canal**. Repeat this 6hrly up to 4 doses.
(3) **A misoprostol tablet (200µg) in the posterior fornix.** This is cheap and effective and does not need refrigeration. In many countries it is not registered for induction of labour but is used extensively anyway. It is certainly the drug of choice if the indication is intra-uterine death or severe foetal abnormalities (*e.g.* anencephaly).
The dose depends on the parity, gestation, ripeness of the cervix, urgency and level of monitoring available. *After 30wks, a scarred uterus is a contraindication*.
The *maximum* dose, *if the aim is for a viable foetus*, is 200µg in the posterior fornix for a nullipara with severe pre-eclampsia at about 35wks (introduce it very early in the morning). You can repeat this dose (if absolutely nothing has happened) at the earliest in 6h. If this is not the first delivery, 100µg is better.
N.B. Misoprostol 100µg (½ tablet) dissolved in 20ml water provides 5µg/ml. The 200µg tablets cannot reliably be broken in pieces smaller than a half.
A multipara 4, 42wk pregnant woman, for example, with very little liquor would need only 20µg orally, to be repeated if *nothing* happens 3hrly.

By contrast, if there is an intra-uterine death or severe foetal abnormality at 22wks and there is no scar in the uterus, put 600µg vaginally starting very early in the morning and repeating 4hrly till labour starts. *With a scar, after 13wks, use only 100µg.*

(4) **A Foley catheter in the extra-amniotic space** is useful if you have no prostaglandins. 12-18h before induction, with careful aseptic precautions, and under direct vision, use a Cusco's speculum to insert a Ch16-24 catheter, with a 30-45ml balloon, into the extra-amniotic space. Inflate this with 30-45ml of sterile water, and leave it in place. This is generally 4h faster than using prostaglandins.

CAUTION! Whenever you induce labour, monitor the foetus carefully.

N.B. Don't waste time trying to induce labour if the Bishop's score is <7 and there is an overwhelming indication to deliver: perform an immediate Caesarean section!

(c) Oxytocin to induce labour at term
(Other uses: 21.5)

INDICATIONS
A high risk factor, particularly for the foetus, such as:
(1) Diabetes.
(2) Gestational hypertension, especially if there is proteinuria.
(3) An unstable lie (22.8).
(4) A confirmed dead foetus (20.4).
(5) Postmaturity (22.5).
(6) Twin or triplet pregnancy.

CAUTION! For all these indications, ripen the cervix first, according to the score given above.

(7) Placental abruption with intra-uterine death (20.12): *here there is a vital urgency, so don't wait!*

CONTRAINDICATIONS
(1) Cephalopelvic disproportion (CPD). This diagnosis is difficult to make before labour, however. *Don't give oxytocin to a multipara:* there is too great a risk of uterine rupture, and artificial rupture of membranes will usually suffice to induce labour.
(2) A previous Caesarean section, unless close round-the-clock supervision is possible, and the previous Caesarean section was certainly not for a CPD nor for 'failure to progress'.
(3) Previous myomectomy, uterine perforation or cornual ectopic gestation.
(4) Foetal distress.
(5) Malpresentation.
(6) Grand multiparity.
(7) Placenta praevia.

N.B. Don't use oxytocin for induction/augmentation of labour without rupturing the membranes first. However, in an HIV+ve patient, try not to rupture during induction till late in the 1st stage of labour.

METHOD
Use only low-dose oxytocin as mentioned below under 'protocols on the wall'. Also, as soon as labour is established, try without oxytocin.

Check the foetal lie and presentation, and try to make sure that one nurse stays with the patient all the time. Start *early* in the morning with a dose of 10mIU/min (10 drops/min of 5IU (usually one vial) in 500ml saline where the system gives 10 drops/ml). Monitor her closely and increase the IV rate every 30mins like this: 10 drops/min, 20 drops/min, 40 drops/min, and 60 drops/min. *Have a poster with the detailed protocol on the wall.* Increase the infusion until the uterus is contracting 3-4 times every 10mins. If vaginal examination shows that the cervix is not dilating, increase the infusion to 60 drops/min as long as she has not >5 contractions in 10mins and there are no foetal cardiac decelerations. If this does not work and the foetal heart rate is fine, it is possible *in nullipara* to increase the concentration to 10IU in 500ml and start again at 40 drops/min. *Don't go above 60 drops/min (=120mIU/min) with this concentration*.

N.B. It is not the oxytocin itself which is dangerous but the strength or frequency of the contractions. Therefore high doses without strong, frequent, or continuous contractions are not dangerous in theory, but lack of close supervision to detect these contractions can still damage the foetus or kill mother and/or child.

When the cervix is >5cm, and contractions are good, you may be able to reduce the rate of the infusion. Do this gradually.
If the contractions diminish, increase it again.
If the membranes have not ruptured, and labour has not started by 7pm, stop the infusion and try again in the morning. If the membranes have ruptured, induction must not stop; add antibiotics after 12h with membranes ruptured.

CAUTION!
(1) Higher doses than the above increase the uterine tone between contractions, and so impair the placental circulation. Palpation does not detect this increased tone, unless it is gross. Too much oxytocin will cause prolonged tetanic contractions, and may rupture the uterus (especially in a multipara).
(2) In a multipara, reduce the starting dose to 1IU, and reduce or stop the infusion as soon as there are regular contractions.
(3) Assess the uterine contractions carefully. If there is no relaxation between contractions, stop the infusion. If there are >5 contractions per 10mins, reduce the dose.
(4) Oxytocin in high doses (>60mIU/min) has an antidiuretic effect. *So beware of 'water intoxication'* (20.4), especially if 5% dextrose was infused instead of saline or Ringer's lactate.
(5) If delivery has not occurred but contractions are satisfactory and progress is good, you can use up to 2L of a solution of 10IU in 500ml. Greater than this volume, there is a risk of water intoxication, as above. If delivery is not about to happen, consider Caesarean section.
(6) *Don't infuse >3L in 24h without careful review.*

All sorts of different protocols exist for the use of oxytocin: ideally, of course, an infusion pump will give a controlled dosage with limited fluid. You should remember that the sensitivity to oxytocin differs from person to person.

By increasing the dose in steps of ½h, you can find the right dose for a particular woman without losing too much time, remembering that the longer membranes are ruptured, the greater the risk of infection.
Excess oxytocin is characterized by:
(1) foetal distress,
(2) too frequent contraction,
(3) pain between contractions,
(4) restlessness,
(5) vaginal haemorrhage suggesting threatened rupture.
As oxytocin is metabolized fast in the body, you can slow down or stop the infusion (and put the woman on her left side): the tissue concentration of oxytocin will quickly decrease and the situation will normalize.
Occasionally, the right dose for a particular uterus to dilate is too high a dose for the foetus to endure. At the peak of contraction there is generally no new oxygen available for the foetus. Most foetuses can cope, *but not if their oxygen supply was already marginal*. (Compare this with somebody with severe asthma who is pushed under water for 30secs.) In that case, a Caesarean section is mandatory. However, a foetus with normal reserve oxygen can still get into trouble if the maternal contractions persist too long or are too frequent. This happens more often if labour has not started spontaneously (unripe cervix/uterus) but was induced.

(d) Protocols on the wall

In the absence of an infusion pump, the dose needed in terms of drops/min is inaccurate. There are paediatric IV systems which form such small drops that it takes 60 drops to constitute 1ml but there are also systems (blood giving sets) with 10 drops/ml. This should be indicated on the wrapping of the IV system. It is best to express the oxytocin dose in mIU ($^1/_{1000}$ of an IU)/min. Not all of your staff will be able to do the calculations correctly, and perhaps also yourself at 3am! Because of this, put a poster on the wall of your maternity department to give simple instructions for the use of infusion pumps if available, or for the different IV systems available in your unit.
Using 5IU oxytocin/500ml saline, the dose is, in mIU/min:

System (drops/ml) Flow rate (drops/min)	10	15	20	25	30	35	40
10	**10**	**6·5**	**5**	**4**	**3·5**	**2·5**	**1·5**
20	**20**	**13**	**10**	**8**	**7**	**5**	**3**
40	**40**	**26**	**20**	**16**	**14**	**10**	**6**
60	**60**	**39**	**30**	**24**	**21**	**15**	**9**

If you use 5IU oxytocin/L saline, halve the above figures.
If you use 10IU oxytocin/500ml, double them.

N.B. Never use >20mIU/min with a scarred uterus or para ≥4, and >40mIU/min with para 2-3. An IV system with 20 drops/ml has to run half as fast to deliver the same dose as one running at 40 drops/ml.
Try to have a nationally agreed schedule, adapted to the IV systems you normally have available, as your basic protocol displayed on the wall.

(e) Rupturing the membranes to induce labour

CONTRAINDICATIONS
(1) A high mobile head: the cord may prolapse.
(2) A dead foetus (except in abruption): labour will be quick.
(3) Maternal polyhydramnios: start by withdrawing some amniotic fluid via the abdomen with a needle or cannula, so as to reduce the uterus to a normal size. A sudden release of much fluid can precipitate abruption, and make malpresentations, such as a shoulder presentation, more likely.
(4) Postmaturity.
(5) A (potentially) HIV+ve patient: prolonged rupture of membranes is a risk factor for vertical transmission. If the patient is not receiving ARV therapy, then rupture the membranes as late as possible; you may, however, be forced to do so earlier in order to make labour progress.

N.B. She might be a candidate for Caesarean section (instead of induction, to prevent vertical transmission) combined with a tubal ligation, to prevent future vertical transmission, *if her family will thus be complete.*

METHOD (GRADE 1.1)
Make sure the bladder is empty. Check the foetal heart, put the patient into the lithotomy position, and use careful aseptic precautions. Flood the vulva with antiseptic solution. Wearing sterile gloves, do a careful vaginal examination and measure Bishop's score (see above). Spread the labia widely, put 2 fingers into the vagina and then into the cervix. If necessary, stretch it to admit your 2 fingers. Gently sweep the membranes away from the lower segment without rupturing them. Feel carefully for the placenta, or the cord.

If you can feel the placenta (which is unlikely if the head is low), it is a placenta praevia and you have a large risk of antepartum haemorrhage. Perform a Caesarean section if it is type III or IV (20.11, 20-9).

If you can feel the cord presenting through the membranes, leave them intact, turn the lady on her side and repeat the examination in 2h. With luck, the cord will have floated away. If it has not, perform a Caesarean section, but feel again just before you start the operation.

CAUTION! If labour has started, rupture the membranes during a contraction, to minimize the risk of prolapse of the cord and let the liquor come out slowly.

If you cannot feel either the placenta or the cord presenting through the membranes, rupture them with Kocher's forceps. Hold these in your left hand, and guide them through the cervix with your right hand. As you prepare to tear the membranes, ask an assistant to push the presenting part into the pelvis. This will allow the fluid to escape in a controlled way, and will minimize the risk of the cord prolapsing.

Grip the membranes and tear them. If fluid flows, or there is foetal hair in your forceps, you have succeeded. Note the

amount and colour of the amniotic fluid, make sure the cord has not prolapsed, and check the foetal heart.

N.B. Finding meconium-stained liquor at this stage is not an indication for a Caesarean section; it just means, especially if it is fresh thick meconium, that supervision should be even closer if at all possible.

Enlarge the opening with your fingers. Keep them in the vagina until the head has descended against the cervix. Check the foetal heart again. If the mother has a sudden persistent bradycardia:
(1) She may have the supine hypotensive syndrome, so turn her on her left side.
(2) The cord may be trapped. *Don't raise the foetal head*, because the cord will probably prolapse further (22.9). Instead, turn the mother on her side and listen again; this usually solves the problem.

If the cord has prolapsed, put the mother in the knee-elbow position, push up the head and prepare for an urgent Caesarean section. If there is a delay, filling the bladder via a catheter with the help of an infusion bag of 500ml fluid will often prevent fatal cord compression. Alternatively, separate the amniotic sac membrane from the cervix (a membrane sweep) at vaginal examination (this releases the prostaglandins) only, and *don't rupture the membranes until she is well advanced in labour*. This can be effective, and there is less risk of infection than when the membranes are ruptured some time before delivery.

N.B. As mentioned above, prolonged rupture of membranes increases the chance of vertical HIV transmission. Still there are indications to perform an ARM. If you use a Kocher's and you push the membranes against the presenting part and then rupture the membranes, you will often catch some hair but also scratch the foetus, increasing the chance of mother-to-child transmission.

N.B. Using misoprostol to prime the cervix/induce labour has a disadvantage in that the medication cannot be controlled as easily as an oxytocin infusion. On the other hand, with misoprostol, the membranes don't need to be ruptured till (very) late in labour, making mother-to-child transmission less likely and making it possible to try again the next day.

On the other hand, failed induction with ruptured membranes soon forces you to perform a Caesarean section especially if an intra-uterine infection develops. Unless the indication for induction is vital, start with a low dose of misoprostol to prevent complications in case of extra sensitivity to this drug. With a low dose, there is of course the risk that you have to repeat the induction (if the cervix has not changed with a somewhat higher dose) the next day.

N.B. If you perform a Caesarean section for prolapsed cord (or nearly every other indication), check the foetal heart just beforehand with the best instrument you have: preferably ultrasound (38.3) or handheld Doppler tool (doptone).
Don't open the abdomen for a dead foetus (21.8)!

22.3 Preterm labour

Strictly speaking, preterm labour is the onset of regular painful contractions <37wks. In practice, you can treat labour between 34-36wks as if it was at term, so that it is only labour <34wks (foetus <2·2kg) that needs managing differently. It may or may not be associated with rupture of the membranes. It is sometimes associated with the use of herbal medicines or abortifacients.

The management of preterm labour is not a success story. *Avoid IV tocolytics*: they are dangerous if not well supervised, especially if there might be a hidden maternal cardiac abnormality. There may be a place for steroids. However, using medication may divert you from the treatment of the cause of the premature labour, which may be antepartum haemorrhage, urinary tract infection, or intra-uterine growth retardation, etc.

In practice, when a mother does start preterm labour, there is little you can do about it. It often stops spontaneously, and 70% of mothers don't deliver within 48h, and start labour normally nearer term. You can try to postpone delivery for at least 48h in order to ripen the foetal lungs with 12mg betamethasone IM od for 2 days. You can also try 20mg nifedipine PO in the 1st hour followed by a maximum of 90mg/day in divided doses. Between 24-30wks, indomethacin 50-100mg (preferably as a suppository) followed by 25mg 4hrly for a maximum of 3 days might help.

If labour starts before 34 completed weeks, **and the membranes have not ruptured**, assess as follows:
(1) In the active phase of labour (the cervix is >3cm), try to delay delivery with nifedipine (or indomethacin if at <30wks) in order to gain time to ripen the foetal lungs with steroids.
(2) In the latent phase of labour (the cervix is <3cm) with regular contractions, look for a possible cause, although you are unlikely to find one. Insist on bed rest, and try to delay delivery as above.

If the contractions are doubtful, consider other common or less common causes of pain:
(1) urinary tract infection or partially kinked ureters (21.5),
(2) constipation (12.15), sometimes the result of pica,
(3) abruption (20.12),
(4) appendicitis (14.1),
(5) bowel obstruction (12.2),
(6) red degeneration of a fibroid
(7) an extra-uterine gestation (rare, 20.9).
If you have the slightest suspicion, treat with antibiotics; in the next pregnancy use them after 13wks for 1wk.

At delivery, the foetus is at high risk, so control the delivery of the head very carefully. Handle the foetus gently and keep him warm, using the kangaroo method (*i.e.* carrying the preterm infant skin-to-skin next to the mother).

If labour starts before 34 completed weeks, and the membranes have ruptured, see below (22.4).

22.4 Premature rupture of membranes and intra-uterine infection

When labour is normal, regular contractions start and the cervix begins to dilate before the membranes rupture and amniotic fluid escapes. Sometimes, the membranes rupture first, before contractions start, either <37wks (preterm) or at term (prelabour). The risks are:
(1) intra-uterine infection, which is by far the most important but is usually not common, and
(2) premature labour.

The advantages of expectant treatment (not inducing labour) are that:
(1) foetal maturity increases, which is important if gestation is <36wks, and
(2) the risks of induction are avoided. These are:
(a) failure, which means that you will need to perform a Caesarean section, and
(b) the side-effects of oxytocin (21.5).

The disadvantage of expectant treatment is the risk of infection (chorio-amnionitis) which may kill the mother and/or the foetus. You can minimize this risk by:
(1) totally avoiding vaginal examination with your fingers until contractions are well established,
(2) avoiding speculum examinations as much as possible,
(3) practising reasonable vulval hygiene,
(4) observing carefully for signs of infection, inducing labour and treating with antibiotics: you should treat all cases of prematurely ruptured membranes preferably with erythromycin for 1wk.
Intra-uterine infection is such a serious risk when membranes are ruptured for >24h that it far outweighs any benefit that might follow from expectant treatment.
Particularly if puerperal infection is common in your area, aim for delivery within 24h. Fortunately, labour usually starts successfully within this time.
Avoid unnecessary vaginal examinations and teach that:
(1) The risk of prolapse of the cord is small, but the risk of infection is great.
(2) Cord prolapse will only physically harm the foetus, but infection will endanger the mother also.
(3) If the cervix is still closed, as it often is, *vaginal examination certainly does not help* because there cannot be a cord prolapse. *Teach that premature rupture of the membranes means there needs to be a very good indication for vaginal examination!*

(a) Abdominal ultrasound (38.3). Ideally use this: occasionally you might detect a cord prolapse; you can usually rule it out confidently; you may also deduce a simple high tear in the membranes if there is still a lot of liquor.

(b) History
Loss of fluid from the vagina, before the onset of regular painful contractions, is diagnostic. If you are not sure of the dates, or there appears to be a discrepancy, assess the foetal age by ultrasound (38.3). If you cannot do this, estimate the fundal height (22-15).

(c) Examination
Start by separating the labia and asking for a cough: Is liquor discharging from the vagina? Is urine coming from the urethra?

If you don't see any fluid, repeat the examination after a few hours, so as not to miss intermittent loss of liquor from a small leak. Do one clean speculum examination, to make sure that the membranes have ruptured, and that there is really draining of liquor. Make sure that a senior person does this, so that it need not be repeated. Ask the patient to cough: you may see fluid escaping from the cervix. Then:
(1) observe the dilatation of the cervix, but remember that a vaginal examination is much more reliable than visual inspection,
(2) feel the degree of cervical effacement,
(3) confirm the presenting part: you may see it if the cervix is open,
(4) exclude prolapse of the cord.

CAUTION! Don't do a vaginal examination with ungloved fingers: the risk of infection is too high.
Alternatively, avoid this examination, and merely 'wait and see'. If fluid continues to flow (as shown by checking the pads), the membranes are obviously ruptured.

If you are not quite sure if the fluid that is draining is liquor or urine:
(1) smell it,
(2) test its pH (urine and vaginal discharge are acid, amniotic fluid is alkaline),
(3) leave some to dry on a slide. Look at it under a microscope. Liquor, but not urine or a discharge, will dry as a pattern of ferns (like a bush or tree). If you have not done this test before, try it with some known liquor.

(d) Management
If the diagnosis is confirmed or suspected, admit the patient, provide her with a clean perineal pad or cloth, make sure she keeps the vulva and perineum clean, check the temperature 4hrly, and inspect the liquor daily by visual inspection of the perineal pad and its smell.

If no liquor can be seen escaping >3 days, the diagnosis is not confirmed, so discharge her. 25% of patients stop leaking liquor in 3 days and can be discharged. 75% start spontaneous labour during this time.

If gestation is <28wks, with a live foetus, and there are no signs of infection, the chances of the pregnancy continuing long enough for the foetus to survive are small, but not zero.

If you diagnose an infection, induce labour, and start antibiotics. Labour will usually start soon, and the pregnancy may survive.

If gestation is 28-35wks, treat prophylactically with antibiotics, preferably erythromycin.
N.B. If you use antibiotics before there are signs of infection, they might prevent infection and labour.

Once an obvious infection is established, induction is needed as well as antibiotics as before to prevent spread of this infection. *As long as the foetus is in the uterus, it is too late to expect infection to be cured without evacuating the uterus.*
If the liquor stops draining, don't intervene. If it continues to drain at 48h, induce labour, if the risk of infection is high. Otherwise, wait until the foetus is more mature at 36wks. Culture the amniotic fluid at delivery.

If gestation is at >36wks, induce labour with oxytocin, if labour does not start spontaneously in 24-48h.
CAUTION! Be sure to induce labour if:
(1) The foetus is dead at any stage of pregnancy.
(2) There are signs of intra-uterine infection at any stage of pregnancy.
(3) Gestation is >36wks, and labour has not started spontaneously in 24-48h. Remember the precautions for the use of oxytocin (21.5, 22.2) and use it with extreme caution in the presence of infection. Consider a Caesarean section if the patient wants a tubal ligation.

(e) Diagnosis of intra-uterine infection:
(1) Foetal tachycardia.
(2) Maternal pyrexia and tachycardia.
(3) Uterine tenderness.
(4) Offensive, blood-stained liquor.

TREATMENT. There is a high risk of septicaemia with development of septic shock. Resuscitate with IV fluids. Treat with broad-spectrum IV antibiotics; *e.g.* chloramphenicol and metronidazole (2.8,9) plus benzylpenicillin against β-haemolytic streptococcal infection in the foetus. Empty the uterus as soon as possible, whatever the duration of pregnancy: it will often empty spontaneously. If it does not, use an oxytocin infusion with great caution, and stop it as soon as there are regular contractions. The foetus usually dies, if it is not already dead when the mother becomes infected.

(f) Difficulties with intra-uterine infection
If gas bubbles emerge from the cervix, or you feel crepitus in the cervix or abdominal wall, this is from gas-producing micro-organisms, which is likely to be GAS GANGRENE (6.24). The uterus and abdominal wall may be distended with gas. Peculiarly, the foetus, which is most often dead, may appear to be crying in the uterus because gas makes it possible to produce sound.
Use large doses of penicillin, chloramphenicol, and metronidazole IV, and evacuate the uterus rapidly.

If the infection has spread to the wall of the uterus, perform a hysterectomy to save the mother's life.

22.5 Postmaturity

A foetus >2wks postmature is at increased risk of stillbirth, but there is little evidence that inducing labour before 42wks significantly reduces the perinatal mortality.

The risks of accidental premature induction are considerable:
(1) A mother's dates may be wrong, because she has had no menstrual period since the previous pregnancy, or simply because of breastfeeding, which causes a longer menstrual cycle. Thus, if periods occur every 2 months, conception will occur 6wks after the last period, instead of 2wks. This means that pregnancy is usually less advanced than mothers think.
(2) Many mothers present so late for their first antenatal visit that the size of the uterus or foetus cannot be reliably used to confirm gestational age.
Even ultrasound is of little help after 26wks and cannot reliably differentiate between 38 and 42wks.

If gestation is >42wks, and the dates are certain, admit the patient and ask her to keep a foetal movement chart. If there are any of the risk factors below, induce labour with low-dose misoprostol (22.2) or oxytocin, if the cervix is ripe:
(1) Nulliparity >30yrs.
(2) Twin pregnancy: *this is postmature at 40wks so should certainly not be allowed to progress to 42wks.*
(3) A bad obstetric history.
(4) Gestational hypertension.
(5) Markedly reduced foetal movements.
(6) A cardiotocograph, if available, that is not reassuring.
(7) No liquor as measured by ultrasound or palpation.
(8) Gestational diabetes.
(9) A dead foetus: *don't rupture the membranes*.
N.B. Repeatedly failed induction is sometimes a clue that there is a large extra-uterine gestation (20.9). Other clues are that a gloved finger pushed through the cervix does not palpate membranes or a foetus: a fibroid is assumed to be present, but is in fact the whole uterus!
N.B. Ultrasound often misses the diagnosis of advanced extra-uterine gestation.

22.6 The malformed foetus

With most congenital malformations, a foetus is not large or misshapen enough to cause difficulty during labour. The important exceptions are anencephaly and hydrocephalus. If you have the misfortune to find a grossly abnormal conjoined twin, Caesarean section is the method of choice. Omphalocoele (33.4) and gastroschisis are, on the other hand, not in themselves indications for Caesarean section.

Anencephaly is complicated in 90% of cases by polyhydramnios; so when you diagnose this, do an ultrasound on the mother to see if the foetus has a brain (38.3). If not, it is usually stillborn, and even when it is born alive, it does not survive >6h. When you have explained the diagnosis to the mother, she may insist that the pregnancy is terminated.

Hydrocephalus is not always easy to diagnose clinically, and is often missed during pregnancy. A common mistake is to misdiagnose a brow presentation (when the head feels big) for hydrocephalus.

If you suspect it, confirm the diagnosis by ultrasound (38.3). Even during labour the diagnosis is easily missed, if you can't feel the widely distended sutures and fontanelles. If the diagnosis is doubtful, wait. If it is obvious, proceed as follows:

(a) Anencephaly

If this is accompanied by (painful) polyhydramnios, drain the mother's amniotic sac with a needle or cannula through the abdominal wall. Sometimes you have to manipulate the foetus through the abdominal wall in longitudinal position while the water is draining. A breech presentation is no problem. Use low doses of misoprostol erring on the side of too little because there is no medical hurry.

If anencephaly is not accompanied by polyhydramnios (10%), pregnancy may rarely be prolonged up to 1yr or more, and make delivery difficult. Misoprostol is the drug of choice also, the dose depending on the parity and possible uterine scarring. If the membranes are ruptured, a higher dose is acceptable because there is now the risk of infection.

(b) Hydrocephalus

If you make the diagnosis during pregnancy, try to measure the size of the foetal head and determine if there is also a spinal deformity. Discuss the problem with parents, explaining the need for a ventriculo-peritoneal shunt or third ventriculostomy and the problems the child may encounter (33.11). *Don't endanger the life of the mother trying to save the foetus!* Try hard to avoid a Caesarean section if you think the chances are that the foetus will die anyway or be severely handicapped. In this case, induce labour without artificial rupture of membranes. If you wish to be sure of saving the foetus and the mother understands that a future pregnancy will almost certainly mean another Caesarean section, prepare for this option.

If you make the diagnosis when labour with a cephalic presentation has been in progress for some time, and the foetal head is more than minimally enlarged, you will have to make it smaller before you can deliver it. Drain the CSF with a lumbar puncture needle (draining the CSF does not kill the foetus!). This is simplest under ultrasound guidance. If you are not sure of the diagnosis, or don't feel you can risk sacrificing the foetus, you may be forced to perform a Caesarean section. However, even then the head may be too large for a routine lower segment Caesarean section.

The alternatives are:
(1) The best, to drain the head before making the uterine incision;
(2) 2nd best, to make a transverse, curved incision 4cm higher than normal with the ends near the attachments of the round ligaments;
(3) 3rd best, to make a vertical incision starting in the midline in the lower segment at the level (2cm lower than the bladder reflection) where you would normally do a lower segment transverse incision.

N.B. Take care that it does not tear further distally because you made the incision in the direction of the fundus too short.

In order to avoid a Caesarean section when there is no cervical dilatation or the foetal head is still very high, it is very easy to drain CSF from the head through the mother's abdominal wall (with an empty bladder) with a thick needle or cannula. The head then collapses, engages and delivers vaginally if the mother is in labour. If she is not, and the membranes are intact, there is no hurry anyway. To perforate the foetal head vaginally, wait until the cervix is >3cm dilated, then drain the cerebrospinal fluid with a large needle or artery forceps between the widely separated skull bones. The collapsed head will slowly settle into the mother's pelvis, and delivery will be simple. In this situation, however, you are committed because the membranes are ruptured and if labour has not started or stops, you might have to perform a Caesarean section on a dead or non-viable foetus. So be sure labour has started before you rupture the membranes.

CAUTION! If possible, perforate the foetal head before the cervix is 5cm dilated, because an over-distended lower segment may rupture if you don't.

If you make the diagnosis during a breech presentation (22.7), the foetus will probably deliver spontaneously as far as the umbilicus. Often you are alerted to the hydrocephalus by seeing clubfeet (32.10) and/or a spina bifida (33.10). Progress will then be arrested as the hydrocephalic head fails to enter the pelvic brim. *Trying to save the foetus is not now your priority!* Draining the CSF is less messy than a craniotomy. If, at this stage, you see the commonly associated meningomyelocoele, pass a steel or gum elastic male catheter through the spinal defect into the ventricles, to drain off the CSF.

If there is no spina bifida, you can easily perforate the head at the back very near the neck with a Kocher's or artery forceps. Alternatively, make sure that the bladder is empty, and then tap the aftercoming head abdominally with a large spinal needle.

N.B. Beware of performing a symphysiotomy to deliver a hydrocephalic baby: the symphysis may separate so much that severe urinary stress incontinence may result.

LINDA (17yrs) started labour normally, but did not progress. The district doctor failed to notice that this was because there was a hydrocephalus: she had not seen such a case before. She decided to perform a Caesarean section, but noticed that she could not deliver the foetal head through the lower segment incision. She did not panic but drained the head with the help of the scalpel. The foetus was put on the resuscitation table but not attended to because everybody thought it was dead. Then it started crying. It survived for 6 months, paralyzed from the waist down because of a spina bifida. It dribbled urine continuously and probably died of a urinary tract infection. The mother became psychotic and needed a long period of rehabilitation.
LESSON Remember hydrocephalus as a cause of failure to progress, and consider draining the liquor.

22.7 Breech presentation

If a foetus presents with his buttocks (breech) or his foot, he is about four times more likely to die than if he presents by his vertex. This is so even if you exclude the excess mortality due to the higher rate of prematurity, multiple pregnancy and foetal abnormality that is associated with breech deliveries.

This increased mortality is due to:
(1) Late diagnosis, in the case of CPD, unless you can perform a swift, skilled symphysiotomy (21.7).
(2) Lack of time for the head to mould so that there is effectively CPD though this would not have been the case in a cephalic presentation.
(3) An incompletely dilated cervix (especially with straining before full dilatation, or prematurity).
(4) The increased risk of cord prolapse.
(5) Nobody being present to solve the problem of the arms being extended.

The following methods may help prevent breech delivery if you find a breech presentation after 36wks:

(a) The knee-chest position (as if praying like a Muslim, 22-2D) is an alternative to manipulation that might succeed. It is also safer. Ask the mother to spend 10mins tds in this position. This may allow the foetal breech to disimpact from the pelvis, so that it can turn spontaneously.

(b) External cephalic version (ECV)
If you can reduce the number of breeches you deliver, you will reduce the perinatal mortality associated with them. Turning a breech presentation is of little value <34wks in a primipara, or <36wks in a multipara, because many breech presentations spontaneously correct themselves before this. After 36wks, a foetus gradually becomes less mobile, which makes version more difficult. On the other hand, if version does succeed, it is more likely to be permanent.

The risks of ECV include:
(1) Knotting of the cord.
(2) Placental abruption.
(3) Uterine rupture.
(4) Vertical transmission of HIV.
These risks must be compared not only with the risks of breech delivery but also of Caesarean section.

Unfortunately, ECV is not often done by doctors or by experienced midwives as often as it should be; *it should not be done by inexperienced practitioners*. If your excess perinatal mortality with breech deliveries is >2%, after correcting for prematurity and foetal abnormality (see below), the risks of ECV are worth taking. *Don't attempt it under GA!*

Corrected perinatal mortality for breech deliveries
This should be fairly easy to calculate from your labour ward record books, which should routinely record presentation, birth weight, obvious abnormalities, and live and stillbirths.
(1) Work out your perinatal mortality for all babies (10-80/1000) excluding breeches, babies <2·5kg, twins, and babies with obvious malformations. The perinatal period lasts (in this case) from the 28th week to the end of the 1st week of life.
(2) Do the same for breech deliveries only. In many district hospitals, it will be 50-200/1000.
Subtract (1) from (2). If the difference is >20/1000, perform ECV.

The problem is of course that the higher the breech-related mortality is, the higher the Caesarean-section-related mortality is likely to be. The reasons for high breech-related mortality usually include absence of permanently available skilled personnel and/or patients arriving too late. These are exactly the same factors which make it very dangerous for women to have a scar on the uterus.

If ECV or the knee-chest position fail, you can deliver a breech:
(1) Vaginally, by assisted breech delivery.
(2) Vaginally, by breech extraction.
(3) Vaginally, adding a symphysiotomy if there is CPD.
(4) Abdominally, by Caesarean section.

In breech extraction you, rather than the mother, provide the power for expulsion. You exert traction on the legs, groins and pelvis, so it is more dangerous for the foetus than an assisted breech delivery, which is the usual way of delivering a breech. Only do this extraction if there is no alternative (foetal distress) or with the 2nd of twins.

Otherwise IV oxytocin will probably help especially if the foetus is smaller than average. If the foetus is large or normal in size, lack of progress of the buttocks in the second stage *might* predict subsequent CPD (see below).

Perhaps an acceptable approach in nullipara would be to prepare for a Caesarean section at 36-37wks. Administer spinal anaesthesia. Try to turn the foetus. There is then a success rate of 2/3 as opposed to 1/3 without spinal anaesthesia.

If you succeed, cancel surgery. This is without danger in a nullipara, but at higher parity where there *is* a risk of rupture, the mother will not be able to warn you by indicating pain. However, a mother who has previously delivered spontaneously vaginally around term has certainly no indication for an elective Caesarean section for a simple breech presentation.

Judgement is difficult nonetheless, if for example a primipara delivered by vacuum extraction the last time. There could have been borderline CPD, or was it just a tired mother or an impatient doctor, occiput posterior position, or foetal distress? Probably the best would be to try ECV but without spinal anaesthesia.

N.B. Performing an elective Caesarean section for breech presentation without very good reasons is irresponsible, if you cannot guarantee good supervision for trial of scar and access to a Caesarean section for the next delivery.

A liberal Caesarean section rate will reduce your perinatal mortality, but you will have to weigh this against the increased maternal morbidity and mortality that will follow. If the difficulties of vaginal breech delivery worry you, and you are tempted to perform a Caesarean section for all breech presentations, remember the dangers of anaesthesia, bleeding, sepsis, and a scarred uterus. However, if your hospital has not the skill available continuously to perform

a vaginal breech delivery and there is no guarantee of a swift referral to a place with these skills, then you might be forced to perform an elective Caesarean section on a breech presentation at 37wks.

If there is any question of CPD before or during the 2nd stage of labour, have everything ready (including infiltrating the symphysis with LA) for a possible symphysiotomy (21.7), or proceed to Caesarean section.
In communities where the contracted pelvis is common, the risks of a breech delivery are great, so that to be sure all these babies survive, you may have to perform a Caesarean section on all mothers without a proven adequately sized pelvis; and this you can only know if they have delivered vaginally at term successfully beforehand.

N.B. Don't allow a mother with a true conjugate (21.4) *of <9cm to deliver a full-term breech vaginally unless you can perform a symphysiotomy!*

A foetus with IUGR or prematurity presenting as a breech is a problem. Much depends on the foetal age:
(1) <28wks gestation (<1kg): the chances of life are small, the lower segment is poorly formed, and it is questionable if Caesarean section will be any less traumatic than vaginal delivery.
(2) From 28-32wks (1-1·5kg), the foetus may have a better chance with Caesarean section, especially if it is a footling presentation. However, about 20% have severe abnormalities, and if you don't have ventilators, even the normal ones have a poor chance of surviving. So, in an area of high parity and high perinatal mortality, you should rarely perform a Caesarean section for a premature breech presentation. It is important to dissuade the mother from pushing before full dilatation: *don't leave her alone!*

Symphysiotomy (21.7) is useful in breech delivery for the unbooked patient, who is admitted in the 2nd stage of labour, and when there is no time for a Caesarean section. However, it is not a good idea to make your *1st unsupervised* symphysiotomy in these circumstances, because if the head is stuck and you have tried everything else, there is then very little time left to deliver an undamaged foetus.

If a breech delivery might end in a symphysiotomy (21.7), it is good practice to infiltrate the symphysis and the skin over it already before it is needed, so as to shorten the time required for the probably necessary subsequent procedure. *Have a catheter and a large size scalpel ready!*

Epidural anaesthesia will prevent a mother bearing down before she is fully dilated, and it will make any manipulations that you have to do in the second stage of a vaginal delivery much easier. An occasional 'stuck breech', and a dead foetus, are more acceptable than a maternal death in most cultures. As your skill and experience and that of your staff improve, so will your successful vaginal deliveries.

EXTERNAL CEPHALIC VERSION is possible at any time >34wks, until labour starts. It is not necessary <34wks. You may not succeed >36wks, but it is still worth trying.

CORRECTING A BREECH PRESENTATION

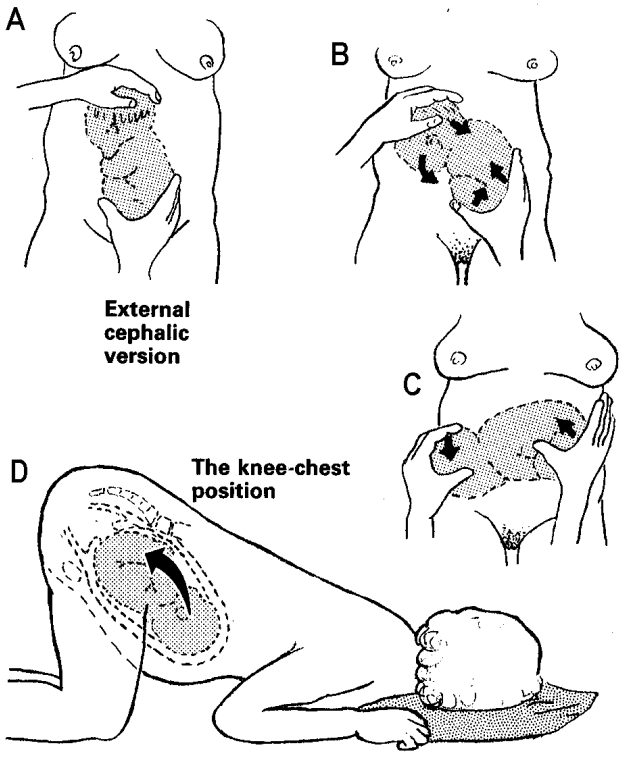


Fig. 22-2 CORRECTING A BREECH PRESENTATION.
A-C, external cephalic version. Flex the foetus between your hands so that you make him do a forward somersault. D, the knee-chest position. Ask the mother to spend 10mins tid like this.

CONTRAINDICATIONS
(1) Multipara >3.
(2) Antepartum bleeding in this pregnancy.
(3) Multiple pregnancy.
(4) Signs of foetal distress.
(5) Premature rupture of membranes.
(6) A previous Caesarean section.
(7) The need to perform a Caesarean section in this pregnancy for some other reason.
(8) A detected foetal abnormality.

RELATIVE CONTRAINDICATIONS
(9) Rh D-ve mother and no anti-D to treat her with. A successful ECV would often prevent more problems than it causes in Rhesus-ve women even when there is no anti-D available. Mortality related to rhesus antibodies seems to be rare in Africa even if allowances are made for the lower prevalence (±4%) of Rh D-ve people.
(10) HIV+ve mother: mixing of blood during ECV might cause vertical transmission.

N.B. If she uses antiviral medication, the risk is probably very small. If she is not using these drugs and she is planning to breastfeed, then perhaps you should proceed with an ECV because the foetus might become infected anyway and a Caesarean section in an untreated HIV+ve patient has greater risks for the mother (not the foetus). If, on the other hand, she wants a sterilization and/or can give safe alternatives to prolonged breastfeeding, then a Caesarean section will be better.

METHOD (GRADE 1.3)

Explain carefully what you are going to do. Empty the bladder and lie the patient supine tilted a little to one side. Flex the knees somewhat. Make sure your hands are warm and she is comfortable. You may find it helpful to lubricate your hands and the abdomen with glove powder. Find out on which side the foetal back is situated. Count the heart rate. Place one hand below the breech, and your other hand above the head. Flex the foetus between your hands, so that you make him do a forward somersault (turn head over heels). Listen to his heart.

If the heart rate slowed to <100, turn the patient on her side and wait until it is >100. If the heart rate has not started to recover within 2mins, turn the foetus into his original position. His umbilical cord may be tight round his neck.

If a forward somersault fails, try turning him in a backward somersault.

If both fail, rest the mother with the foot of the bed raised. If she is anxious, use diazepam 5mg orally. Try again in an hour. If you fail again, try again at the next visit.

If you succeed, see her again 1wk later to make sure the presentation is still cephalic.

If you cannot turn the foetus by 37wks, manage her as a breech delivery.

INDICATIONS FOR CAESAREAN SECTION

N.B. If the patient has a normal or large pelvis, and the foetus is normal-sized, she will probably deliver vaginally. At vaginal examination, if you cannot touch the sacral promontory easily with your middle finger, which means the diagonal conjugate is >11cm (for a size 7 glove hand), the pelvis is probably large enough. The true conjugate, the narrowest diameter the foetal head must pass, is usually 1·5-2cm smaller.

N.B. The best assurance of an adequate pelvis is of course a previous uncomplicated vaginal delivery at term, especially if that was a boy, who has on average a somewhat larger head than a girl.

(a) ANTENATALLY:
(1) Suspected CPD.
(2) A large foetus; if he feels as if he is big, that is >3.7kg (fundal height >40cm), regardless of the size of the pelvis.
(3) A previous Caesarean section.
(4) Other obstetric hazards, such as placenta praevia, diabetes, gestational hypertension, or APH.
(5) An elderly primigravida, or if there is a long history of infertility.
(6) A previous stillbirth, especially if it was associated with a breech or instrumental delivery.
(7) Postmaturity >42wks.
(8) Previous operative vaginal deliveries unless certainly unrelated to (borderline) CPD.

(b) DURING LABOUR:
(1) A prolonged first stage with good contractions or failure to dilate fully. *The best approach is to use the partogram and the routine active management of labour: look for failure to progress which is not quickly amenable to the use of oxytocin.*
(2) Arrest at the brim, or delay in the descent of the breech during the 2nd stage.
(3) A footling presentation (usually with one hip and knee extended): here, a woman can develop an irresistible desire to push before full dilatation, as the foetal feet enter the vagina. This can result in the head being caught behind the undilated cervix. On the other hand, if she can restrain herself till that moment, a multipara with full dilatation can often deliver a foetus in one or two contractions.
(4) Cord presentation or prolapse: this is especially a problem in a frank breech (the feet touching the ears). In a footling breech, the cord is less likely to be compressed even if prolapsed.
(5) Foetal distress before full cervical dilatation.
(6) Prolonged rupture of the membranes with infection, but when labour is not advanced.

N.B. If the membranes have ruptured but there most probably is no CPD, then starting labour with oxytocin and stopping the IV infusion when labour is established, is quite acceptable.

Most additional factors which compromise the wellbeing of a foetus are indications for a Caesarean section. Only for a healthy normal-sized mother with a foetus <3·7kg (as indicated by a fundal height of <40cm), who progresses normally in both stages of labour, should you allow a vaginal delivery.

ASSISTED BREECH DELIVERY (GRADE 1.5)

CAUTION! For breech delivery you need a quiet atmosphere and good communication with the patient. *A crowd of supporters crying "Push!" is not what you want.* Keep calm and explain what is happening. You will need an assistant.

(1) **The 1st stage.** If the cervix dilates at <1cm/h in the active phase, or there are any other signs of delay not quickly remediable by oxytocin, perform a Caesarean section. Until the foetal buttocks are delivered, you can still elect to perform a Caesarean section. If there is any delay before the delivery of the buttocks, go ahead with the Caesarean section.

If extended legs prevent the buttocks coming down, deliver one foot into the vagina by putting two fingers along the foetal inner thigh and pushing it away from the midline (Pinard's manoeuvre).

(2) **The 2nd stage.** A common fault is to try to deliver a breech through an incompletely dilated cervix, which may force the arms to extend and make the head difficult to deliver. Full dilation may not be easy to diagnose in a breech, so take your time for a proper vaginal examination. Put the mother into the lithotomy position (essential if you effect the Burns-Marshall manoeuvre) when the posterior buttock is distending the perineum. As soon as she wants to bear down, do a vaginal examination to make sure that the cervix is fully dilated.

The breech should advance with every contraction. Infiltrate the perineum with LA, and make an episiotomy in a nullipara, when the buttocks are distending it, and you can see a boy's scrotum (or a girl's labia). Protect the scrotum: you don't want the episiotomy to castrate him! The buttocks and legs will then deliver.

N.B. If the mother could have an HIV infection, it is best not to make an episiotomy or do it as belatedly as possible because the mother's blood will come in contact with the foetal genitals and face and increase the risk of transmission.

When the umbilicus delivers, there is often a temporary halt in descent. Look at the clock. The foetus should be delivered in the next 3mins.

Wait for progress to resume with the next contraction. The shoulders and arms should deliver with a twisting movement, and the head should follow immediately. *Don't touch the baby, or try to disentangle the legs*, until you see the umbilicus. Put your hand on the mother's fundus, observe each contraction, and keep a steady gentle pressure on the foetal head. When the umbilicus appears, disengage the extended legs.

CAUTION! Try to make sure that the foetal back is uppermost. *Never allow the foetal abdomen to face upwards.*

When the scapulae appear (and not before), search for the arms in front of the chest. If, as is usual, the arms are not extended, they will both be in front of the chest. You should be able to deliver one or both of them. If you have difficulty, feel up to the shoulder from the foetal back and from there push down the arm, first one then the other.

Allow the body to hang (22-3A). Its own weight will make the head descend through the birth canal. It will have been entering the pelvis, and may now be compressing the cord. Assist its descent with gentle suprapubic pressure. The foetus must be able to breathe in the next 1-2mins.

BURNS-MARSHALL MANOEUVRE

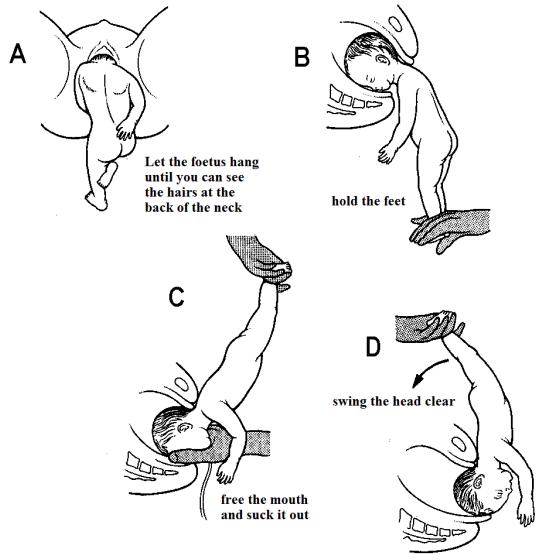

A
Let the foetus hang until you can see the hairs at the back of the neck
B
hold the feet
C
free the mouth and suck it out
D
swing the head clear

Fig. 22-3 THE BURNS-MARSHALL MANOEUVRE for delivering the head in a breech delivery, if it does not deliver spontaneously. A, allow the body to hang, until you can see the hair at the back of the neck. B, hold the feet. C, swing the feet upwards over the mother's abdomen. Free the mouth and pause while you clean it. D, finish delivery by swinging the baby over the abdomen.

If the head does not immediately deliver spontaneously when the arms are out, try the BURNS-MARSHALL MANOEUVRE (22-3: GRADE 2.1). Wait until you can see the hairs on the back of the neck (22-3A,B), pull the foetus outwards a little and draw him outwards over the pubis. Put the back of the baby on his mother's abdomen. Guard the perineum with your left hand (or get an assistant to do so) and prevent the head from emerging too quickly. As soon as the mouth and nose appear, pause, and ask your assistant to clear the airways and allow the baby to breathe (22-3C). Then, carefully deliver the rest of the head (22-3D).

If you cannot get at least the mouth and nose into fresh air with the Burns-Marshall method: (1) use the MAURICEAU-SMELLIE-VEIT MANOEUVRE, or (2) apply Wrigley (outlet) forceps to the aftercoming head.

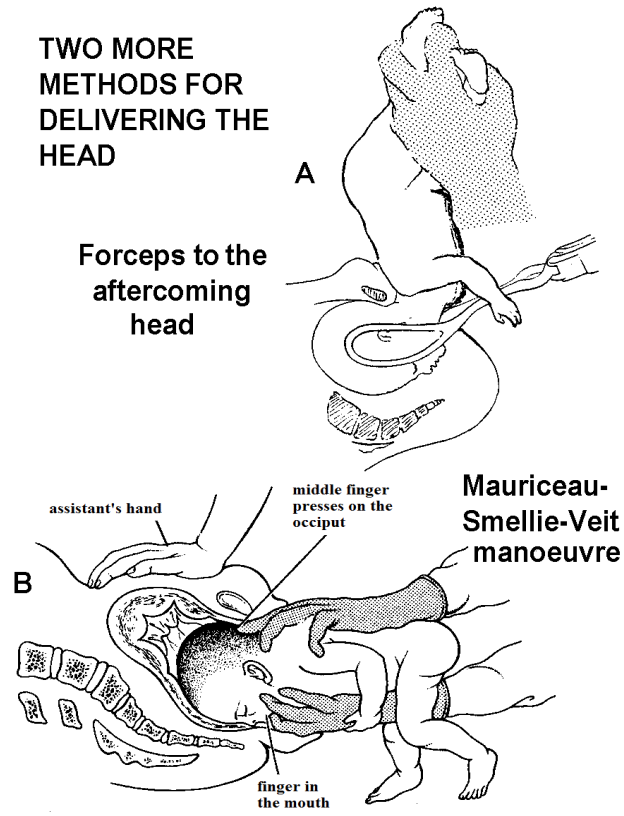


Fig. 22-4 TWO METHODS FOR DELIVERING THE HEAD IN A BREECH PRESENTATION.
A, applying forceps to the aftercoming head. B, using the Mauriceau-Smellie-Veit manoeuvre: Rest the baby's belly and chest on your right forearm; put your right middle finger in the mouth, and your index and ring fingers on the cheek bones. Put your left hand over the back; put your middle finger on the occiput and your index and ring fingers over the shoulders. This will give you some control over the flexion and rotation of the head. Guide the head through the mother's birth canal but *don't pull on the shoulders*. The finger in the mouth is for convenience and orientation only. *Don't apply traction on the jaw!*

N.B. Although 'Mauriceau-Smellie-Veit' is a cumbersome eponym, it is preferred to the alternative, which is 'jaw-shoulder traction', since this suggests, although unintentionally, *traction on the neck, which is very dangerous for the foetus*.

Rest the foetal belly and chest on your right forearm; put your gloved right middle finger in the mouth, and your index and ring fingers on the cheek bones. Put your left hand over the back; put your middle finger on the occiput and your index and ring fingers over the shoulders. This will give you some control over the flexion and rotation of the head. Grip the skull and guide it through the birth canal. Ask the mother to stop pushing. Ask your assistant to put his fist on the foetal head, which is still palpable above the pubis, and to press obliquely downwards in the direction of the coccyx. You will feel a 'plop' indicating that the head has gone into the pelvis, and further delivery should then be easy.

CAUTION! This is a method for getting a grip directly on the head. *NEVER pull on the shoulders:* you can too easily distract the cervical vertebrae and damage the cord.

Early difficulties delivering a breech

CAUTION!
(1) Perform an episiotomy (except in a multipara with a lax outlet) before you do any manipulations, because there is a high risk of a perineal laceration, but lacerations tend to bleed less than early episiotomies, so consider the risk of HIV transmission.
(2) *Don't squeeze the abdomen!*
(3) If the head fails to descend, *don't pull on the neck.*
(4) If the head becomes impacted and the foetus dies, *don't sever the neck, nor be tempted to open the uterus from above*.

If the breech is delayed at the brim, or in mid-cavity, this is probably a warning sign of CPD; perform a Caesarean section. *Don't try to effect a delivery using oxytocin unless you are prepared to make a symphysiotomy*.

If the breech is delayed at the outlet, make sure that the episiotomy is adequate. There may be CPD. If the pelvis feels contracted, or the foetus (or the feet) are large, perform a Caesarean section. If all is otherwise well, continue gentle groin traction, as for breech extraction.

If you have delivered the legs but both shoulders have now stuck above the pelvic brim, the arms are probably extended (22-5A). Normally you can put a finger up the posterior vaginal wall and easily bring them down. If you cannot, they are probably forced into extension. Try LØVSET'S MANOEUVRE (GRADE 2.2). It is a breech extraction for obstruction late in delivery, and should rarely be necessary. The delivery of the shoulders is prevented by two obstructions at different levels: the sacral promontory and the pubis.

The principle of this method is that, by pulling the foetus tightly down, and by turning the body 180º, the shoulder which was held up above the pubis will turn to pass into the hollow of the sacrum.

The shoulder which was above the sacrum will now be above the pubis. A hand in the posterior vaginal space may ease the arm down. Two further 'unscrewing' half-turns like this, each bringing the shoulders progressively below these obstructions, will deliver the foetus.

Grasp the thighs and pelvis with both hands (if the baby is slippery, use a gauze swab or small towel), your thumbs along the foetal sacrum, your forefingers on the foetal symphysis, and your remaining fingers round the foetal thighs.

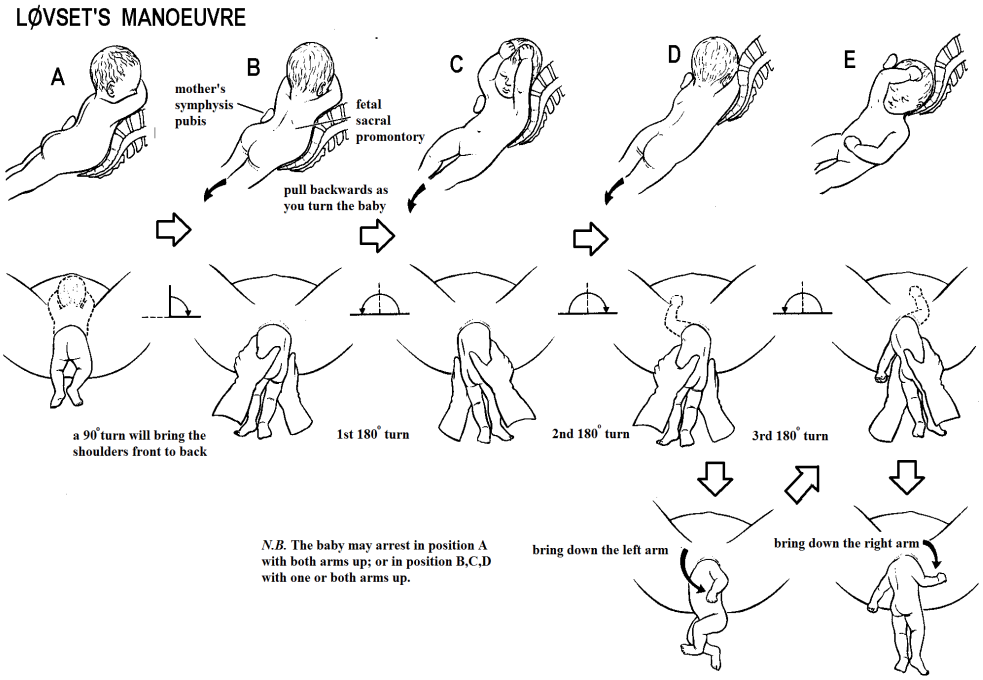

LØVSET'S MANOEUVRE
A
B
C
D
E
mother's symphysis pubis
fetal sacral promontory
pull backwards as you turn the baby
a 90° turn will bring the shoulders front to back
1st 180° turn
2nd 180° turn
3rd 180° turn
N.B. The baby may arrest in position A with both arms up; or in position B,C,D with one or both arms up.
bring down the left arm
bring down the right arm

Fig. 22-5 LØVSET'S MANOEUVRE for the delivery of the shoulders in a breech presentation. The bottom row of drawings shows a view from the patient's perineum. The top row shows the same stage viewed from her left. Remember: if you don't know which way to turn the foetus, keep the back anterior, so that it passes under the clitoris. Many practitioners merely wiggle the foetus one way then the other, pull, and try to find an arm, but this is a detailed manoeuvre. Practise it on a model.

If, in the extreme case, the foetus obstructs transversely (22-5A), start by turning the body through 90º, so that the back faces to the left. The left shoulder will then be above the symphysis, and the right shoulder above the sacrum (22-5B). With your 1st 180º turn (22-5C), bring the left shoulder under the sacrum. With your 2nd turn (22-5D), bring the right shoulder under the sacrum. The left arm will now be low enough for you to sweep it gently down. With your 3rd turn (22-5E), bring the right shoulder under the pubis; it will now be low enough for you to bring the right arm down.

CAUTION!
(1) These three 180º turns are in opposite directions, so that the back always passes under the clitoris, and the arm which started posterior always drags across the face. *The belly should never pass under the clitoris.*
(2) In the worst case, you start in 22-5A with both arms extended, so you have to begin with a 90º turn, followed by three 180º turns. If the foetus arrests at a later stage, with only one arm extended, you may only need 2 turns or perhaps only one.
(3) The first 2 turns release the shoulder which was arrested above the symphysis when you started it. The 3rd turn enables you to bring down the right arm.
(4) *Don't squeeze the belly or back:* you may rupture the liver, kidneys, spleen, or adrenals (which are relatively huge in the newborn). If you hold the chest, *take care not to compress the abdomen*.
(5) Remember that the upper part of the birth canal, in which the foetus is stuck, is directed backwards, so start by pulling the foetus dorsally relative to the mother.

If Løvset's manoeuvre fails to deliver the shoulders, it is usually a failure of technique. You may have to be a little firmer, or reach up a little higher to get the arm down. A broken arm will soon heal, so it is no disaster, and is better than letting the foetus die.

Later difficulties delivering a breech

CPD is the most important cause.

If the head is stuck above the brim, you are really in trouble. You may be able to draw it into the pelvis with the Mauriceau-Smellie-Veit manoeuvre. If this fails, the foetus will probably be dead, and the best treatment will be craniotomy (see below).

If the head has entered the pelvis and the Mauriceau-Smellie-Veit manoeuvre fails to deliver it, rotating the head in the pelvis may help. Stop struggling and think: What is the cause? If it is CPD, a quick symphysiotomy (21.7) may save the foetus.

If there is hydrocephalus, see 22.6.

If CPD is the cause or the cervix is not fully dilated, and you cannot deliver the foetus, apply gentle traction, and try to slip the cervix over his head. *If this fails, avoid harming the mother and allow the foetus to die*. While she is still in the lithotomy position, sedate the mother with pethidine 50mg and let the foetus hang for a while. The head will usually mould, or the cervix dilate, so that the foetus delivers in <1h. If this does not happen, traction with a bandage around the foetal legs and 1-3kg infusion bags as weights over the foot of the bed will succeed after some time.

If the above measures fail and CPD is severe, you may have to perform a CRANIOTOMY through the foramen magnum (unpleasant but effective: 21.8). Ask an assistant to pull down the body. Retract the anterior vaginal wall with a Sims' speculum and expose the back of the neck. Pick up a fold of the skin over the cervical spine with toothed forceps, and incise it transversely. Use curved Mayo's scissors to cut a tunnel under the skin up to the occipital bone, and push scissors into the head. Open the scissors and rotate them a few times to break up the brain compartments, withdrawing the scissors in an open position to enlarge the hole. Pull gently on the neck while the brain gradually escapes. Apply traction if delivery does not occur immediately.

If the dead foetus protrudes from the vulva, examine to feel if the cervix is fully dilated or not. If it is fully dilated, proceed directly to decompress the head with a craniotomy. If it is not fully dilated, apply traction. If this fails, perform a craniotomy.

CAUTION! Don't try to pull the head forcefully through the undilated cervix: you may cause tears which extend into the lower segment.

If the neck has been severed, but the head has retracted into the uterus, it will be difficult to find and remove. Use ultrasound and craniotomy equipment. *Don't leave the head to deliver:* its bones will splinter and perforate the uterus.

If the cord prolapses, manage as you would with a cephalic presentation: perform a Caesarean section, unless the cervix is fully dilated, and delivery is imminent. Cord prolapse is more common with breech deliveries, especially with a footling, but the foetal parts surrounding the cord are softer so that the cord is often not compressed completely.

Other methods for breech delivery

BREECH EXTRACTION uses your pulling forces, rather than the mother's pushing forces. It is a quick way of delivering a small breech, usually a 2nd twin. It may be indicated for:
(1) Delay with the 2nd twin.
(2) Foetal distress with the 2nd twin.
(3) Cord prolapse at full dilatation with a breech.
(4) A transverse lie in a second twin, following internal version.
(5) A dead foetus.

METHOD
The mother must be in the lithotomy position. Proceed as for an assisted breech delivery. An episiotomy is usually indicated. Hook the index fingers of each hand into the foetal groins and pull, preferably during a contraction.

When the umbilicus appears, hook out the legs by flexing the knees. Do this by applying lateral and dorsal pressure in the popliteal fossae, and by sweeping each leg laterally and downwards. Pull on the pelvis, keeping the back anterior. Pull posteriorly. A common error is to pull the foetus towards you, which is not in the axis of the birth canal. When you see the scapulae, hook out the arms.

N.B. If the arms are not across the chest, perform Løvset's manoeuvre.

Then push the head into the mother's pelvis from above. Then, if necessary, consider applying forceps to the aftercoming head.

The main difficulty is that the arms are more likely to be extended above the head, and the head is more likely to become deflexed. Løvset's manoeuvre and the Mauriceau-Smellie-Veit manoeuvre should solve these problems.

If the foetus is dead:
(1) Pull on the leg(s), if you can reach them, or
(2) Use a combined breech hook and crotchet (19-1).
Pass the blunt hook end of this instrument over an extended leg into the groin, and pull on that. If the foetus is macerated, the leg may be pulled off. If this is the case, turn the instrument round and hook the sharp crotchet end over the iliac crest. *Take care not to damage the birth canal!*

If the foetus is dead and its presenting part is high and it is a frank breech so that you cannot get a grip on the legs, then it is often easy to introduce a Foley's catheter with a large balloon in the foetal pelvis via the anus. Inflate the balloon with 30-50ml water and pull. The traction you can apply is considerable because the balloon is caught behind the pelvic bones.
Never do this with a live foetus: you will damage the rectum. A dead retained (perhaps macerated) 2nd twin can be delivered in this way; likewise if the mother cannot push (due to shock, eclampsia, or panic). There is no overwhelming hurry in these cases and if this does not work immediately, traction with a weight on the catheter will.

N.B. USE FORCEPS FOR THE AFTERCOMING HEAD.

Standard obstetric forceps, e.g. *Neville Barnes type:*
(1) *are not easy to use on the aftercoming head,*
(2) *are liable to be misused if they are in the labour ward at all,*
(3) *create the impression for midwifery students that a breech delivery is something that only doctors can do.*
Outlet forceps (Wrigley's) are not long enough when you really need them. If they will reach the head, they are hardly necessary in a breech delivery.

22.8 More malpresentations

A transverse lie occurs most frequently in multipara, and in mothers with polyhydramnios. Various causes may be present:
(1) Twins,
(2) A major degree of placenta praevia, or CPD,
(3) A congenital uterine or foetal abnormality,
(4) Premature rupture of membranes,
(5) A grossly abnormal pelvic brim,
(6) A fibroid,
(7) An ovarian tumour,
(8) Advanced extra-uterine pregnancy.

When labour is obstructed by a transverse lie, the lower segment of the uterus is particularly vulnerable, so *don't stretch it any more by doing an internal version in advanced labour with a dead foetus*: perform a destructive operation (21.8).

(a) Transverse lie

When ≥32wks pregnant, effect an external cephalic version (ECV, 22.7). This is safe provided there is no antepartum haemorrhage, no hypertension with a diastolic blood pressure of >100mm Hg, nor twins (22.10). If you fail, try again a week later. For obstructed labour with a transverse lie, see 21.5.

If labour starts with a transverse lie, before 30wks, or when the foetus feels as if it is <1½kg, spontaneous delivery of a 'folded-up' foetus may occur, although this foetus is unlikely to survive. There is also an increased risk of prolapse of the cord.

In the latent phase of labour, when the membranes are still intact and uterine contractions are not strong, effect an ECV to produce a cephalic presentation. If this is successful, and there are no signs of CPD, and the position is still unstable, rupture the membranes while an assistant holds the foetal head over or in the pelvis. If the mother is of low parity, start oxytocin IV. Check the foetal lie and heartbeat every 15mins, until the head is fixed in the pelvic brim.

If there is a small pelvis with an estimated true conjugate of <9cm (22.7), perform a Caesarean section.

If the foetus is alive and the active phase of labour has begun with intact or ruptured membranes, and the cervix is <8cm, perform a Caesarean section.
If the membranes are still intact, and you can feel a leg through the lower segment, you can deliver the foetus through a lower segment transverse incision. But if the membranes have ruptured, and especially if an arm has prolapsed, a de Lee incision (21.9) is better, because you can extend this into the upper segment as necessary.

If the foetus is alive and the cervix is fully dilated or nearly so, perform a Caesarean section.

If the foetus is dead, and the cervix is not yet 8cm dilated, perform a lower segment Caesarean section.

If the foetus is dead, with an impacted shoulder, and the cervix is >8cm dilated, and the uterus is not ruptured, perform a destructive operation (21.8).

(b) Brow presentation

You will feel:
(1) the foetal anterior fontanelle,
(2) the supra-orbital ridges, and
(3) the base of the nose.

N.B. Brow presentation is often missed:
(1) during labour. The head is high, but by the time it descends, the sutures and fontanelles by which it might have been diagnosed have become obscured by caput;
(2) at Caesarean section, until the typical moulding makes the diagnosis obvious.
Unless the foetus is premature, or the mother's pelvis is enormous, the foetus will not deliver vaginally.

If you diagnose a brow presentation in early labour, the pelvis is large, and the foetus is of normal size, the head may flex, and the foetus may deliver vaginally. You may be able to assist flexion by putting your hand through the cervix, pushing the head up and trying to flex it. But, if you fail to flex the head, if the membranes rupture, if there is no progress, or if there is any sign of obstruction, perform a Caesarean section.

(c) Face presentation

You will feel (1) the foetal eyes, (2) mouth and gums, (3) nose, and (4) chin.

Varieties are ('mento' refers to the chin):
(1) mento-anterior,
(2) mento-transverse,
(3) mento-posterior.

If the pelvis is large and there are no signs of CPD, allow labour to progress. The foetal position is most likely to be mento-lateral, and will probably rotate anteriorly and deliver spontaneously. You may be able to help by turning the foetus with your hand.

If the position remains mento-posterior, perform a Caesarean section.

If the 2nd stage is prolonged and the foetus is in the mento-anterior position, with $<^2/_5$ of his head above the pelvic brim, make a symphysiotomy (21.7) if CPD is mild, or perform a Caesarean section depending on your experience. Remember that the head moulds less in a face presentation. If CPD is significant, perform a Caesarean section in any case.

CAUTION!
(1) Remember the possibility of anencephaly. An anencephalic foetus often presents by the face, but usually delivers easily. You should be able to distinguish anencephaly, a face and a breech presentation vaginally, once the cervix is 8cm dilated; feel for the foetal brow and mouth. An ultrasound is useful.
(2) Use oxytocin with the greatest caution.
(3) *Never use a vacuum extractor!*

22.9 Prolapse and presentation of the cord

If the cervix is not well applied to the presenting part, the umbilical cord can prolapse when the membranes rupture, especially if the head is high, there is a transverse lie, a breech, a face presentation, or twins.

The cord is said to be presenting when it lies below the presenting part, inside intact membranes. Both prolapse and, to a lesser degree, presentation can obstruct the circulation in the cord, and so endanger foetal life. Other presenting parts press less firmly on the cord than does the head, but *don't let this delay you*.

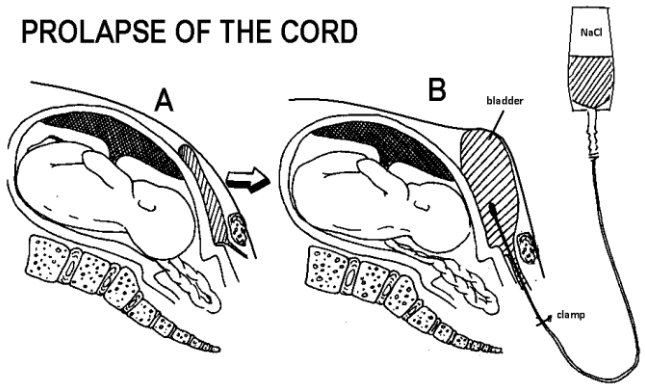


Fig. 22-6 TREATING PROLAPSE OF THE CORD BY FILLING THE BLADDER. A, the head pressing on the cord. B, to free the cord, fill the mother's bladder *via* a catheter, and clamp it.

(a) Prolapse
A routine vaginal examination immediately the membranes rupture spontaneously may diagnose a cord prolapse, but this is *not indicated* if:
(1) the gestation is <36wks and there are no contractions,
(2) the foetal head is well down (not $>^2/_5$ above the brim),
(3) the cervix is closed,
(4) you have not followed the advice in 22.4 on premature membrane rupture.

If you find a prolapsed cord, *don't take your hand out of the vagina!* Instead, push the foetal head (or breech) off the cord.

While you are holding the head, ask an assistant to insert a Foley catheter and fill the bladder with 500ml of saline, and clamp the catheter (22-6B). A full bladder will keep the head away from the cord and may inhibit the contractions of the uterus.

Listen to the foetal heart. It may still be beating, even if you can't feel the cord pulsating. Assess the foetal size, and try to exclude gross congenital abnormalities, particularly hydrocephalus.

N.B. A completely compressed cord, a complete abruption, a uterine rupture or the sudden death of the mother can still be compatible with an undamaged surviving foetus if delivery occurs within 15mins, provided the foetus was in excellent condition initially.

Remove your fingers, and apply a pad to the perineum, so that the cord remains in the vagina. Place mother in the knee-chest position (22-2D), and cover her embarrassing position with a sheet during transport to theatre. Perform a Caesarean section as soon as possible.

There is probably no time for a spinal, and sitting up and curving the back will probably endanger the cord circulation. The best option is probably to use ketamine. *Don't empty the bladder until you are ready to open the parietal peritoneum*. Then simply remove the clamp on the catheter.

Always perform a Caesarean section unless:
(1) the cervix is fully dilated and the head is only $<^2/_5$ above the brim and the patient is a primipara (unusual). If so, apply a vacuum extractor;
(2) prolapse of the cord complicates the delivery of a second twin with a cephalic presentation. If there is no CPD, you can usually apply a vacuum extractor, or effect a breech extraction preceded by internal version if necessary;
(3) the foetus is dead.

(b) Presentation of the cord
If you feel the cord vaginally when the membranes are intact, observe carefully for foetal heart changes which indicate cord compression. Put the patient into the head-down or knee-chest position, with the foot of the bed raised for 24h. This will nearly always allow the cord to rise above the head. Alternatively, <37wks, try ECV. Turning the foetus may draw the cord from under the presenting part. Otherwise perform a Caesarean section, unless the foetus is dead or too small to survive.

22.10 Multiple pregnancy

You can deliver most twins vaginally, and only perform a Caesarean section on the same indications as for a singleton pregnancy (21.5). Twins do, however, have problems:
(1) Labour is more often premature, which puts the foetuses at risk.
(2) Uterine inertia is more common; this prolongs the 1st and 2nd stages of labour, and makes postpartum haemorrhage more likely.
(3) Malpresentations are more common, especially with the second twin (22-7).
(4) Prolapse of the cord is also more common.
(5) When the 1st twin has been born, the 2nd may suffer as the uterus retracts and constricts the placental site.

As soon as you diagnose twins, plan for:
(1) Hospital delivery.
(2) Rest from 32-37wks at the shelters of a hospital. *Rest in itself has no proven benefit* but being near a hospital has. You will in any case have to admit a mother at 34-35wks to the mothers' waiting area.

The patient is more likely to become anaemic, so be sure she is taking iron and folic acid. Watch for gestational hypertension. She should not labour for longer with twins than she would with a single pregnancy.

Use oxytocin in the 1st stage with the greatest care. Oxytocin IV to restart the contractions after the delivery of the 1st twin is prudent and also later to prevent (or treat) postpartum haemorrhage.

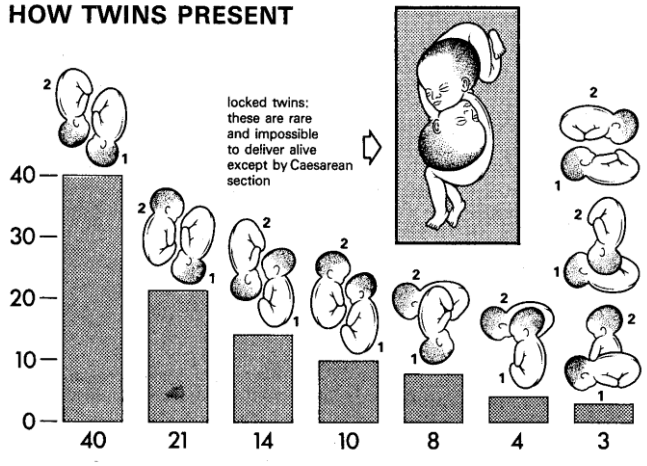


Fig. 22-7 HOW TWINS PRESENT: in 40% of cases both twins are cephalic; in 21% the 2nd twin is a breech; in 14% the 1st twin is a breech; in 10% of cases both twins are breeches. In all remaining cases one or the other twin, or occasionally both, are transverse.

Deliver triplets (or quadruplets) as you would twins. Expect the same problems as with twins, but expect them more often.

(a) Twins 1st stage
As soon as a woman is admitted in labour, determine the lie and presentation of the 1st twin by abdominal palpation. Confirm this by vaginal examination and ultrasound (38.3), and at the same time assess the pelvis. Manage as for a singleton pregnancy and use a partogram.
If there is delay during the active stage, apply the same criteria for the use of oxytocin and performing a Caesarean section. Remember the rare possibility of locked twins.
If the 1st twin is cephalic, or a fully flexed hip-breech, manage the 1st stage as an ordinary trial of labour, unless the foetus is very big, or the pelvis is very contracted.
If the 1st twin has a transverse lie, or is a footling (one or both thighs flexed and one extended at the hip), perform a Caesarean section, unless the foetus is very small (<1½). In this case, it may slip through an undilated cervix, and there is an increased risk of cord prolapse.

(b) Twins 2nd stage
Find an assistant who will be ready to look after the 1st twin while you deliver the 2nd. Be prepared for an operative delivery of the 2nd twin, and for a postpartum haemorrhage. Insert an IV line, and have oxytocin, misoprostol or ergometrine within easy reach.

Deliver the 1st twin as usual for a cephalic or breech presentation. This is usually no problem although the distended abdomen might interfere with the strength of the forces of expulsion. Oxytocin or a vacuum extraction may help. Nearly immediately after delivery, divide the cord between clamps, and then replace the maternal clamp by a ligature (22.11).

CAUTION! As soon as the 1st twin is born, look at the clock. Deliver the 2nd twin as soon as possible, but without undue hurry: 15mins is a reasonable time.

Feel, or ultrasound (38.3), the abdomen to find the lie, which you can make longitudinal if it is transverse or oblique. Version of a 2nd twin is usually easy, provided you do it without delay, immediately after the 1st twin has been delivered, while the membranes remain intact, and before uterine contractions restart. Then do a vaginal examination to feel how it fits the pelvis. Use 4 fingers or even your whole hand, instead of the usual 2; there will always be room for them immediately after delivery of the 1st twin. The presenting part of the 2nd twin is likely to be high: you may not be able to reach it with 2 fingers or you might just feel small parts through the membranes not being sure what they are. If you have a skilled assistant, ask her to do the abdominal palpation and keep the foetus in a longitudinal lie while you rupture the membranes. At this stage it is good to have an IV oxytocin infusion running because otherwise it may take long for the contractions to resume. Be proactive in rupturing the membranes to avoid the cervix from closing; the presenting part will come down after the stimulus of rupturing the membranes.

If the presenting part comes down, this is a sign of impending success. Sometimes this success has to be clinched by fundal pressure, a vacuum extraction or a breech/delivery extraction but this is bound to be easy.
If the head stays high, increase the speed of the oxytocin infusion and encourage the mother to push. A breech is better than a cephalic presentation if the preventing part stays high. A breech extraction is usually easy.
If you fear the cervix is closing and there is a high head (or a transverse lie), effect an internal version and extraction. It is much easier than you may think.
(Have long gloves ready somewhere for this eventuality.)

N.B. A vacuum extraction with a floppy cervix may be difficult especially if the 1st twin's umbilical cord gets in the way of the cup.
If the cervix closes, the diagnosis is then a retained 2nd twin. Active management with an oxytocin infusion, aiming at rupturing the membranes 3mins after delivery of the 1st twin, will almost always prevent this problem.

N.B. Many Caesarean sections are wrongly performed for retained (originally sometimes unrecognized) twins, often after the 2nd twin has already died. *It is too late to refer a mother for intervention at this stage!*

CAUTION!
(1) Try to know what the presenting part is before you rupture the membranes, although this is not always easy without ultrasound (38.3).
(2) The 2nd twin may be larger than the 1st. If you are motivated, a vaginal delivery will always be quicker than organizing a Caesarean section.

N.B. CPD is unusual with twins.

If ECV fails, *don't delay!* Rupture the membranes in the labour ward and immediately effect an internal version (see below) and deliver the foetus. Otherwise proceed immediately to a Caesarean section.

N.B. Internal version is dangerous if the membranes have been ruptured for some time.

(c) Twins 3rd stage
Manage this actively to minimize blood loss. Speed up the IV oxytocin infusion with the birth of the anterior shoulder of the 2nd twin, and then deliver the placentas by controlled traction on both cords. If bleeding continues or the uterus is lax, put 800µg misoprostol rectally, massage the uterus and express clots if it rises, till the tablets start working.

(d) Internal version (GRADE 2.2) is often possible without GA. It is kinder however under sedation.
Put the woman into the lithotomy position. Make sure the bladder is empty. Prepare the vulva with antiseptic as usual, and the abdominal wall also. Wait until she is relaxed between contractions, and then put your long-gloved right hand through the fully dilated cervix into the uterus, until you can feel the intact membranes.
Rupture them so you can get a grip on a foot. Palpate the abdomen with your left hand. Search for a foot, which you will recognize by its heel.

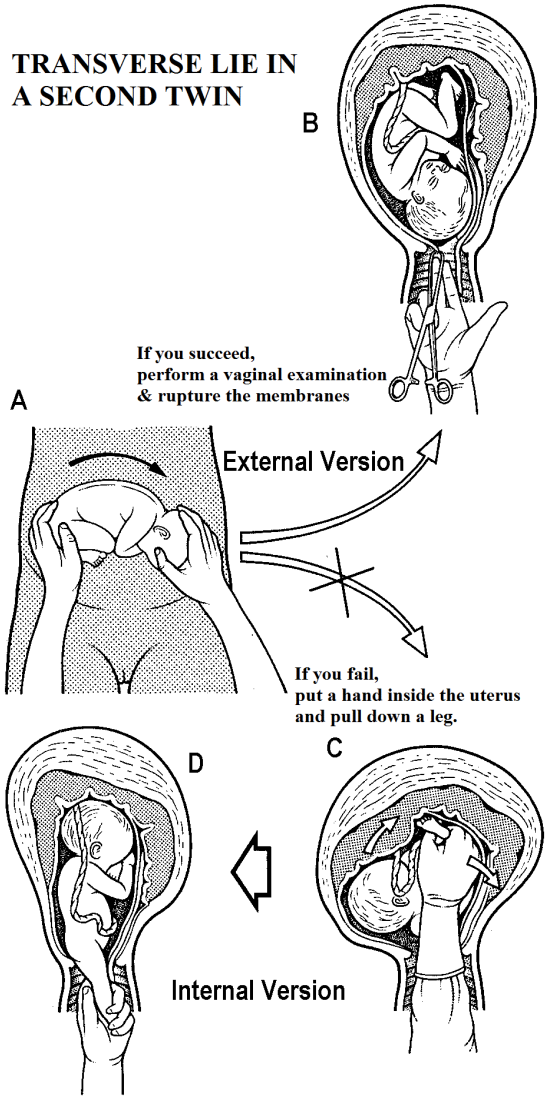


Fig. 22-8. IF THE 2nd TWIN IS TRANSVERSE, first try ECV. If you succeed, rupture the membranes. If you fail, bring down a leg so convert to a breech presentation. This is internal version. C,D, membranes ruptured.

If you find this difficult, work out which way round the foetus is lying, and then feel in the direction of the buttocks. Find a leg and follow this down. Use your other hand if this seems easier. When you have found a foot, bring this down. Hold the ankle between your index and middle finger, with your thumb on the dorsum of the foot. Gently pull the foot, so as to bring one of the legs over the pelvic brim, and down the vagina as far as you can, if possible as far as the vulva. The buttocks and other leg will follow.
At the same time, push the head upwards towards the fundus. Keep pulling on the leg in the direction of the floor. If necessary, squat to do this.
As more of the leg appears, hold it higher along its length. When the anterior buttock appears on the perineum, pull horizontally, and then upwards (breech extraction). When the buttocks are out, deliver the shoulders by Løvset's manoeuvre and the head by the Mauriceau-Smellie-Veit manoeuvre (22.7).

Occasionally, it is enough to pull down a leg into the vagina, and let the patient do the pushing (an assisted breech delivery); but *don't rely on this*, and be ready to assist her if she is uncooperative or exhausted.

CAUTION!

(1) Internal version is only for the 2nd twin with intact or recently ruptured membranes, during a delivery which you have been supervising. *It is not suitable for a retained 2nd twin*.
(2) Make quite sure you bring down a foot, and not a hand! *Don't, in exasperation, bring down any limb! It is not possible for the foetus to hide its feet so well that a committed doctor can't find them*.
(3) If you still don't know what is presenting, *don't waste time waiting for the presenting part to come down*. While you wait, the membranes will probably rupture spontaneously, and the presenting part may be an arm! Once the membranes rupture, labour will commence, and if the head remains high, especially with a transverse lie, the uterus is likely to rupture.

CAESAREAN SECTION is indicated if there is:
(1) A contracted pelvis with a diagonal conjugate of <11cm, or a true conjugate <9cm (21.4, 22.7).
(2) A major malpresentation of the leading twin, such as a transverse lie, locked twin or footling breech.
(3) Lack of progress in labour, not amenable to oxytocin after artificial rupture of membranes.
(4) A 2nd twin with a transverse lie which you cannot correct because the membranes have long been ruptured.
(5) A uterine scar, *e.g.* from previous Caesarean section: this is only a relative contraindication to vaginal delivery.

(e) Difficulties with multiple pregnancies

If there is delay in the 1st stage, you can use oxytocin, provided there is no obvious CPD. Rupture the membranes of the 1st twin. Try to deliver the 2nd twin within 15mins of the 1st, or preferably less.

If, after the delivery of the 1st twin, you feel the head or breech of the 2nd twin, but the cervix is only 7-8cm dilated, assistance with manual fundal pressure will help very well.
Rupture the membranes and make the patient push. The cervix will dilate again, as soon as the presenting part of the second twin comes down. Contraction of the cervix will not at first delay delivery of the 2nd twin, and is no reason for delaying rupture of the membranes.

If there is heavy bleeding before delivery of the 2nd twin, the placenta of the 1st foetus has probably separated. Deliver the 2nd twin quickly, and then deliver both placentas together.

If either twin is a breech presentation and the patient pushes well and the breech descends well, it will be an assisted breech delivery. If there is foetal distress, delay, or poor pushing, don't hesitate to apply more traction, and turn delivery into a breech extraction (22.7).

22.11 Primary postpartum haemorrhage (PPH)

Postpartum haemorrhage (PPH) is caused by:
(1) Most importantly, bleeding from the placental site after the placenta is delivered because the uterus fails to contract.
(2) Retention of all or part of the placenta.
(3) Lacerations of the genital tract: rupture of the uterus, cervical lacerations, lacerations of the upper vagina, and vulval lacerations, especially near the urethra and clitoris.
(4) Occasionally, a clotting defect, especially disseminated intravascular coagulation (DIC, 3.5), which produces a fibrinogen deficiency.
(5) Faulty suturing technique during a Caesarean section.

Aim to stop the bleeding, resuscitate the patient, monitoring her carefully. Alert your anaesthetist. *Act always on the side of caution*. Since bleeding most often occurs from the placental site, your first objective must be to expel the placenta together with any residual clots. If you have achieved this but there is still bleeding, consider the other causes.

Sometimes the only way to keep the uterus contracted (you can feel it) is to massage it. This may need only 15mins till the misoprostol takes effect, but sometimes 1-2h. This can be quite painful for the patient.

(a) Ergometrine IV or IM will give, as it works, a sustained contraction of the uterus. Therefore *don't use it before delivery of the foetus*. If you do use it before delivery of the placenta, there is more chance (2-3%) of a disconnected placenta being trapped behind the closed cervix. Ergometrine might also cause an eclamptic attack in women with pre-eclampsia, which may be masked by a drop in blood pressure due to bleeding.
Ergometrine can of course be very useful especially for the poorly contracting, empty, bleeding uterus after oxytocin proves ineffective. It is not very stable (especially under the influence of light), so store it in a dark place *(often not done)*. Ergometrine kept in labour wards and theatres in a drawer for years will not work. Moreover, it might precipitate vomiting, which is particularly dangerous if the patient is unconscious and not intubated, *e.g.* if you are using ketamine.

(b) Misoprostol is very useful for incomplete miscarriage, priming the cervix and induction. Overdosing is dangerous after 25wks' gestation, but *it is not a problem in the treatment or prevention of PPH*.

(c) Oxytocin is probably the best drug for PPH in most circumstances. If there is extra risk or oxytocin is not working or unavailable, use misoprostol as well or instead.

For home deliveries by traditional birth attendants (TBAs), PPH is the severest risk. Delivery in a health institution would be better. If that is not feasible and there is some TBA training, the preventive use of misoprostol (3x 0.2mg, *i.e.* 600µg orally, directly after delivery of the foetus) *without* controlled cord traction might save lives and is cost-effective.

In a clinic, misoprostol (800µg rectally) probably should be added prophylactically to the oxytocin if there are extra risks of PPH, or added when oxytocin turns out not to be effective enough. A frequent side-effect is shivering.

(d) DIC (3.5) is probably the commonest cause of a massive PPH when the uterus is empty and is satisfactorily contracted. It is the commonest clotting defect, and is an important and mostly preventable cause of maternal death. It is uncommon after a normal delivery, and is more common after abruption (20.12), an obstructed infected labour (21.4), amniotic fluid embolism, (pre-)eclampsia, sepsis (22.4), or an intra-uterine death (20.4).
Try always to have fresh frozen plasma (FFP) in stock. This has the clotting factors which are practicable for you to stock.
Try to prevent shock by infusing normal saline, FFP and stored blood and by compressing the uterus bimanually or even compressing the aorta against the spine just above the uterine fundus.
In time new clotting factors will be made in the liver, *but this will not help if severe bleeding continues*. So, if bleeding continues, you need to transfuse fresh blood.
But when you are in the above situation you don't know if your management is going to work, so set the collection of fresh blood in motion and also organize the theatre staff in case you have to operate. *This is a major alarm.* Remember there may be a big danger of HIV transmission when you use FFP and more so when you use fresh blood, *so you should use it only when you absolutely have to*, but *don't hesitate when the indication is clear!*

If your patient has a complete abruption during labour, deliver the foetus as quickly as possible, very preferably, vaginally. Pulling the foetus with the help of a weight, rope and forceps on the skin of its head can save critical time.
Stay with the mother because your adequate management in the first 5mins after delivery can easily make the difference between life and death. She might have just enough clotting factors left, but if the uterus takes 10mins to contract properly she will probably lose a critical amount and bleed uncontrollably. Use oxytocin and misoprostol (use it rectally 10mins before the expected time of delivery) and massage the uterus.

N.B. If with a complete abruption, the patient's situation is so very serious that you are thinking of performing a Caesarean section, you will run into problems because the incision will never stop bleeding. If, on the other hand, the situation is not so desperate, you will have time to deliver the foetus vaginally. *In other words, if you can perform a Caesarean section, it is not needed; if you do perform one, it won't help you.*

If you do have to give fresh blood, you will find it helpful if all your permanent medical, nursing and ancillary staff know their own blood groups, and can be called upon in an emergency. Perhaps there are more people in your town who are prepared to be tested for HIV regularly. You probably need fresh blood only once a year but then its availability may save a life. Occasionally, the husband has the same blood group or has group O Rh-ve. (If the mother is HIV+ve and he also, this added problem would also be removed!)

N.B. Make sure you have access to collection bags with citrate day and night.

(e) Preventing PPH before labour
The following are RISK FACTORS which make it more likely that a woman will have a PPH and so should deliver in hospital.

(i) **Identifiable during pregnancy:**
(1) APH in this pregnancy.
(2) PPH, or a retained placenta, in a previous pregnancy.
(3) Multiple pregnancy or other cause of extremely distended uterus.
(4) Combination of previous Caesarean sections and a placenta low on the anterior wall.
(5) Placenta accreta, percreta or increta.

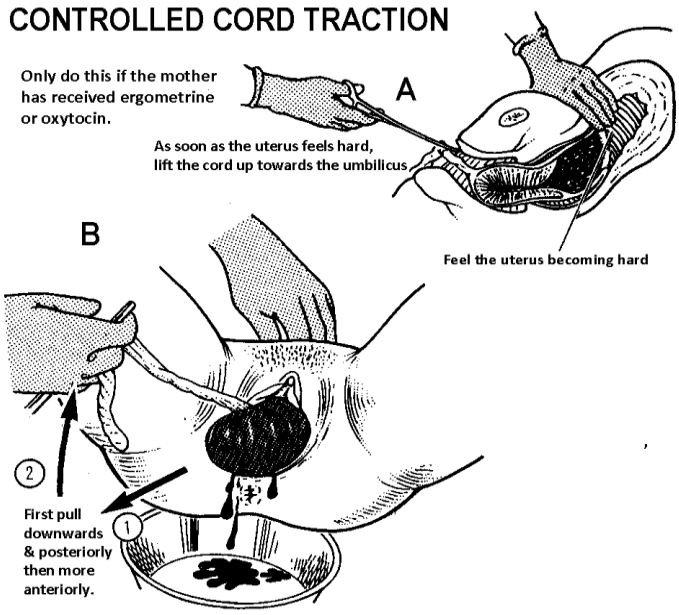

CONTROLLED CORD TRACTION
Only do this if the mother has received ergometrine or oxytocin.
A
As soon as the uterus feels hard, lift the cord up towards the umbilicus.
B
Feel the uterus becoming hard
2
First pull downwards & posteriorly then more anteriorly.
1

Fig. 22-9 CONTROLLED CORD TRACTION.
As soon as the uterus is contracting firmly from the action of oxytocin or ergometrine, grasp the uterus, push it upwards towards the umbilicus and gently pull on the cord, first downwards and backwards, and then more anteriorly as the cord comes out.

N.B. Grand multiparity (>4 children) is not really a risk factor for PPH, but the consequences of haemorrhage in an older multipara are much worse than with a 1st pregnancy.

(ii) **Identifiable during labour:**

(1) Prolonged, especially infected, labour.
(2) Anaesthesia, using ether, halothane or spinal.
(3) A full bladder.
(4) Placenta praevia.
(5) Placental abruption, mainly because this causes a clotting defect, but also because there is already much blood lost in the uterus (concealed bleeding) and there are few clotting factors left to prevent even more loss.
(6) A clotting defect, especially DIC (3.5).
(7) Incomplete expulsion of the placenta.

CAUTION! PPH may occur without there being any risk factors: it is best to have an oxytocin infusion running routinely after Caesarean section.

PREVENTING PPH DURING LABOUR

Treat every mother, especially those with risk factors, with 5IU oxytocin IM. (Ergometrine 0·5mg IM is not such a good option because it can cause retention of the placenta (see below) but it is better than nothing.) Oxytocin will work quicker if you use it IV, but there may be nobody around to monitor the patient.

Routinely use oxytocin IM as soon as the foetus is born, *and you are sure there is no twin still in the uterus*. Then (unless the mother is HIV+ve), after 3mins' delay, clamp the cord to increase the foetal iron stores and deliver the placenta by controlled cord traction.

If there is a risk factor for PPH, set up a perfusion of IV saline with a large bore cannula before the patient reaches the 2nd stage. When the foetus and the placenta have been delivered, add 20IU oxytocin IV in 500ml at 30 drops/min for at least 3h. Also, for patients seriously at risk, insert 800µg misoprostol rectally as soon as the placenta is delivered and massage the uterus for a contraction. This is likely to help because it works via different pathways. If misoprostol is not available, use ergometrine 0·5mg IM and massage the uterus.

As soon as the placenta is delivered, check to make sure:
(1) it is complete and that no lobes have been left behind (see below), and
(2) there are no obvious large lacerations in the birth canal.
Keep the mother in the labour ward, and monitor her for at least 1h, before returning her to the ward. Check that the uterus is well contracted and note any bleeding.

Ideally, you should never apply controlled cord traction before the uterus has hardened under the effect of an oxytocic drug; so, if you don't have any oxytocics, you should not do it. In practice, little harm results if there are already signs of placental separation (lengthening of the cord and hardness of the uterus).

Although it is a very valuable procedure, there is a risk, particularly if you do it incorrectly, that you may invert the uterus.

CAUTION! Don't squeeze the uterus to try to get the placenta out. This is very painful.

(f) Controlled cord traction

Unless the mother is HIV+ve, clamp the cord after a delay of 3mins. Then, as soon as the uterus is contracting firmly from the action of oxytocin (or ergometrine), put your left hand on the abdomen, above the pubic symphysis, and turn your palm towards the head. Grasp the uterus. As soon as it feels hard from the effect of the oxytocic, push it upwards towards the umbilicus (deliver the placenta more by pushing the uterus up than by pulling on the cord).

Wind 2-3 loops of cord round your index finger and gently pull on the cord, first downwards and backwards, and then more anteriorly as the cord comes out. Very, very rarely you might feel an inversion of the uterus originating at this stage if you pull without a contraction.

RESUSCITATION FOR PPH

As soon as you are called to a patient with PPH, quickly call an assistant: at least 2 people are needed.

Start vigorous resuscitation. What is the state of the peripheral circulation? How much blood has been lost? Is it clotting normally in the receiver used to collect it? It may clot to start with, and then stop clotting later. What has been done so far?

Monitor the volume of blood loss, the warmth of the peripheries, pulse and blood pressure, and the urine output.

If she is still bleeding: is the uterus still contracted? Is the placenta out and complete? Is the bladder empty? Does she have any obvious lacerations of the vulva, vagina or perineum? Could the uterus be ruptured?

If she has stopped bleeding, is the uterus well contracted?

CAUTION! Make sure that one nurse is allocated solely to observe and monitor this patient, until bleeding has stopped, and her condition is stable. Poor supervision is an important cause of death in PPH.

(g) PPH with a retained placenta

In low-income countries, retained placenta affects c.0·1% of all deliveries, but has a case fatality rate up to 10%. There are large regional differences: for example, the incidence is very high in Papua New Guinea.

Some placentas are simply trapped (the incarcerated or trapped placenta) behind a closed cervix; the use of ergometrine promotes this situation. Some are adherent to the uterine wall but usually easily separated manually (placenta adherens), and some have grown into the wall in a small area, needing manual or instrumental removal usually in piecemeal fashion though blood loss is controllable. Others are pathologically invading the myometrium (placenta accreta, increta, percreta) over a large area, needing ligation of uterine blood vessels, or even hysterectomy. These last cases are quite rare and mostly seen during Caesarean section because they are often related to more than one previous Caesarean section combined with placenta praevia.

Active management of the 3rd stage limits blood loss and shortens this stage but after 1h the number of retained placentas is similar whether there was active management or not; the same applies to the use of misoprostol.
It seems that the placenta becomes separated by contractions *at the place of the placental insertion*. Using ultrasound (38.3), you can see that the uterus is thin behind an adherent placenta but thick everywhere when the placenta is merely trapped.

If the placenta is retained for <1h, try to make the uterus contract.
(1) If you have not used oxytocin, use it now.
(2) If this fails to stimulate a contraction, gently massage the uterus for a contraction.
(3) Remove the placenta by controlled cord traction, as soon as the uterus is contracting firmly. It should deliver immediately.
(4) You can reduce the need for manual removal by c.20% by the use of intra-umbilical vein oxytocin (50IU in 30ml saline). This will stimulate exactly only that uterine area where a massive contraction is wanted. An even more effective alternative is 30ml saline with 4 misoprostol tablets (800µg) dissolved in it. Cut the cord 5cm in front of the vagina or open a vein in the cord near the introitus with a scalpel and thread a Ch10 gastric tube in the direction of the placenta till you feel resistance. Withdraw it 5cm to allow for branching of the vein and inject the 30ml with either oxytocin or misoprostol (Pipingas technique) while preventing back flow by clamping the cord.

If the placenta is retained for >1h, this is an indication for manual removal. Of course, if there is bleeding (placenta partly separated or incarcerated), something should be done fast, such as manual removal with IV pethidine with diazepam or ketamine in the labour ward.
Before doing a formal manual removal, perform a vaginal examination, and see if the placenta is stuck in the cervix, from which you can remove it quite easily.
While preparing to do a manual removal, concentrate on:
(1) continuing resuscitation,
(2) keeping the uterus contracted with 20-40IU oxytocin IV, and
(3) if the oxytocin or misoprostol does not work against bleeding, gently massaging the uterus for a contraction.

(h) Manual removal of the placenta (GRADE 1.5) can be either fairly easy when it is merely trapped behind a closed cervix; rather difficult, needing removal because it is adherent or locally invading the uterus; or impossible (the clinical definition of placenta accreta), when most of or the entire placenta has grown in the uterus.

It is usually best done in the labour ward (which must be equipped for anaesthetic resuscitation) 30-60mins postpartum rather than in the theatre, which usually requires moving the patient and will cause delay. *You will need stirrups to maintain a lithotomy position, and a good light*.
Before you start, set up IV saline or Ringer's lactate, if necessary with 2 IV lines.

If an infusion of oxytocin IV is already running, stop this just before manual removal to allow the cervix to relax, so that you can get your fingers inside the uterus.

Ketamine is safest; *don't use sedation in a hypotensive patient.* Use aseptic procedures.

Hold the cord in your right hand. Put the tips of the fingers of your left hand together, and introduce it into the upper part of the vagina. If the placenta has stuck in the cervix, grasp it and slowly remove it. Now let go of the cord, and place your right hand on the fundus (over the towel). Prevent the fundus from being pushed up, as you gradually work your way into the uterus with your left hand. Feel for the part of the placenta which has already separated, and push your fingers between it and the wall of the uterus. Gently separate the placenta from the wall of the uterus with a slow sawing movement, with the side of your hand.
CAUTION! All this time keep your right hand pressing on the fundus, so as to bring the uterus as close to your left hand as you can. *If you don't do this, there is a danger you may lacerate the placenta*.

As soon as the placenta has separated, grasp it with your left hand, remove it, and ask your assistant to inspect it. Meanwhile, whether it looks complete or not, explore the uterus for any pieces left behind, and remove them. Only now remove your right hand from the uterus.

Finally, restart the IV oxytocin infusion, and wait for the uterus to contract. As it begins to do this, remove your hand. As you do so, check that the lower segment is intact. Before you finish, make sure that there are no other sites of bleeding; so explore the uterus as described below.

INSPECT THE PLACENTA to see if part of it has been left behind, or a vessel is running off one edge of it. In either case, you must remove the missing piece of placenta. For small pieces left in, suction using a 12mm Karman cannula may be the solution; *don't use a small sharp curette*.

If the bleeding continues, apply BIMANUAL COMPRESSION (22-10A). Put your left hand into the upper vagina. Put your right hand on the abdomen, and use it to push the fundus down onto your left hand. Press for at least 5mins, and then review the situation. Continue IV oxytocin 20IU in 500ml and infuse it at a rate that will keep the uterus contracted. Continue for at least 12h, using more IV fluid and oxytocin as necessary, or use 800µg misoprostol 4hrly rectally. Monitor the mother carefully. Treat her with antibiotic prophylaxis. Keep her in hospital for at least 5 days, because of the higher risk of puerperal sepsis, particularly endometritis. Check the Hb level.

(i) PPH after placental expulsion
Failure of the uterus to contract is the most important cause of PPH, so aim for an empty, well-contracted uterus.

Feel the fundus. It should be hard and round, and below the umbilicus. If it is soft and difficult to feel, it may be relaxing. Massage it to make it contract. This may expel

some blood and clots. If the bladder is full, catheterize it. Use misoprostol (or if this is unavailable, ergometrine 0·5mg) in addition to the oxytocin infusion.

Resuscitate with 2 IV infusions of *warmed* saline or Ringer's lactate. To the first, add 20IU oxytocin. Infuse this fast, until the uterus contracts well. Then slow it to 40 drops/min. Continue this for 2h afterwards. Use the second IV infusion to replace the blood lost with 3 times as much saline. Aim for a systolic blood pressure ≥100mm Hg.

Inspect the placenta for missing pieces with great care, if you have not already done so. If a piece is retained, it will have to be removed. If there are any obvious perineal lacerations, suture them.

If bleeding stops, continue to monitor, resuscitate if necessary, and to use IV oxytocin.

If the blood fails to clot normally, try FFP. It needs fine judgement to decide if you need to use blood or even fresh whole blood. Consider any pre-existing anaemia, availability of FFP and blood, the patient's age and fitness, and risks of transfusion in your environment.

N.B. A young fit person can usually handle the loss of 2L blood if the volume is replaced by saline.

If the blood fails to clot in the receiver as it comes from the vagina, there is probably DIC. If necessary, you can confirm this with a bedside clotting test (3.5), but *don't let this delay you!* Infuse 3-4 units FFP rapidly and also infuse the red cells needed: this will probably mean using fresh whole blood. The clotting defect will probably correct itself within 6h of delivery of the placenta, so if you can only keep the patient alive during this period, she will probably live. She might need (peritoneal) dialysis a few days later. These patients are at risk of clotting too much after they have been cured of clotting too little.

In circumstances where it is routine to use heparin during or after operations, you should use it for these women once they stop bleeding. If you normally don't use heparin, early mobilization is important.

If bleeding continues with an empty poorly contracted uterus, despite oxytocin, increase the rate of infusion. If this fails, there may be a piece of placenta left inside, or, much less commonly, a ruptured uterus. An ultrasound (38.3) can help but it is not easy to exclude with confidence a retained part of placenta, so if you are in doubt, suction the uterus. With a ruptured uterus, there will nearly always be blood in the abdomen which you can diagnose by ultrasound (38.2l), a bloody abdominal tap, shifting dullness or uterine digital exploration.

If bleeding continues with a contracted uterus, explore the genital tract for lacerations, from the fundus to the clitoris. If you find large lacerations, suture them.

N.B. Repairing cervical lacerations needs good light, an assistant and experience; it might be safer to use a compression pad.

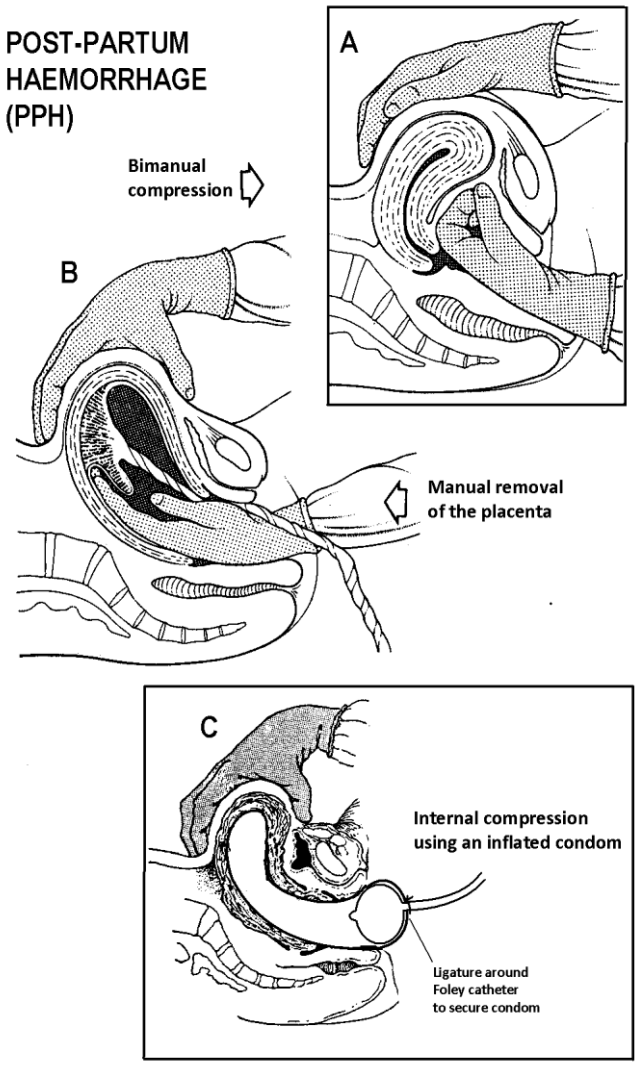


Fig. 22-10 POSTPARTUM HAEMORRHAGE (PPH).
A, bimanual compression of a bleeding uterus between a fist in the vagina and a hand on the abdominal wall. B, manual removal of the placenta. Gently separate it from the wall of the uterus with a *slow* sawing movement with the side of your hand. C, internal uterine compression, best by use of an inflated condom, is only occasionally necessary. Its main use is to control bleeding from the cervix, and it is much less effective in controlling bleeding from the uterus. Much the best way to do this is to use drugs to make it contract.
C, kindly contributed by Robert Lange.

(1) **Exploration of the cervix** (GRADE 1.1)

METHOD. Use sterile precautions. Catheterize the bladder. Use ketamine. Use the lithotomy position, get a good light, and find a Sims' speculum or one which is wider, and an assistant to help hold it. Wipe out the blood in the vagina with gauze swabs. Look at its walls. Check that the vaginal wall, and the perineal and vulval skin are intact.

To inspect the cervix, use 2 swab-holding forceps. Grasp the front lip of the cervix with one of them. Pull the cervix gently down, and look for lacerations on it. If there are none, use the 2nd forceps to pull down the next portion of cervix, and look at that. Continue round the cervix in this way, looking at every part (22-11).

Then put your hand into the uterus and carefully feel its front, sides, back, and fundus. Feel for a rupture of the uterus (21.17), and for any pieces of adherent placenta.

If there are lacerations on the perineum, vagina, or cervix, suture them. Only suture a cervical laceration if it is causing arterial bleeding. A venous ooze is not a sufficient indication for suturing, which itself can cause new bleeding.

If there are multiple small lacerations rather than one large one which you can easily suture, or there is a steady ooze, pack the vagina.

If there is a retained piece of placenta, use suction. If this fails, scrape it off with your fingers. *Don't persist if you cannot get it all off*, as this will be due to an abnormally adherent placenta.

If you find a rupture of the uterus and bleeding is severe, apply bimanual compression or compress the aorta against the spinal column just above the uterus, until you can get someone to organize for an immediate laparotomy (21.17). *Don't then leave the patient without continuing the compression!*

If bleeding persists *despite all other measures* when you have used oxytocics properly, sometimes the only way to keep the uterus contracted is to massage it for hours. This can be quite painful for the woman.

(2) **Balloon tamponade of the uterus and vagina** (GRADE 1.2)

Packing is messy and time-consuming. If there is a steady ooze, blood is scarce, and HIV common, packing may save a mother's life. In practice it is very useful as a near-last resort, before tying the uterine arteries or removing the uterus. It is much less effective in controlling bleeding from the uterus, than from the cervix. Much the best way to do this is to use oxytocin and misoprostol to make the uterus contract.

METHOD. Use sterile precautions. Use the lithotomy position. Pack the uterus and vagina with a condom, attached to tubing or a Foley catheter and filled with 1L water (22-10), or occasionally 2 such condoms. You can use another such condom in the vagina to compress the cervix. This is far more effective and cheaper than using sterile gauze, which you may have difficulty getting through the cervix.

CAUTION!
(1) Be sure to compress the whole genital tract from the fundus to the introitus.
(2) *Don't only compress the vagina*, because bleeding will continue invisibly in the uterus, the only sign of which may be increasing hypovolaemic shock.
(3) If you do use gauze, tie it in one long piece to prevent bits getting lost.

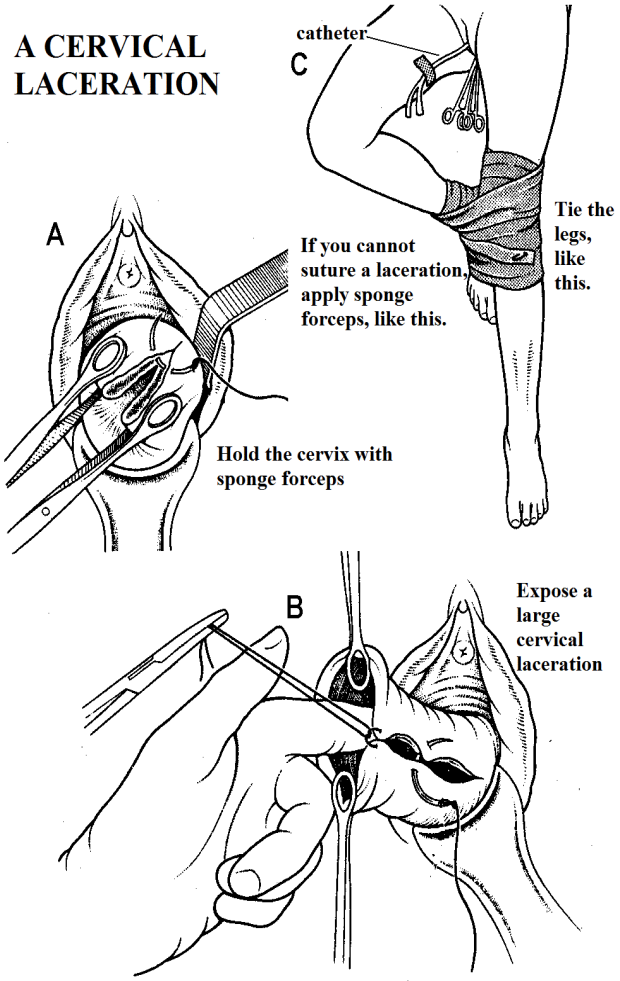


Fig. 22-11 REPAIRING A CERVICAL LACERATION.
A, search all round the cervix with ring forceps, until you find the injury. B, suturing a longer laceration. C, if midwives cannot control bleeding they should be asked to apply ring forceps, tie the legs together, and refer the patient like this.

With a balloon inflated in the uterus, it will be difficult to pass urine, so catheterize the bladder.

Continue monitoring and infuse IV fluid or blood as necessary. Remove the compression after 24h.

(j) Difficulties with PPH
If there is severe bleeding and there is going to be some delay, compress the aorta. Stand on the patient's left and feel for the left femoral pulse with your left hand. Clench your right fist and, with your index finger level with the umbilicus and your knuckles in the line of the spine, press gently and firmly through the abdominal wall so as to compress the aorta against the spine. You will feel it pulsating. Press so that you no longer feel any pulsations and obliterate the femoral pulse. If necessary, this method can be kept up for hours, while the patient is referred or while preparations for surgery are being made, changing hands and workers as required. If the legs become numb, allow a little blood to flow through them.

If you cannot get your whole hand through the cervix to perform a manual removal (not uncommon if a lot of ergometrine is used shortly before the manual removal is done, or there has been a long delay), you are in difficulty. Avoid this problem, if you can, by using IV oxytocin, rather than ergometrine, and by discontinuing it just before manual removal. Try to get one or two fingers through the cervix, and push the fundus well down with your other hand. Usually, the cervix relaxes gradually so that, if you are slow and gentle, you can put your whole hand into the uterus.

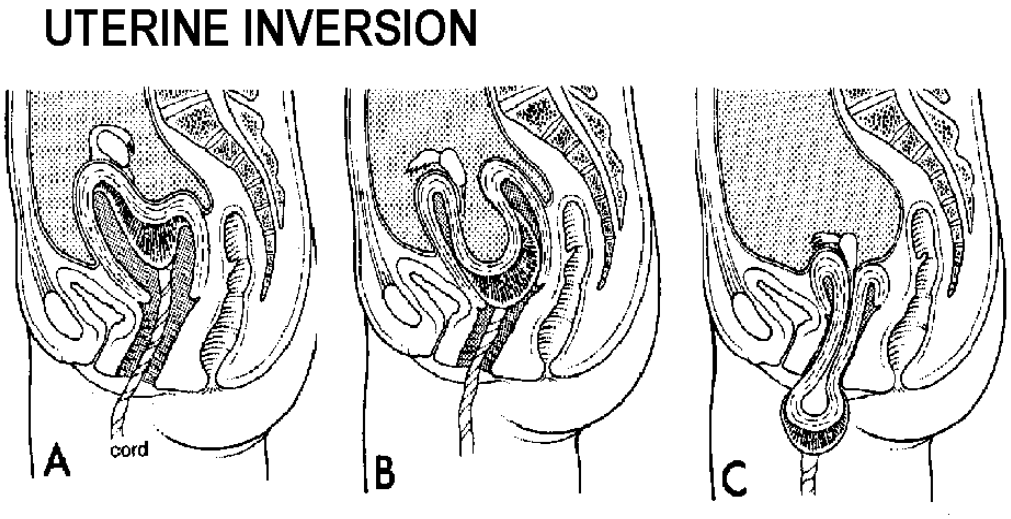


Fig. 22-12 INVERSION OF THE UTERUS.
A,B,C, increasing degrees of inversion. If this happens spontaneously, or as a complication of controlled cord traction, immediately push it back. If there is any delay, replacing it will be much more difficult.
After Bonney V. Gynaecological Surgery Baillière Tindall 2nd ed 1974 Fig 431, permission requested.

Long forceps to remove the placenta piecemeal are an option; keep your non-dominant hand on the fundus to prevent perforation: you will feel the forceps.
This method can be combined with suction via a large (12mm) Karman cannula.

If the placenta seems abnormally adherent to the uterus (placenta accreta), remove what you safely can piecemeal, without perforating the uterus, and leave the rest. This is not a problem as long as the uterus contracts sufficiently afterwards to obstruct the blood flow to the relevant areas. *Avoid sharp curettage*: the myometrium might be very thin at the point of adherence and there is a high risk of perforation.
The placenta which you have to leave will be slowly absorbed. There is serious risk from sepsis and secondary postpartum haemorrhage. Continue IV oxytocin for 48h, then stop if all is satisfactory. Treat with prophylactic chloramphenicol and metronidazole. Monitor carefully in hospital.

N.B. Placenta accreta over a large area will often need a hysterectomy; suspect this if there have been several previous Caesarean sections.

If bleeding continues from an empty uterus, *despite all the above measures*, try infusing oxytocin 40IU in 500ml saline fast IV with repeated doses of ergometrine 0·5mg IV. Try prostaglandins if you have them.

B-LYNCH UTERINE SUTURE (GRADE 3.3)
If you cannot control bleeding despite the measures outlined, open the abdomen as for a Caesarean section. Apply a special type of tourniquet (brace suture) around the uterus: open the visceral peritoneum where the bladder ends and the uterus starts, reflect the peritoneum and push the bladder down bluntly. Insert a #2 long-lasting absorbable suture (22-13) on a large Colt's needle (4-7O) 2-3cm lower than where you would normally open the uterus for a Caesarean section.

Go through the anterior uterine wall 4cm from the lateral margin on the right side. Then loop the suture over the fundus and insert it through the posterior wall across the midline. Ask your assistant to squeeze the uterus and pull the suture tight. Continue with the same suture, looping over the fundus on the left, and pass it again through the anterior uterine wall, and knot it tight. Check if bleeding continues. (Get an assistant to check under the towels.)

If bleeding continues and the condition is deteriorating, make a tourniquet from any rubber sling or catheter and push it as low as possible around the uterus. Push it lower than the fimbria and tie it with a half-knot tight, putting a forceps on the knot. This will give you time to resuscitate the patient, finish essential suturing, and organize subsequent care without further blood loss. It is even possible, if you don't have blood or the right expertise, to refer a patient on with the tourniquet *in situ*.

Certainly, it will make a hysterectomy later a much less bloody and dangerous procedure.

N.B. A tourniquet, put on in this way, stops blood supply to both the uterine *and* the ovarian arteries. This means that the uterus and ovaries will necrose if the tourniquet is left on for >3-6h.

If you place the tourniquet so it only stops blood supply in the uterine arteries, sparing the ovarian vessels, this might still control the bleeding without causing the ovaries to necrose.

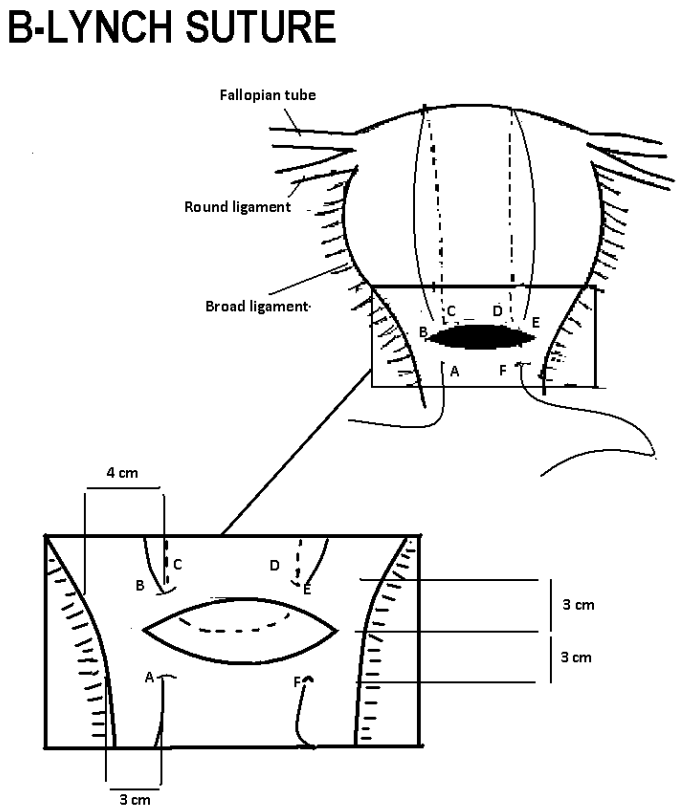


Fig. 22-13 B-LYNCH UTERINE SUTURE.
A, introduce the suture here and exit at B. Loop the suture over the fundus to re-enter the uterine cavity posteriorly at C, directly behind point B. Pull the suture tight, and then pass it through the posterior uterine wall to come out at D. Loop it back over the fundus, and enter the uterine cavity anteriorly again at E, coming out at F. Then tie the suture tight to compress the uterus.
After B-Lynch C et al, The B-Lynch surgical technique for the control of massive postpartum haemorrhage: an alternative to hysterectomy? Five cases reported. Br J Obs Gyn 1997;104(3):372-5.

To do this, you have to push your tourniquet through the broad ligament just lateral to the uterus near the cervix from front to back, then go round behind the uterus and do the same on the other side from back to front and tie.

CAUTION! Do it bluntly with a small artery forceps first, and then pull the catheter through, avoiding many distended veins in this area.

Alternatively, control the haemorrhage with the tourniquet, and then formally ligate both uterine arteries (22-14).

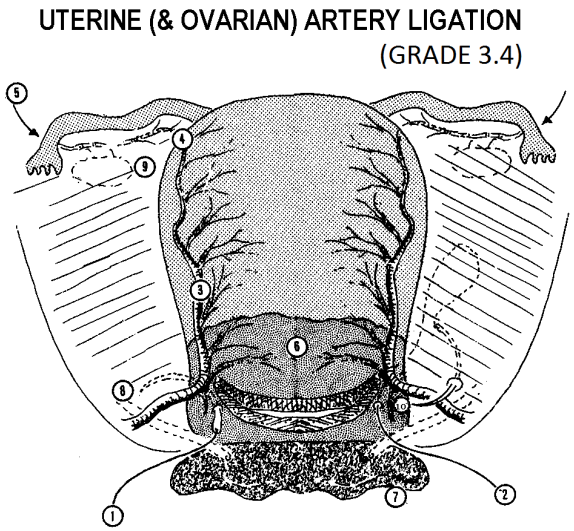


Fig. 22-14 LIGATING THE UTERINE ARTERIES.
First suture on the right side: pass the needle taking a good bite of the uterine muscle through to the back. Following the same procedure, then pass the suture back through the broad ligament on the left side. You can place additional sutures higher up if necessary.
(1) uterine artery. (2) anastomosis between uterine and ovarian arteries. (3) ovarian artery in the infundibulo-pelvic ligament. (4) bladder reflected off the lower uterus. (5) bladder. (6) right ureter. (7) left ureter. (8) right ovary. (9) inferior cervical branch of uterine artery.

22.12 Secondary postpartum (puerperal) haemorrhage

This is vaginal bleeding between 24h and 12wks after delivery, usually between the 6th-14th day, and typically on the 10th. It is usually due to infection, particularly in association with:
(1) Retained pieces of placenta.
(2) Obstructed labour, causing necrosis of the cervix and vaginal wall.
(3) An exposed artery in the endocervix or endometrium.
(4) Caesarean section and breakdown of the uterine wound (21.14).
(5) A form of gestational trophoblastic disease (23.10).

If bleeding is mild, observation may be all the patient needs. It is not rare anyway for a woman to have a bloody discharge continuing 2 months after delivery. If she continues to bleed, or has signs of infection, treat her with chloramphenicol and metronidazole, and supply iron supplements.

If bleeding is severe, the patient needs antibiotics, resuscitation, and an ultrasound examination for retained pieces of placenta (38.3). Alert your anaesthetist. Make sure you monitor your patient carefully! Be sure she is well resuscitated before you start evacuating the uterus!

EXPLORING AND EVACUATING A SEPTIC UTERUS IS DIFFICULT

EQUIPMENT Sterilize 2 ring forceps (or swab holders), a Sims' speculum, and a big, blunt curette. If available, a large (12mm) suction curette is best. Add them to the vaginal examination tray.

Start an oxytocin infusion and place 800µg misoprostol rectally, so that you have the best chance of a well-contracted uterus which will also help you avoid a perforation because it is easily felt when hard.

Use aseptic precautions. Clean the vulva with an antiseptic solution. Put the Sims' speculum into the vagina. Ask an assistant to hold it, so that you can see the cervix. Hold the front of the cervix with one ring forceps. Put the other ring forceps into the uterus. Push it in very gently, until it is at the fundus. Feel the size of the uterine cavity. Open the handles, turn the forceps and close them again. Pull out any placenta you have grasped. Do this several times in different parts of the uterus, until nothing more comes out.

Suction-curette the uterus. Scrape it down the anterior wall, then the 2 side walls, and then the posterior wall. If you use a suction curette, rotate it while moving it carefully up and down. Lastly, scrape it across the fundus. *Don't scrape too hard*, or you may harm the uterine lining. It will be harder to move and you feel it scraping when the uterus is empty. If available, an ultrasound (held by an assistant) will be of help.

CAUTION! Emptying a uterus in the puerperium is difficult, and can be dangerous. Its wall is soft, and you can easily perforate it.
Never use a small curette, or any small instrument, because they will make a hole very easily. Work gently and carefully, and don't use a metal sound.

N.B. If you see fat tissue or bowel in your forceps, or clear fluid flows out: *stop at once!* You have perforated the uterus.

If the uterus is empty and severe bleeding continues, tamponade or pack the uterus and vagina (22.11). If this fails, proceed to laparotomy, as for an uncontrollable PPH (21.17, 22-13, 22-14).

22.13 Intra-uterine growth retardation (IUGR)

Most perinatal deaths occur in normally formed, normally grown babies weighing >2·5kg, as the result of birth trauma and asphyxia related to CPD, pre-eclampsia, abruption, cord prolapse, and malpresentation. These deaths are much more preventable than those from prematurity and IUGR.

Babies who are sufficiently small to be classified as being of low birth weight (<2·5kg) may:
(1) have been born after a pregnancy which was abnormally short, or
(2) have grown abnormally slowly during a pregnancy of normal length.

These 'small for dates' babies have not grown properly. In low- and middle-income countries, 25% of babies may be of low birth weight, and of these, 70% may have IUGR. Its causes, in approximate order of frequency, include: malnutrition, placental malaria, HIV disease, gestational hypertension, essential hypertension, recurrent antepartum haemorrhage, sickle cell disease, malformations and chromosome abnormalities, other virus infections, smoking, and alcohol.

There is also an 'idiopathic' group (30% in the developed world) in whom there is no obvious cause, but who are generally considered to be suffering from utero-placental vascular insufficiency. A hungry starving foetus from any of these causes readily dies, particularly during early labour, when his heart suddenly stops.

Because of the overwhelming importance of malnutrition as a cause, 21 of the 22 million low-birth-weight babies who are born each year are in low-income countries. Their chances of dying are 20 times higher than those of other babies. Malnutrition is the most potentially preventable cause.

IUGR is not easy to detect clinically. The risk factors for it, some of which are determined by malnutrition, include:
(1) IUGR in previous pregnancies.
(2) Low weight before pregnancy began.
(3) Low weight gain during pregnancy.
(4) Multiple pregnancy.
(5) Smoking.
(6) HIV disease.

Even so, 30-50% of cases commonly remain undiagnosed. The only way, if you have no ultrasound, of diagnosing IUGR is to encourage your midwives to measure the fundal height as carefully as they can with a tape measure between 20-36wks. If the uterus is 5cm lower than it should be, and there are <10 movements in 12h, you can diagnose IUGR.

Unfortunately, many mothers are unsure of their dates, and most health workers (including doctors) are unable to record the height of the fundus with sufficient accuracy. Even so, it is of little value in multiple pregnancy, polyhydramnios, a transverse lie, or in a very obese mother.

The fundal height chart (22-15) is derived from the UK; check if you have local charts: expect your normal heights to be 3cm less. If low-birth-weight babies are common in your district, you will find many mothers falling below the 10th centile, either because their babies have IUGR or because they are genetically small: the relative importance of these factors is unknown.

If you diagnose IUGR during pregnancy, and decide to deliver a mother before term for other reasons (it is not one of the indications for induction: 22.2), *don't do so before 34wks*. Do the surfactant test (22.1), in case the dates are wrong.

You then have a choice between inducing labour (22.2) and elective Caesarean section (21.10). Babies with IUGR tolerate asphyxia badly.

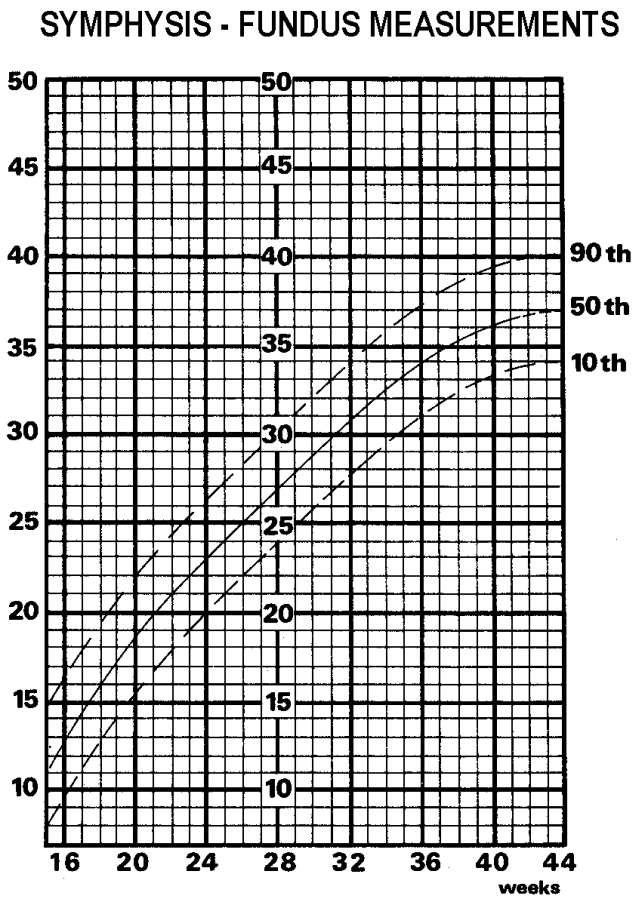


Fig. 22-15 A FUNDAL HEIGHT CHART.
If low-birth-weight babies are common in your district, you will find many mothers falling below the 10th centile, either because their babies have IUGR or because they are genetically small (the relative importance of these factors is unknown).

Babies with IUGR born at term have only a slightly increased risk of a major handicap, such as cerebral palsy or mental retardation. But between 1-30% of them have some minimal cerebral dysfunction, such as problems with speech, language, and learning.

The babies at greatest risk of some major handicap associated with IUGR, particularly cerebral palsy, are:
(1) The badly asphyxiated foetus with severe IUGR born at or post-term.
(2) The foetus with IUGR delivered <34wks.
Try to diagnose and deliver babies in the 'window' between 34-36wks if you can. Delivering a foetus whose mother has diabetes presents similar problems in judging the best time for delivery, the main difference being that he is too big rather than too small.

Much of the effort of modern obstetrics is devoted to detecting babies with IUGR, monitoring them, and getting them out into the world at just the right moment, when the risks outside the uterus are less than those inside it. If you can judge the moment of induction successfully, you may increase a child's chance of survival. Because of the enormous progress made in neonatal intensive care in high-technology surroundings, survival without handicap has improved dramatically.

In your case, delivering a foetus too soon might be much more risky. A foetus suspected of having IUGR is often found to be normal or simply premature and vice versa. However, treat the more manageable causes of perinatal mortality, some of which express themselves as IUGR: malaria, gestational hypertension, syphilis, obstructed labour, and poorly managed breech and twin deliveries. HIV however remains a huge challenge.

22.14 Puerperal sepsis

After childbirth a woman's genital tract has a large bare surface, which can become infected. Infection may be limited to the cavity and wall of the uterus, or it may spread beyond to cause peritonitis (10.1), septicaemia, and death, especially when resistance is lowered by a long labour, severe bleeding, or HIV disease. Sometimes the infection may be walled off by bowel and omentum. There may be a pelvic collection of pus in the pouch of Douglas, or there may be pus high in the pelvis or in the lower abdomen.

If sepsis is localized, only the lower abdomen is distended, there is guarding in both iliac fossae, and an ill-defined tender mass arising from the pelvis. There may be hyperactive bowel sounds. Vaginally, there are signs of recent childbirth or miscarriage, and there may be infected lacerations. The cervix is open and tender, painful on movement, and may be drawn up behind the symphysis. The pouch of Douglas may be thickened or swollen, but you cannot feel a fluctuant mass vaginally. The uterus and appendages form a mass which is difficult to define because of their tenderness.

If sepsis is generalized, the patient is weak, with anorexia, fever (perhaps with rigors) and generalized abdominal pain; walking is almost impossible. There may be diarrhoea initially. Usually there is a rapid thready pulse and a low blood pressure. The abdomen is uniformly distended, tympanitic, silent, and acutely tender. There may be a visible mass extending up to the umbilicus; you may have to pass a catheter to make sure that it is not merely a distended bladder.

MANAGEMENT
Resuscitate the patient (10.1). Treat her with chloramphenicol, ampicillin or gentamicin, *and* metronidazole. Monitor her 4-6hrly for signs of spread of infection.

If there is persistent vaginal bleeding and sepsis, there may be retained pieces of placenta. This is a common cause of puerperal sepsis, which will not resolve until the uterus is empty. Resuscitate the patient, administer antibiotics IV and curette the uterus with great care! Use your fingers if you have access or the largest curette which will be less likely to perforate the uterus.

N.B. Curetting a large, soft, infected uterus is dangerous.

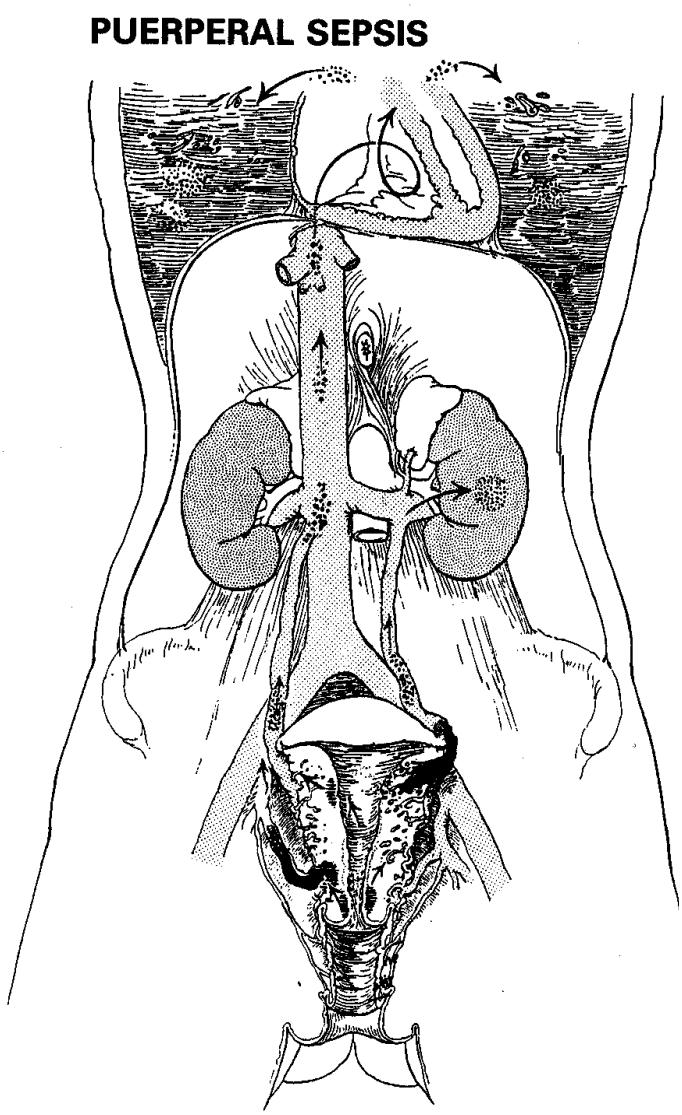


Fig. 22-16 PUERPERAL SEPSIS.
There is septic thrombophlebitis. Septic emboli are spreading through the ovarian and internal iliac veins to cause septicaemia and abscesses in the lungs and kidneys.

If the uterus is enlarged and tender, with a closed cervix as the result of scarring or carcinoma, it may be full of pus (pyometra, 23-9E). This can occur 2wks or more after delivery. Drain pus through the cervix by dilation with Hegar's dilators: Ch10 is usually enough.

If there is a definite swelling at one side of the uterus, this is parametritis.

If there is peritonitis with localizing signs, make a muscle-splitting incision as for an appendicectomy in the appropriate iliac fossa. Open the peritoneum, sweep gently with your finger, and insert a sump sucker. Several litres of thin pus may escape. As you enter an abscess cavity, gently free any adhesions and open up all loculi. Lavage the cavity (10.1).

If there is generalized peritonitis, make a midline incision, clear out the pus and wash the abdomen thoroughly. You might have to repeat this (11.10).

If fever recurs after initial improvement, there is more pus somewhere which you should drain, preferably through a midline incision. Perform an ultrasound examination of the abdomen (38.2l). If you fail to drain a subphrenic abscess (10.2), or other residual purulent collections, the patient will die.

If the patient recovers from the acute episode, but is left with a mass, she may eventually need a full laparotomy, with the separation of adhesions and the removal of a tubo-ovarian mass (23.1).

23 Gynaecology

23.1 Pelvic inflammatory disease (PID)

PID will probably be one of the commonest gynaecological diseases you will see, and may account for a third of your surgical admissions. It describes a syndrome of pelvic infection as seen in women. If it is common in your area, many of the women of childbearing age who are *not* pregnant and who present with abdominal pain may have it. You may admit 2-3 every week, and treat 10 times as many outpatients. The numbers of these patients, their frequently long stays in hospital, the surgery they need, the complications that follow and their mortality, make PID a major public health problem.

Infection elsewhere in the abdominal cavity usually originates in the bowel, but infection in a woman's pelvis usually starts in the genital tract. With the rare exception of tuberculosis, it always ascends from the vagina and cervix. PID is thus only a disease of women. It is even more common and severe with HIV disease, although it may appear less impressive because of the body's failure to mount a counter-attack.

Be very careful with the diagnosis of PID in pregnancy. You may only make this diagnosis if you have actually seen it during laparoscopy or laparotomy. Otherwise it is far more likely that the diagnosis is something else, *e.g.* torsion of adnexae/fibroid/bowel, an ectopic gestation, an extra-uterine pregnancy, malaria, septic miscarriage, appendicitis, uterine rupture, red degeneration of a fibroid, typhoid, urinary tract infection or obstruction.

Infection spreads from the vagina to cause:
(1) cervicitis.
(2) endometritis.
(3) salpingitis or pyo- or hydro-salpinx.
(4) salpingo-oophoritis or a tubo-ovarian abscess.
(5) pelvic peritonitis or pelvic abscess.
(6) generalized peritonitis or peritoneal abscesses.

Infection may also spread through the uterine wall into the broad ligaments to cause metritis, pelvic cellulitis (parametritis), a broad ligament abscess, or septic thrombophlebitis of the ovarian or the uterine veins. This is very serious and causes septicaemia with few local signs. This occurs mostly after a pregnancy and is then called either puerperal sepsis (22.14) or post-abortal sepsis. This is by definition *not* called PID although the first-line treatment is the same.

If antibiotics don't help in an infection related to pregnancy, a hysterectomy may be necessary to save a patient's life. Hysterectomy is not helpful in severe PID, but of course if both tubes are blocked or need removal because of pus, the result is just the same: infertility.

Both acute and chronic PID may cause an inflammatory mass involving the inflamed tubes, the ovaries, the uterus, the omentum, and loops of bowel. Between all these there are collections of pus, and in chronic cases fluid-filled collections between peritoneal surfaces (pseudocysts). PID is often but not always bilateral.
It is convenient to discuss separately:
(1) Infection unrelated to pregnancy, that is PID which does not obviously follow miscarriage or delivery. Because PID typically follows a period, it is sometimes called 'postmenstrual PID'. It is one of the most serious effects of sexually transmitted disease in women.
(2) Post-abortal infection (septic miscarriage: 23.2).
(3) Infected obstructed labour (21.5).
(4) Puerperal sepsis (22.14).
(5) Sepsis after Caesarean section (21.13).

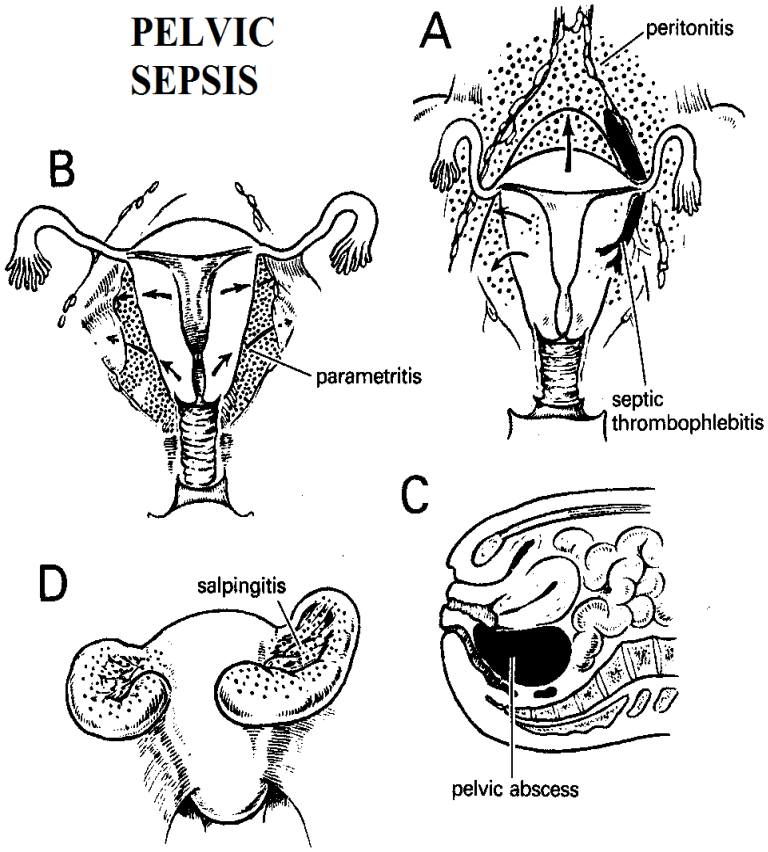


Fig. 23-1 PELVIC SEPSIS. A, infection spreading from the uterus to cause peritonitis. Infection can also spread as an infected thrombus (thrombophlebitis). B, infection of the connective tissue beside the uterus (parametritis). Infection may spread into the broad ligament, round the vagina or uterus, or up into the loin. C, collection of pelvic pus. D, salpingitis. *After Garrey MM. Obstetrics Illustrated Churchill Livingstone 1974, p.319-20 with kind permission.*

A patient with 'postmenstrual PID' is not pregnant; she has suffered no birth trauma and there are no infected products of conception. She may however have an intra-uterine device (IUD) in the uterus, which increases the risk of infection, but not as seriously as was previously supposed. PID is seldom fatal, and never causes septic thrombophlebitis. The pregnancy-related infections are all dangerous, and can kill. Post-abortal peritonitis is particularly deadly and has a mortality of 50%.

The organisms (*gonococci*, *mycoplasma*, *chlamydia*) responsible for PID may be sexually transmitted. These can pave the way for other organisms, especially anaerobes, and through tubal damage allow infestation by other organisms which may normally live in the vagina, *viz.* coliforms, various anaerobes, especially *bacteroides*, and rarely *actinomyces*. The latter organisms (and sometimes even the former) live harmlessly in the vagina and cervix, and only cause disease when the barriers to spread are removed by:
(1) Miscarriage or delivery.
(2) Menstruation.
(3) After D&C, IUD insertion, or hysterosalpingogram.
(4) HIV disease.
Many *gonococci*, and typically all *chlamydia* and *mycoplasma* are sensitive to tetracycline. But when infection follows pregnancy or a miscarriage, a mixture of organisms is responsible, including anaerobes, for which the patient needs metronidazole with chloramphenicol. By the time many patients present, secondary infection is likely, whatever the primary cause of the infection.

The clinical manifestations of pelvic sepsis are wide. They range from an otherwise symptomless infertility caused by blocked tubes, to generalized peritonitis, septicaemia and septic shock, with everything between these two extremes. Like a fire, PID can be of any degree of severity, from smouldering to fulminating. Also, like a fire, it can die down only to light up again later.

The typical acute presentation is of fever, bilateral lower abdominal pain, and tenderness, but seldom severe rigidity. There is also usually urinary frequency, dyspareunia, irregular or prolonged periods, and also a vaginal discharge. The patient may not mention all these symptoms, especially if she is a young unmarried girl.
The symptoms are usually mild, but can be severe with signs of peritonitis and occasionally septic shock.
Acute cases are often atypical, either because the disease is mild, or because it has been modified by previous treatment or HIV disease.
On pelvic examination, there is usually acute tenderness especially with moving the cervix side-to-side (cervical excitation). The pain may be so intense that you have to repeat the examination after you have administered an analgesic. There may also be a lower abdominal mass, vomiting, fever and a very high ESR.
Then you might find a minimally tender swelling in the left or right iliac fossa from which you can aspirate greenish pus (which typically does not yield a responsible organism on culture).
The typical chronic presentation includes infertility, and pelvic pain, often with dyspareunia, heavy and irregular menses, dysmenorrhoea, perhaps a chronic vaginal discharge, poor general health, and much misery. The diagnosis may be difficult; the differential diagnoses include endometriosis and psychosomatic pain.

ULTRASOUND will show thickened adnexae, uterine enlargement and free fluid in the pelvis, especially the pouch of Douglas, and will reliably confirm the diagnosis (38.2k).

You can usually treat PID non-operatively, but remove an IUD if present. *Make sure she can then get an alternative contraceptive*. Condoms provide some protection against sexually transmitted infections (STI) and hence PID; medroxy-progesterone, implants and the contraceptive pill don't prevent STI but often prevent PID, which is a complication of STI, and hence reduce infertility.
Occasionally, you will need to drain pus. Unfortunately, once PID has become chronic, there may be recurrent pain. The pain threshold is likely to be low if there is also infertility. *Don't operate on chronic PID*, because once it has been present for >4wks, the pelvic organs will be so densely stuck to one another that freeing them will be difficult and dangerous: *you can easily injure the bowel*. If surgery is indicated, be conservative. Leave the pelvic organs intact unless there is a tubo-ovarian abscess. Removing this can be difficult, so open it and drain it. If you leave the uterus and some ovarian tissue intact, menstruation will continue. Otherwise (if the ovaries have to be removed) advise hormone supplements for younger women (in the form of the contraceptive pill) till the age of 45yrs, but *beware: oestrogen therapy increases the risk of breast and uterine cancer.*

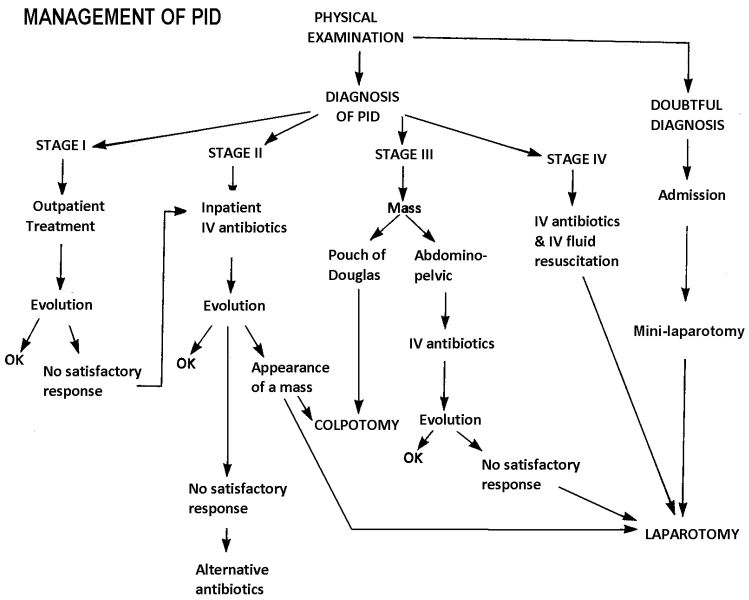


Fig. 23-2 GUIDELINES FOR MANAGING PID.
Stage I: there is no peritoneal irritation, and no pelvic mass. Stage II: there is peritonitis as shown by bilateral lower quadrant rebound tenderness. Stage III: there is a mass in the adnexa (tubo-ovarian abscess) or pouch of Douglas. Stage IV: there is generalized peritonitis.
After De Mulder X, Pelvic inflammatory disease in Zimbabwe, Tropical Doctor 1988;2:85.

(a) Acute PID

DIFFERENTIAL DIAGNOSIS

Acute PID has mostly to be distinguished from other causes of acute lower abdominal pain (10.1), including appendicitis (14.1), mesenteric adenitis and a urinary infection. The main gynaecological differential diagnosis is a ruptured ectopic gestation (20.6), torsion of an ovarian cyst or uterine fibroid, or a corpus luteum haemorrhage.

Fixity of the pelvic organs on vaginal examination is no help in distinguishing between PID, tuberculosis, and endometriosis, because all have these features.

Suggesting a ruptured ectopic gestation (20.6):
(1) Significant anaemia or circulatory shock.
(2) One or more periods missed by more than a few days, often followed by a small loss of dark or brownish blood vaginally.
(3) More tender on one side of the vagina than the other.
(4) A mass on one side or in the pouch of Douglas, which is not growing.
(5) No fever.

N.B. A -ve monoclonal urine pregnancy test excludes an ectopic gestation.

N.B. If an ectopic gestation is suspected, *don't perform a vaginal examination if there is no immediate access to a theatre*. Numerous are the tales of patients collapsing in shock because a vaginal examination re-ruptured the ectopic.

An elegant diagnostic tool is peritoneal lavage: run 200ml of warm saline, *via* a drip system, into the lower abdomen. Then ask the patient to roll herself a few times from left to right. Then put the fluid bag on the floor. Observe fluid running back into the bag: you may need to manipulate the cannula a little to achieve a nice flow. If the fluid is clear, there is most probably no acutely bleeding ruptured ectopic gestation.

Suggesting a twisted ovarian cyst or uterine fibroid: very acute colicky lower abdominal pain, sometimes with vomiting; a mass with no fever initially.

CAUTION!
(1) The amount of vaginal discharge is not proportional to the severity of PID. *Candida* and *trichomonas* cause a profuse discharge, but don't cause PID. *Gonococci* and *chlamydia* cause a less obvious mucopurulent discharge.
(2) Expect the diagnosis of PID to be wrong in about 20% of patients but examine the partner if possible: if he has a urethral discharge, PID is virtually certain.

GRACE (17yrs) was admitted with vaginal bleeding and fever, having attempted to induce an abortion on herself at 16wks. The cervix was wide open, the products of conception were visible, and there was a foul discharge. She was treated with antibiotics for 24h and then the uterus was evacuated. A few days later she was very ill with a distended abdomen. 3L of thin pus was washed out of the abdominal cavity and tetracycline was instilled abdominally. There was no perforation of the uterus. She was treated with more antibiotics, and intravenous fluids, and high-calorie liquid fluid was given orally in the hope of preventing severe acute malnutrition affecting her immune system. After 2wks, she was still febrile and very ill. A 2nd laparatomy was done to drain residual abscesses. Chronic sores developed at the sites of the drainage tubes, which continued to discharge pus. She did not eat well, and vomited from episodes of subacute obstruction, but was not well enough for a 3rd laparotomy. Three months after admission she died extremely wasted. She did *not* have HIV disease.
LESSONS (1) This is a typical history; any septic abortion, particularly an unskilfully induced one, is dangerous. (2) Re-laparotomy should have been done earlier at 48h and the abdomen left open. Although it is dramatic, this should probably be combined with a hysterectomy to save her life if the first operation does not result in an obvious improvement. An off-pink flaccid non-elastic-feeling uterus will in this situation probably maintain its sepsis. Hysterectomy is also needed in case of post-abortal tetanus. (3) Septic retained products of conception are not reached by antibiotics and are an ideal meal for micro-organisms. IV antibiotics followed as soon as possible by an evacuation is the correct treatment: no 24h delay is justified.

MANAGEMENT
You can usually treat PID as an outpatient if there is no peritoneal irritation and no pelvic mass (Stage I). Otherwise arrange admission if:
(1) There is lower quadrant peritoneal irritation and/or an IUD *in situ* (Stage II).
(2) There is an adnexal mass (Stage III).
(3) There is septic shock (Stage IV).
(4) You cannot exclude an acute surgical condition, especially an ectopic gestation.
(5) Outpatient treatment has failed.
(6) There is poor patient compliance.

ANTIBIOTICS
N.B. Cervical smears and cultures are of little help in choosing an antibiotic, because the organisms in the cervix may not be those which are causing the infection elsewhere. *The absence of gonococci in a cervical smear does not exclude gonococcal infection*. Usually, you will need to treat blindly with a broad-spectrum antibiotic. If possible, follow up and treat any sexual partners.

N.B. The regional university, preferably with an adequate laboratory, should perform culture and sensitivity testing of pus obtained from the abdominal cavity of a number of PIDs every few years and inform you about the best antibiotics to use (initially, blindly) in your region.

For Stage I, use tetracycline 500mg qds and metronidazole 400mg tds, for 7-10 days (2.8). Doxycycline 100mg bd is better than tetracycline but is more expensive. Insist on completion of the course. Also, use an analgesic, and advise abstinence from sexual intercourse until symptoms have resolved and until all sexual partners have been treated.

If there is no response (no better, rising temperature, more pain), check:
(1) Is the diagnosis correct?
(2) Is there a mass or collection of pus somewhere which needs draining?
(3) Are you using the right antibiotic at the right dose with the right frequency for at least 3 days?
(4) Is there an IUD which has not been removed?
Otherwise, proceed to Stage II.

For Stage II, use broad-spectrum IV antibiotics:
(1) benzylpenicillin 1·2g qds,
(2) chloramphenicol 1g stat followed by 500mg qds, and/or depending on severity,
(3) gentamicin 240mg od, plus
(4) metronidazole 500mg tds rectally.

If there is no response or a mass develops (Stage III), check whether the mass is in the pouch of Douglas, or if it is extending from the pelvis into the abdomen.

N.B. Many patients with acute PID develop a mass of matted viscera (distinct from an abscess which needs drainage). Check with an ultrasound (38.2k). This may take 6wks to resolve, but *there is no point in continuing antibiotics for more than 2wks*. If the illness persists (raised ESR with a spiking temperature after 2wks), there probably is an abscess which needs draining vaginally (colpotomy, 10.3) or suprapubically.

If you are not sure of the diagnosis, which may be beyond Stage II, perform a MINI-LAPAROTOMY to examine the pelvic organs, looking for red, sticky, and oedematous tubes. *This is safer than laparoscopy, which may result in bowel perforation if there are dense adhesions*, and the incision can be extended to a formal laparotomy if required.

LAPAROTOMY FOR ACUTE PID (GRADE 3.2)
INDICATIONS
(1) After 2wks of treatment with IV antibiotics for Stage II PID without resolution.
(2) Failure of 48hr IV antibiotic treatment for post-abortal or post-partum sepsis. Of course if there is a hole in the uterus with peritonitis present, the operation cannot be postponed that long. Make sure the uterus is empty.
(3) Generalized peritonitis due to rupture of a tubo-ovarian abscess. This may be spontaneous or it may follow a vaginal or rectal examination.
(4) Septic shock *after adequate IV fluid resuscitation*.
(5) The diagnosis is in doubt, and there is a possibility that there might be an ectopic gestation or appendicitis, for example: start with a mini-laparotomy.

N.B. This is not an easy operation and has a significant mortality.

PREPARATION. Resuscitate with IV fluids: you may need to infuse 3-4L saline or more during the first 24h. There may be considerable bleeding from the raw surfaces that will form when you free the adhesions between the loops of the bowel, so have 2 units of blood cross-matched. If the patient is seriously ill, there is danger of renal failure, so insert an indwelling catheter and monitor the urine output. Pass a nasogastric tube. Start antibiotics before the operation, if this has not already been done. Discuss the possibility of salpingectomy and/or hysterectomy, and get consent.

INCISION. Make a lower midline incision (11.2, 11-1) and extend it above the umbilicus if necessary. Be prepared on occasion to find some other, quite unexpected condition. *Don't use a Pfannenstiel incision.*

If the infection is limited to the pelvis, examine the upper abdominal cavity before you explore the pelvis and disturb the adhesions, which are limiting the spread of infection. Examine the subphrenic and subhepatic spaces, and the paracolic gutters; look for pus between the loops of the small bowel as far as you can reach them. If you find pus, wash it out with warm fluid. If you find dense adhesions, separate them very gently (12-8). If you don't find pus in the upper abdomen, carefully protect the upper uninfected part of the abdominal cavity with large abdominal packs. Slowly and methodically divide the adhesions between the bowel and the uterus, and look for pus. Divide the adhesions round the tubes and ovaries, and release the pus you find there. Try to get right down into the pouch of Douglas.

There is usually no need to remove the tubes or ovaries, however diseased they may look. The tubes have a double blood supply which prevents them becoming gangrenous: anyway, *you need consent for sterilization in order to remove both tubes! Even if there is a ruptured tubo-ovarian abscess, leave the Fallopian tube*.

When you find the uterine fundus, push your fingers down behind it, between the tubes, which will almost meet in the middle. You need not fear perforating the bowel here.

Gradually work your fingers down below the tubes. Free them from the bowel from below upwards. Remember the anatomy. Both tubes will be stuck down behind the uterus, over the top of each ovary. The rectum and colon will be adherent from below upwards to the back of the uterus, and then to both the tubes. Loops of small bowel and omentum will have stuck to them on top. If you can find the fundus, you will know where you are.

Don't panic when you find a mass of adherent bowel and omentum. It will always come clear in the end. First get down to the fundus by carefully easing off the bowel and omentum. *Don't use force!* Divide all adhesions and release all the pockets of pus.
Don't be tempted to remove a normal appendix.

CAUTION! Never pull the bowel. Avoid tearing it by going slowly, and squeezing and pinching the plane of cleavage between your fingers (12.6). Cut dense adhesions with scissors.

If there is generalized peritonitis (10.1), suck away as much pus as you can, then suck out the paracolic gutters. Wash with copious amounts of warm fluid. Make sure you release any collections of pus under the abdominal wall, between the large bowel and the abdominal wall, and under the diaphragm and the liver (subphrenic and subhepatic spaces). Bring out the whole small bowel over its full length in stages. Break down adhesions between loops of bowel, by careful blunt dissection, to release the many collections you will find there. Cover the bowel with moist warm cloths. Then go to the pelvis, and proceed as above for a localized pelvic infection.

LAVAGE depends on the extent of the sepsis (10.1):
If the pus is localized to the pelvis, wash it out of the pelvis only, before you remove the packs protecting the rest of the abdominal cavity.
If there is generalized peritonitis, wash out the whole abdominal cavity with warm fluid.

CLOSURE. Close the abdomen as a single layer, taking care to pick up the peritoneum and posterior rectus sheath which may be retracted, and leave the skin open for secondary closure if there is a great deal of pus (11.8). *Don't insert tension sutures*.

(b) Difficulties with acute PID

Be prepared for small bowel fistulae (11.15) and a burst abdomen (11.14), especially if abdominal distension persists for some time post-operatively.

If there is a septic miscarriage, you will have to make the difficult decision as to whether or not to perform a hysterectomy. Assess the state of the uterus and adnexa; perform a hysterectomy if:
(1) the uterus is perforated.
(2) the patient is no longer young and has had children (*though even if you leave the uterus she will probably be infertile*).
(3) you are not so experienced (a subtotal hysterectomy will be enough).
Perform a salpingo-oophorectomy (*avoid a hysterectomy*) if generalized peritonitis seems to originate in an abscess in one of the adnexa only.

If there is a mass and you are not sure if there is a ruptured ectopic gestation or pelvic abscess, and you have no reliable pregnancy test, perform a culdocentesis (20-6) preferably with ultrasound guidance, under GA. If you find pus, drain it through the vagina. If you find blood which fails to clot, or liquor, perform a laparotomy (20.6).

If there is an IUD *in situ*, and fever with great tenderness, infuse IV antibiotics and remove the IUD. Otherwise, if the fever is not high and the response to IV antibiotics good, you can leave the IUD *in situ.*

N.B. Sometimes PID is related to the insertion of an IUD while there is an existing chlamydia or gonococcal infection.

If you enter the abdomen and find little or no pus and few signs of inflammation, examine the pelvic organs and particularly the infundibulo-pelvic ligaments (23-21). One or both may be thickened and oedematous, and the thickening may extend under the ovaries to the uterus. This is SEPTIC THROMBOPHLEBITIS of the ovarian veins. If you find nothing, the thrombophlebitis is probably in a uterine vein which is not so easily seen. Continue with antibiotics in high doses. If possible, 2h after the operation start SC or IV heparin 1,000-10,000 units by bolus qds, controlled by estimating the clotting time and lengthening it to about 15mins (dosage varies depending on the type of heparin you use). Continue this for 1wk.
Watch carefully for abnormal bleeding, particularly from the abdominal incision, the urinary or intestinal tracts. Improvement should be quick.

If you find disseminated yellowish-white nodules throughout the pelvis, or a localized infection in the pelvis with nodules on the tubes and perhaps a CASEOUS ABSCESS or a shrunken thickened omentum, suspect TUBERCULOSIS (16.1). Take a biopsy and send this for histology. Start treatment early, and check for HIV disease (5.7).

If you inadvertently tear the small bowel, repair the perforation transversely in two layers. If there are several holes in the bowel, it is better to sacrifice a segment and perform one formal anastomosis (11.3) than close several holes which may leak. Lavage the abdomen thoroughly and be prepared to reopen the abdomen in 48h to inspect the bowel, and do a further lavage.

If you accidentally tear the pelvic colon, what you should do depends on the size of the tear and where it is. If it is small, oversew it in two layers. If it is large, but there is minimal soiling, freshen the edges and repair it formally, lavage the abdomen thoroughly and leave a drain. If there is considerable faecal spillage, close it as before and make a defunctioning colostomy (11.5,6) higher up. There is rarely a need to perform a Hartmann's procedure (12.9).

If there is persistent sepsis in the abdominal cavity, in spite of repeated attempts at drainage, leave the abdomen open (laparostomy) for daily irrigation (11.10).

(c) Chronic PID

DIFFERENTIAL DIAGNOSIS: urinary tract infection, endometriosis, schistosomiasis and pelvic tuberculosis.

ANTIBIOTICS
If there is recurrent or continuing infection suggested by raised ESR counts, try doxycyline with ceftriaxone, and maybe adding azithromycin for a maximum of 14 days.

If there is no improvement, either your diagnosis is wrong, or there is a collection of pus, perhaps a chronic tubo-ovarian abscess or a pyosalpinx.
Always consider TB.

Suggesting endometriosis: no children, >30yrs, chronic menorrhalgia since puberty, getting worse.

(d) Difficulties with chronic PID

If, on laparoscopy or laparotomy, you see bluish or brown nodules on the surface of the peritoneum and particularly on the utero-sacral ligaments, surrounded by serosal folding, suspect ENDOMETRIOSIS. You are most likely to see such nodules on the utero-sacral ligaments, in the pouch of Douglas, on the ovaries, on the posterior surface of the broad ligament, or on the fimbrial ends of the tubes.

If there is pain, use a non-cyclical progestagen to suppress menstruation, such as norethisterone 10mg od starting on the 5th day of the cycle (increased, if spotting occurs, to 25mg od in divided doses to prevent break-through bleeding) for at least 6 months. Or, use medroxy-progesterone 150mg monthly for 3 months and then 3-monthly: this will probably cause amenorrhoea and hence also stop the bleeding in the ectopic endometrial mucosa.

N.B. Norethisterone in the above formulation is, in most countries, far more expensive than a progestagen-only contraceptive pill.

The amount of norethisterone in the progestagen-only pill is 0·35mg. It is financially far more feasible for women to use this instead of the special norethisterone tablet: if used for endometriosis, 28 tablets (a whole monthly packet) are needed per day (in divided doses).

In a young woman who complains of infertility, menstrual irregularity, and chronic pelvic discomfort, TUBERCULOSIS (16.1) is a possibility.

If there is chronic PID and the woman is worried about PAIN but is not worried about having any more children, unilateral or bilateral salpingectomy without hysterectomy is usually possible but is difficult, so *don't attempt it* unless you have considerable operative experience, because the trouble with complications will be greater than the pain she had before!

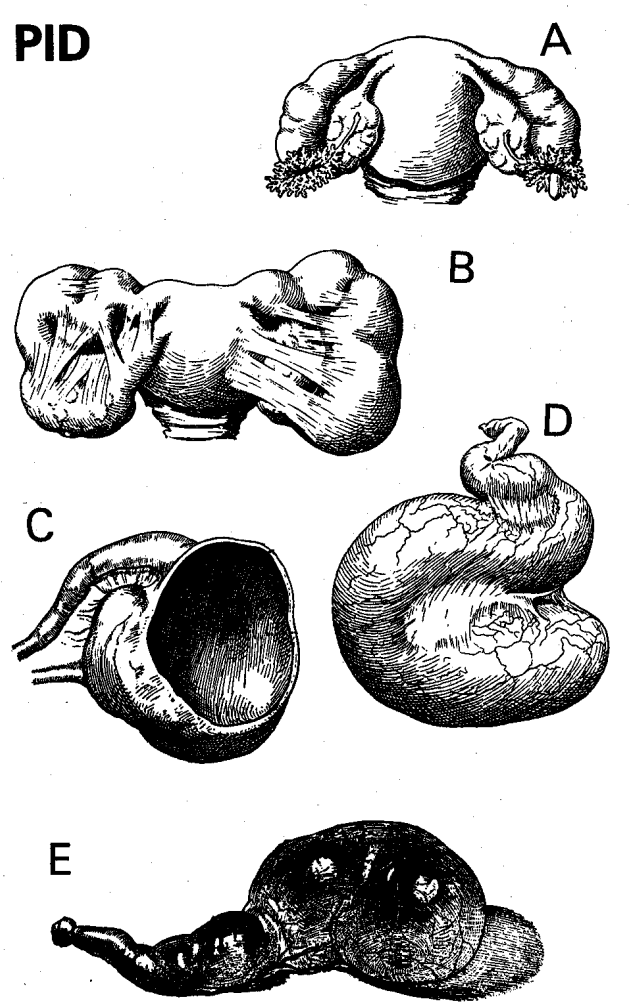


Fig. 23-3 PID AND PELVIC TUBERCULOSIS.
A, acute salpingitis with swollen congested tubes and pus leaking from the ostium. B, chronic salpingo-oophoritis with the tubes and ovaries densely bound by adhesions. C, tubo-ovarian cyst. D, tuberculous pyosalpinx. E, hydrosalpinx.
After Young J, A Textbook of Gynaecology, A&C Black 5th ed 1939.

23.2 Septic miscarriage

If there is fever and pus discharging from the cervix after an incomplete miscarriage, the products of conception have almost certainly become infected. This can follow a neglected spontaneous miscarriage, or it can follow unskilled interference. It is nearly impossible to prove which of the two is the case unless you find a perforation or foreign body.

N.B. In some cultures it is dangerous for the woman if you document '*criminal abortion*' in the notes. If women know that going to the hospital might result in prison, they will not come or arrive much too late. If a woman is brought by the police or angry members of her family for you to examine her, judge carefully where your professional duties lie. Note that it is virtually impossible to collect watertight proof of criminal interference as long as she denies this. Remember you are not the judge! Most of the time you can only state that a pregnancy was there but is now lost.

The diagnosis is usually easy if the history is clear of an actual or recent pregnancy. Unfortunately, the woman may be so frightened that she denies having tried to induce an abortion, even when she is very ill. The only way to avoid a misdiagnosis is to remember that any acute pelvic inflammation in a woman of childbearing age may be the result of a miscarriage.

Fortunately, the uterus is usually a good barrier to the spread of infection, but sepsis does sometimes spread as pelvic cellulitis or localized peritonitis. You can usually treat this without a laparotomy, although usually you should evacuate the uterus. If there is generalized peritonitis, perform a laparotomy.

Bleeding is often best controlled by emptying the uterus as fast as possible. If need be (no facilities, waiting list, the anaesthetist refuses to get involved because of anaemia), you can often do this digitally. One hand pushes down the uterus while the gloved index finger of the other hand evacuates the uterus. This is painful for the woman but better than waiting for possibly dangerous transfusions. Another advantage is that in this way you might detect a hole caused by an unskilled abortion before you have pushed instruments through it. You are very unlikely to make a hole yourself with your finger. In a septic miscarriage the speedy removal (just after an initial dose of IV antibiotics) of infected products of conception may make the difference between life and death.

Dramatic bleeding can also be caused by a piece of placenta hanging out of the cervix. You can deal with that rather painlessly with the help of a speculum and a sponge holding forceps. Alternatively, 800µg misoprostol PR while waiting for an evacuation might easily reduce bleeding enormously and even empty the uterus for you.

Rarely, a hysterectomy may be the only way to save life. The great dangers are septic shock and renal failure.

IF A WOMAN IS OF CHILDBEARING AGE, DECIDE IF PELVIC INFLAMMATION IS THE RESULT OF A MISCARRIAGE OR PREVIOUS PREGNANCY

(a) Diagnosis should not be difficult. Serious illness and fever (>38°C) after a miscarriage is typical. There is a foul bloody vaginal discharge, and sometimes frank pus.

Start by taking an endocervical swab for culture aerobically and anaerobically (if possible). This is better than a high vaginal swab. *Don't delegate this task.* Then use your fingers to remove any of the products of conception, which will come away easily.

Examine the uterus bimanually: it is tender bilaterally, perhaps with a mass. Sometimes there is local or general peritonitis (10.1). You might find a foreign body *in situ.* Look also for anaemia, jaundice (caused by haemolysis from septicaemia) and chest signs (septic emboli from thrombophlebitis). Your main concern will be to know how far infection has spread, and if you should perform a laparotomy.

CAUTION! There may be haemolysis, jaundice and high fever from severe malaria or dengue. *If you misdiagnose this as a septic miscarriage, the patient may well die*.

If the pulse is >120/min, the infection has probably spread beyond the uterus.

If moving the cervix causes great pain and the lateral fornices are hot, thickened, and tender, perhaps with a mass, the infection has spread to the pelvic connective tissue (parametritis, uncommon).

If you are uncertain about the diagnosis, yet the patient is very sick, resuscitate her, start antibiotics, and arrange to aspirate the posterior fornix of the vaginal vault in theatre. A seriously infected uterus can be silent, apart from a very sick patient.

If the history suggests that the uterus has been perforated with some instrument, the prognosis is worse. If it is leaking pus into the abdominal cavity, you may ultimately have to perform a hysterectomy.

(b) Treatment

If the patient is not very ill, and there are no signs that infection has spread beyond the uterus, a single broad-spectrum antibiotic, such as ampicillin 500mg IV qds, may be enough. Alternatively, use gentamicin 240mg IV od.

If the patient is very ill, with signs of spread outside the uterus, treat as peritonitis with septic shock (10.1). Resuscitate with rapid infusion of Ringer's lactate or normal saline. Measure the Hb, and cross-match blood. Get blood cultures. Transfuse blood if the Hb <60g/L.

N.B. Antibiotics will not control the infection if infected products of conception remain inside the uterus. So empty it; you will not cure the patient until you have done so.

ANALGESIA. Treat with pethidine or NSAIDs.

CONTROL THE BLEEDING. While waiting to perform an evacuation, or if there is bleeding with an empty uterus, administer ergometrine with oxytocin ('syntometrine') 1ml IV, or ergometrine alone.

Better, if you can, use misoprostol (800μg PR): this will make the uterus contract, stop post-evacuation bleeding and might complete an incomplete miscarriage before you have used mechanical means, *but don't rely on this alone!*

EVACUATION. Empty the uterus (GRADE 2.1) after starting antibiotics. Use a suction curette, but if there is serious bleeding or severe anaemia, do it immediately digitally. The uterus will be infected and soft, so be especially careful not to perforate it. Continue antibiotics after the evacuation.

POST-EVACUATION MANAGEMENT

Monitor the patient carefully, especially the urine output. There should be a dramatic improvement, and fever should settle in 48-72h.

If there is no improvement within 24h after evacuation, but signs of peritonitis are not obvious, there is probably a pelvic abscess. Try to confirm this by ultrasound (38.2k). Aspirate the posterior fornix. *Avoid the lateral fornices*, or you may injure the ureters or the uterine arteries. If you aspirate pus or blood-stained smelly fluid, drain it through the posterior fornix (colpotomy: 10.3).

If there is no improvement within 24h after evacuation, and clear signs of generalized peritonitis (pain, tenderness, rigidity, and abdominal distension), this indicates serious trouble. The uterus may have been perforated by an unskilled abortionist, or some harmful fluid might have been injected into the uterine cavity. *The laparotomy needed may well be difficult.* Improve the general condition as best you can by rehydration, transfusion (if Hb <80g/L), and have at least 2 units of blood available. Then perform a laparotomy (10.1).

N.B. Failure to counsel a woman about contraception if she has had an induced abortion (septic or otherwise) is malpractice. If she wants contraceptive services, provide them and *don't refer her elsewhere.* An IUD can be inserted immediately after an evacuation if there is no obvious sepsis.

(c) Difficulties with septic miscarriage

If you perforate the uterus when you evacuate a septic miscarriage, there is no easy answer. If you stop with an incompletely evacuated uterus, the risk of sepsis remains. If you complete the evacuation, you may enlarge the hole and even damage bowel. This also is dangerous. As a general rule, if you perforate a pregnant uterus, filled with infected products of conception, complete the evacuation as best you can *using your fingers*, then proceed with a laparotomy.

Repair the uterus with a single layer of interrupted sutures (21.17), and perform a tubal ligation if there is a complete family anyway. If the tissues don't hold, try to plug the

laceration with omentum, if there is minimal bleeding. Otherwise you may be forced to perform a hysterectomy (23.15). Look for any structures, especially small bowel, which may have been damaged in the perforation.

N.B. The treatment of an accidental perforation of a non-pregnant uterus or a small perforation in the midline of a uterus without infected products is different (23.4).

If septicaemia develops, treat vigorously with IV fluids, broad-spectrum antibiotics, and oxygen. Correct the acidosis and support the ventilation.

If you feel crepitations in the tissues, suspect GAS GANGRENE (6.24).

23.3 Abnormal and dysfunctional uterine bleeding (DUB)

Abnormal uterine bleeding includes any bleeding which is abnormal in its degree and timing. Dysfunctional uterine bleeding (DUB) is irregular, heavy or long-lasting bleeding *not* related to infection, pregnancy, medication or neoplasm. In low-resource countries, abnormal uterine bleeding usually has some obvious pathology. The list of its possible causes is a long one and is given below. Only diagnose DUB after you have excluded obvious pathology. DUB occurs most commonly at the extremes of reproductive life, in young girls not long after menarche, and in older women nearing the menopause, before complete amenorrhoea sets in. DUB should be an uncommon diagnosis in the prime of life; if you make it often, you are probably misdiagnosing miscarriages, *chlamydia* and *trichomonas* infections, cervical cancer, submucosal fibroids, or chronic ectopic gestation.

The commonest cause of DUB is the failure to ovulate. Because ovulation does not occur in the middle of a cycle as it should, the corpus luteum does not develop and produce progesterone normally. The endometrium grows abnormally thick under the influence of unopposed oestrogen, and eventually begins to shed unevenly. Courses of progestagen stop bleeding temporarily, and when these are stopped, normal periods usually follow.

The important diseases not to miss are carcinoma of the cervix (very common), and, usually in women >35yrs, mostly postmenopausal, carcinoma of the endometrium. The investigation of abnormal bleeding often requires intervention, but you may have to limit yourself to priority cases. These are inter-menstrual bleeding, and especially post-coital bleeding, which does not have some more obvious cause. Heavy regular periods are a common complaint, and are usually benign but may result in severe anaemia.

(a) History. A careful history and examination will nearly always reveal some obvious cause.
"When did the bleeding start?" "For how many days do you bleed and when?" "Are you bleeding now?" "Were you bleeding last week? Last month?"

Ask the patient to describe the bleeding pattern by giving approximate dates and amounts. Make sure she distinguishes blood escaping vaginally, from blood in the urine. If she sometimes does not bleed at all for a week or so, a malignancy is unlikely.

CAUTION!
(1) *Avoid labels like 'polymenorrhoea', 'menorrhagia', 'metrorrhagia' etc.* because they are too vague.
(2) Ask about post-coital bleeding.

(b) Examination. Is there anaemia? Examine the pelvic abdomen bimanually. Examine the cervix with a speculum.

DIAGNOSE DUB by exclusion, and remember that *a D&C is not automatic treatment for all forms of uterine bleeding*.
Exclude:
(1) Chronic PID (23.1), vaginitis (due to foreign body, *chlamydia*, *trichomonas*, or atrophic menopausal change), cervicitis and cervical ectopy,
(2) Miscarriage (20.2), ectopic gestation (20.6),
(3) Contraceptive medication, devices or hormone treatment,
(4) Fibroids (23.7), cervical or intra-uterine prolapsing polyp (23-8),
(5) Ovarian cysts and tumours (23.9),
(6) Cervical or endometrial carcinoma (23.8),
(7) Gestational trophoblastic disease (23.10),
(8) Tuberculosis (16.1).

At age <10yrs, there is probably a foreign body *in situ*.
At age <20yrs, this is probably truly DUB.
At age 20-40yrs, there may be any of the pathologies listed above. Don't miss carcinoma of the cervix. If there is inter-menstrual or post-coital bleeding, be sure to take a biopsy of any hard, friable, or ulcerated area on the cervix.

N.B. A D&C will not diagnose carcinoma of the cervix nor will a Papanicolau (Pap) smear (23.8); you can almost always diagnose carcinoma advanced enough to bleed by looking at the cervix with a speculum and taking a biopsy. In early pre-malignant cases you need to inspect it with 4% acetic acid. However, if you need acetic acid to see if there is something wrong with the cervix, then the bleeding is not caused by carcinoma in the visible part of the cervix, but there might coincidentally be a very early carcinoma, or one situated in the cervical canal or endometrium.
At age >40yrs, and especially if there is postmenopausal bleeding (bleeding ≥1yr after the menopause), always inspect the cervix, and if normal, do a suction curettage to exclude carcinoma of the endometrium. Other common causes include fibroids, especially prolapsed submucosal fibroids (23-7), and atrophic vaginitis.

(c) Treatment
Use the contraceptive pill bd for 10 days, then od. Bleeding will probably stop while taking the medication. There will be a withdrawal bleed (normal, scanty, or heavy) 2-3 days after stopping, but this should not last >1wk, after which normal periods should restart. Explain this to the patient. Review her again in a month, to see if treatment has worked and bleeding has stopped. Of course, if she needs contraception, continue the pill.

If bleeding has not stopped:
(1) your diagnosis was wrong, or
(2) she did not take the tablets regularly, or
(3) the DUB is unsuited to hormonal treatment.
Perform a suction curettage because this will be either curative or diagnostic (23.4).

In young girls with severe anaemia which does not allow for time for pills to take effect, and which makes GA dangerous, suction the uterus with a 4mm cannula; this will often cure the problem instantly, dramatically and avoid the need for blood transfusion, which may be dangerous unless you add furosemide to prevent fluid overload.

23.4 Dilation and curettage (D&C)
(GRADE 1.4)

There are two superficially similar operations: the evacuation of an incomplete or septic miscarriage, which does not usually require that the cervix be dilated (20.2), and dilation and curettage of the uterus, which is described here. Although both operations have similar complications, they have different indications.

Most gynaecologists now use SUCTION CURETTAGE if there is a pregnancy involved, and for the outpatient diagnosis of postmenopausal bleeding and (although rarely indicated) in case of infertility investigation.

D&C is a complement to a carefully taken history and examination, and is not a substitute for them. It is also one of the commonest operations in gynaecology, and one of the most abused, *so make sure that you only do it on the proper indications*:
(1) To diagnose the cause of abnormal bleeding.
(2) To diagnose carcinoma of the endometrium and tuberculous endometritis.
(3) To make sure that a patient is ovulating, when you are investigating for infertility and you have a poor history and no other means (vaginal ultrasound, or laboratory test for progestagen).

Ideally, all curettings should be sent for histology. Unfortunately, this is unlikely to be possible, so send priority cases. At age <40yrs, sending curettings for histology is probably unnecessary, unless they look abnormal macroscopically (profuse, thick, 'cheesy', or infected), or you suspect gestational trophoblastic disease (23.10).

Although D&C is usually simple, the long list of difficulties described below shows that it can be dangerous, and even fatal.

The main risks are:
(1) Perforation of the uterus, perhaps followed by haemorrhage or sepsis.
(2) Injury to a nulliparous cervix.
Most complications listed are rare.

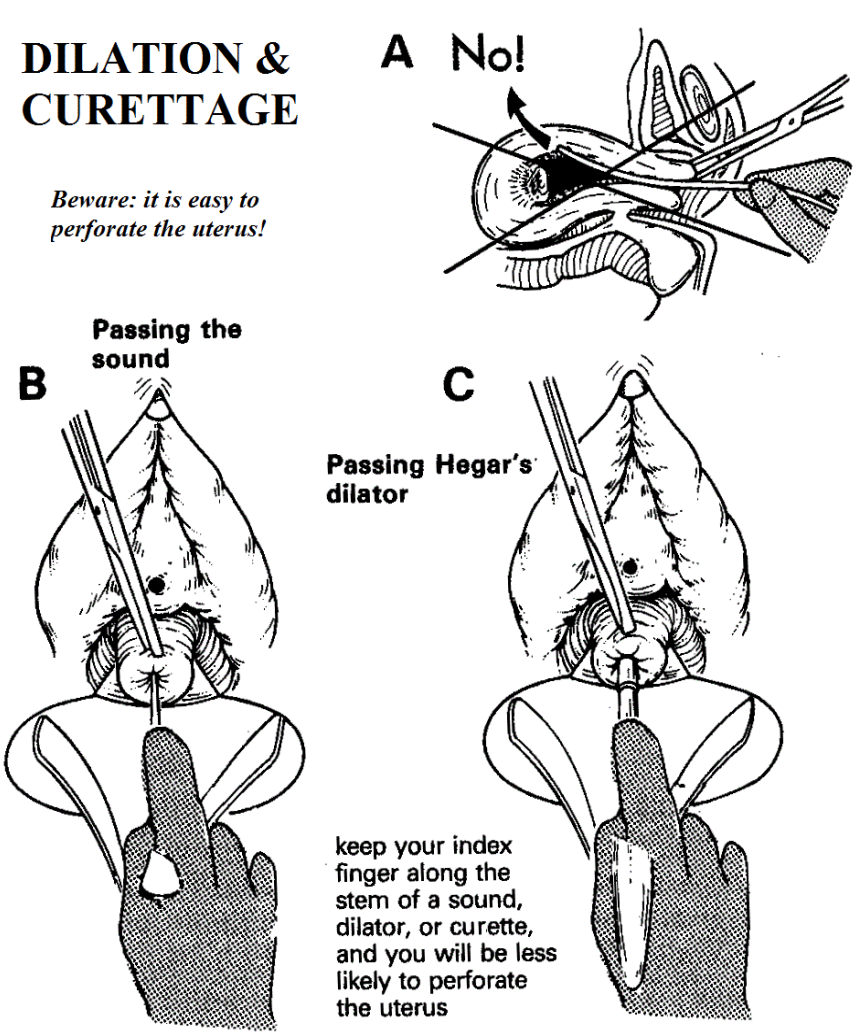

DILATION & CURETTAGE
Beware: it is easy to perforate the uterus!
A No!
Passing the sound
B
C
Passing Hegar's dilator
keep your index finger along the stem of a sound, dilator, or curette, and you will be less likely to perforate the uterus

Fig. 23-4 DILATION AND CURETTAGE.
A, the main danger is perforating the uterus. B, pass a sound. C, insert Hegar's dilator. Perforation of the uterus is less likely if you use your finger as a guide and steadier like this, with the finger acting as a brake. *After Bonney V. Gynaecological Surgery. Baillière Tindall, 1964 with kind permission.*

(a) Dilation followed by curettage
INDICATION
(1) To investigate abnormal bleeding. It may reveal: carcinoma of endometrium, endocervical adenocarcinoma (but not squamous carcinoma of the cervix, see below), choriocarcinoma, 'chronic endometritis', tuberculous endometritis, chronic anovulation, or submucous fibroids.
(2) To treat postmenopausal cervical occlusion causing pyometra, and to exclude carcinoma as its cause.

(b) Dilation only, without curettage
INDICATION
(1) To correct cervical stenosis after amputation or cone biopsy (23.8).
(2) To permit the insertion of an IUD.

CAUTION!
(1) *Don't perform a D&C to treat dysmenorrhoea occurring for the first time*, even if other methods have failed. Persevere with analgesics. If necessary, treat with the combined contraceptive pill or medroxy-progesterone to suppress ovulation.
(2) Where menses have never occurred, check if there is a uterus at all, after failing to produce a withdrawal bleed with hormones.
(3) Where menses have occurred and then stop, after also failing to produce a withdrawal bleed with hormones, check if, after an earlier traumatic evacuation, the anterior wall has fused with the posterior wall of the uterus (Ascherman syndrome).

N.B. D&C will not diagnose carcinoma of the ecto-cervix, for which a biopsy is necessary (23.8). *Don't do a D&C if you suspect there is a tubo-ovarian abscess*, which you should be able to diagnose clinically. Infection will have fixed the uterus; *dilating it with dilators may perforate it, spread the pus, and cause a fatal peritonitis*.

N.B. Ovulation can be proved by a regular period of biphasic basal temperature curve or by examination of the cervical mucus: a D&C is not necessary. Anovulation outside pregnancy can be investigated, not by D&C, but by seeing if you can initiate a withdrawal bleed with progestagens. If so, there are likely to be enough oestrogens. If not, there are anatomical problems (Ascherman syndrome as above, a blocked vagina or absent uterus) or she is postmenopausal. If withdrawal from using a combined oral contraceptive pill produces bleeding, then the anatomy is in order *(unless there is a bicornuate uterus).*

N.B. If you only want to take an endometrial biopsy, use a pipette or small Karman cannula; you can do this as an outpatient procedure.

You will usually make the diagnosis of tuberculosis histologically, but look at a separate specimen under microscopy, especially in the presence of HIV disease and if you are working in an area of high incidence.

EQUIPMENT
A catheter, Sims' and Auvard's vaginal specula, 2 vulsellum forceps, a pair of narrow ovum forceps, sharp curettes of different sizes, and a set of Hegar's uterine dilators. Arrange these in order of size on the trolley.

(c) Examination
(1) Effect a bimanual examination with disposable gloves to feel the size, position, and mobility of the uterus (feel also for disease in the adnexa). *Note particularly if the uterus is retroverted*, because this increases the chance of perforating it with a misdirected dilator.
(2) Then put on sterile gloves. If it is not already empty, drain the bladder.
(3) Swab the vulva and vagina. When you dilate the cervix, you will need a mental picture of its shape. Measure the depth of the uterus with a dilator or suction curette, except when you suspect a miscarriage.

CAUTION! Don't use a sound, which is thin and can easily perforate the uterus. A sound is useful, however, outside pregnancy, if there is a tortuous cervical channel. Adapt the curve of the sound to the cervical channel, and carefully try to find your way into the uterine cavity. If you succeed, you don't need to push it against the fundus. Sometimes sideward pressure or massaging (up or down) is needed to make it possible to follow the sound with a small dilator (for a D&C), a suction curette (DUB, PMB) or for insertion of an IUD.

(d) Dilation
If you expect a difficult dilation, insert misoprostol 200μg into the vagina a few hours before the procedure. (This is routine in many places for inserting an IUD, especially in nullipara.)

Start by making sure that the buttocks are well over the end of the table. If you are not using GA, inject the cervix with 3-4ml 1% lidocaine, using the thinnest needle possible and making sure it is fixed tightly onto the syringe. Then grasp the anterior lip of the cervix with a vulsellum forceps transversely. Then inject more lidocaine, just under the surface of the vagina in the fornices beside the cervix at 2, 6, 10 & 12o'clock. Or, perhaps better, inject from inside out, *i.e.* from endocervix into the body of the cervix. Wait 2mins.

Pull the cervix well down. This will bring a sharply anteverted or retroverted uterus towards the axial position, and reduce the risk of perforation. If it is very soft, as after labour, use sponge forceps.

With the picture of the uterine cavity in your mind, dilate the cervix, starting with the smallest dilator. As you do so, place a finger beside it to act as a 'brake' if you enter the cervix suddenly.

Insert the dilator in the direction which minimizes the resistance to it. When it has been in place for ≥30secs, insert the next size without delay, and without waiting for the cervix to contract again. Dilate a large uterus more than a small one. Stay in the midline: if you cause a perforation in the fundus, it is much less dangerous than in the parametria where there are large blood vessels.

CAUTION!
(1) Be gentle.
(2) Dilate slowly, leaving each dilator in place for ≥30secs.
(3) *Don't twist the dilators.*
(4) *Be particularly careful not to perforate the uterus*, especially in the first few months after delivery, or if you suspect a missed or incomplete miscarriage, carcinoma of the endometrium or trophoblastic disease. All these make it soft, friable and easily perforated.
(5) If you suspect a carcinoma, make sure you dilate the cervix enough to let you explore the uterus adequately.

(e) Curettage
Start scraping at the fundus, and scrape towards you all round the anterior, posterior, and lateral surfaces of the uterine cavity. Continue until there is a scratching feeling.

(f) Early difficulties during a D&C

If you cannot pass a sound or small dilator, the uterus is probably acutely flexed, either forwards or backwards. Feel it carefully:

If the uterus is anteverted (flexed forwards), pass the sound under direct vision though the external os, remove the speculum, and depress the handle of the sound posteriorly on to the perineum. When it is in the axis of the uterine canal, it will probably pass.

If the uterus is retroverted (flexed backwards), it may be held in place by adhesions. If a bimanual examination shows that it is fixed, abandon the operation *unless a D&C is essential.* Then put the vulsellum on the posterior lip of the cervix and pull it well down; pass the dilators with their points backwards.

CAUTION! If you tear the adhesions that are holding the uterus, she may bleed into the pouch of Douglas, or into the peritoneum. You may then have to open the abdomen to secure haemostasis.

If the cervix is so rigid that the larger dilators will not pass without the risk of causing tearing, leave one dilator in place for several minutes, before introducing the next one. If a dilator is tightly gripped as you remove it, reinsert it and leave it in a little longer before inserting the next largest size. A nulliparous or old person's cervix is often stiff. *Don't use excessive force*. You can usually do an adequate suction or curettage with a small, sharp curette, even if the cervical canal is only dilated to Hegar 6 (Ch20).

If larger dilators don't pass as far as smaller ones, you are inserting successive dilators a progressively shorter distance into the uterus. If you fail to realize what you are doing, you may only curette the cervical canal, and not the body of the uterus. Return to the smaller dilators, and start again.

If you find inserting larger dilators is unnaturally easy, *stop!* You have probably lacerated the cervix, and increased the risk of bleeding and sepsis. The tear may run into the vaginal vault from the external os, or it may start near the internal os, so that the tips of succeeding dilators ultimately enter the broad ligament.

If a dilator suddenly slips in much further than the one before (not uncommon), you have probably perforated the uterus into the peritoneal cavity, or into the broad ligament on either side, or into the bladder. Even experts occasionally do this, especially if a patient is pregnant, postpartum, or post-miscarriage, or if the uterus has been softened by an endometrial carcinoma, or gestational trophoblastic disease:
(1) Abandon the operation, and *don't try to confirm the diagnosis by probing the uterus*.
(2) *Don't irrigate the uterus*.
Infuse Ringer's lactate or normal saline IV and monitor the pulse, blood pressure, and temperature ½hrly. Treat with IV gentamycin, ampicillin and metronidazole. Expect recovery, but if there is deterioration, perform a laparotomy.

If you perforate the uterus and a loop of bowel appears at the vagina, *don't be tempted to resect it and anastomose it at the vagina,* and *don't push it back through the tear and plug the uterus with gauze*. Instead, perform a laparotomy, reduce the herniated bowel holding it firmly in your fingers to prevent spillage. Clean it, resect it, if it is damaged, and inspect the rest of the bowel.

If you split the tight vagina of a postmenopausal woman with a speculum, suture it, especially if it bleeds.

(g) Late difficulties with a D&C

If, post-operatively, there is pain on one side and a swelling in the broad ligament, a haematoma has formed. Occasionally, it may be so severe as to raise the peritoneum of the side wall of the pelvis, and extend even to the loin. If so, she will have the signs of a mass and of hypovolaemia: hopefully volume replacement will suffice to stabilize her. Otherwise perform a laparotomy to secure haemostasis, which is very difficult in that area. Try compression of the broad ligament for a full 15mins, before you start looking for a bleeding point. If this fails, put in packs of large gauzes to maintain the compression: when you remove them the following day, bleeding will usually have stopped or be readily controllable.

If symptoms of low abdominal pain and fever ensue, suspect salpingitis (23.1).

If peritonitis develops, either:
(1) there has been bleeding into the peritoneal cavity after a perforation,
(2) you have missed an ectopic gestation and ruptured it with your D&C,
(3) there is severe PID,
(4) there is iatrogenic bowel perforation, or
(5) there is a non-gynaecological cause.

Immediately explore the pelvis through an abdominal incision. Find and suture any uterine perforation. If it is extensive, and sutures will not control the bleeding, you can buy time by passing a rubber catheter around the uterus as far down towards the cervix as possible, and tightening it as a tourniquet.

N.B. This tourniquet occludes the uterine and ovarian arteries and so will cause necrosis of the uterus and ovaries if left on >3h. You can, however, pass the tourniquet bilaterally through a vessel-free area of the mesosalpinx instead and spare the ovaries and some blood supply to the uterus. This might be your best option if you are afraid to remove the uterus and are referring the patient. If you remove the uterus, leave the vagina open to allow free drainage. If there is extensive sepsis, wash out the peritoneal cavity. Look for damage to the bowel, and repair it.

23.5 Bartholin's cyst and abscess

If a cyst develops within the labia minora, it is usually due to blockage of a duct of a Bartholin's gland. *Don't try to excise it completely*; marsupialize it instead, which means everting its wall as a pouch, and then allowing it to heal. This is easier than trying to excise it, which is liable to be bloody and cause painful scars. Use a balloon catheter to keep a Bartholin's abscess open as its wall is unlikely to hold sutures.

(a) Marsupializing a Bartholin's cyst (GRADE 1.3)
Ask your assistant to immobilize the cyst with sponges on forceps. Make a longitudinal incision, with extensions at either end, in the margin between the pink vaginal and vulval skin, on the inside of the labium minus (23-5). Let the fluid escape. Apply Allis forceps on the edges of the labium minus, and retract them laterally. If necessary, push the cyst forwards by putting a finger behind it. Use interrupted absorbable sutures to tie the edges of the cyst wall to the skin, and to stop bleeding.

(b) Draining a Bartholin's abscess (GRADE 1.3)
Insert a small amount of LA subcutaneously; then open the abscess with a stab incision to allow pus to drain. Cut the end of the smallest Foley catheter available carefully and introduce it (with the balloon deflated) through the stab wound you have made.

Place the balloon within the abscess cavity and inflate the balloon with a few ml of water. Tie a knot very tightly round the catheter and cut excess tubing off so that the balloon, still inflated, remains in the cavity. Leave it there for 7-10 days; if it hasn't fallen out on its own by then, remove it after deflating the balloon.

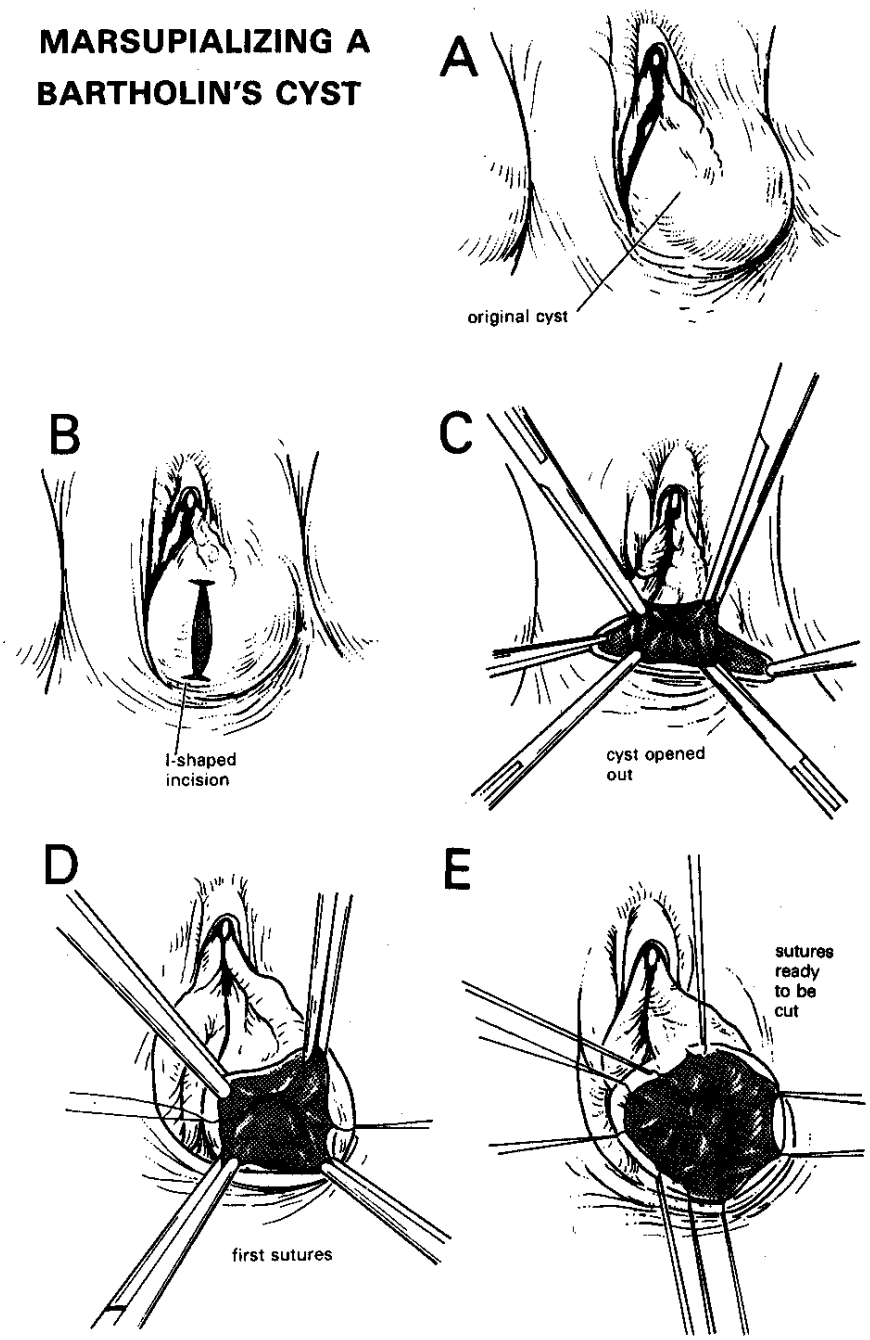


Fig. 23-5 MARSUPIALIZING A BARTHOLIN'S CYST.
A, the cyst. B, the incision with its extensions. C, opening out the cyst. D, the first sutures. E, sutures almost complete.

23.6 Urethral prolapse

In some communities prolapse of the mucosa of the urethra is common in young black girls between 6 months and 8 years. It usually causes no symptoms, but a child may have slight dysuria, or the mother may notice blood on the clothes. While most of the urethra remains in its normal place, its mucosa is gradually extruded at the external orifice to form a deep red or bluish tubular mass, which swells and becomes oedematous, and occasionally even gangrenous.

You cannot replace the prolapsed urethral mucosa. Distinguish it from a schistosomal granuloma, a urethral caruncle (23.17), a wart, or a sarcoma.

Treat it with oestrogen creams. Only operate if this fails; *don't do so just to appease the mother.*

URETHROPLASTY FOR URETHRAL PROLAPSE (GRADE 3.3)
Use a small sound to find the meatus in the prolapsed mass of tissue. Pass a catheter, withdraw it, and then replace this by fine artery forceps. Open the points slightly to distend the urethra. With the forceps as a guide, transfix segments of the prolapsing mucosa from side to side and then from front to back with strands of 3/0 absorbable. Use a knife or scissors, or, better, diathermy, to cut off the mucosa distal to the point at which the sutures cross the lumen.

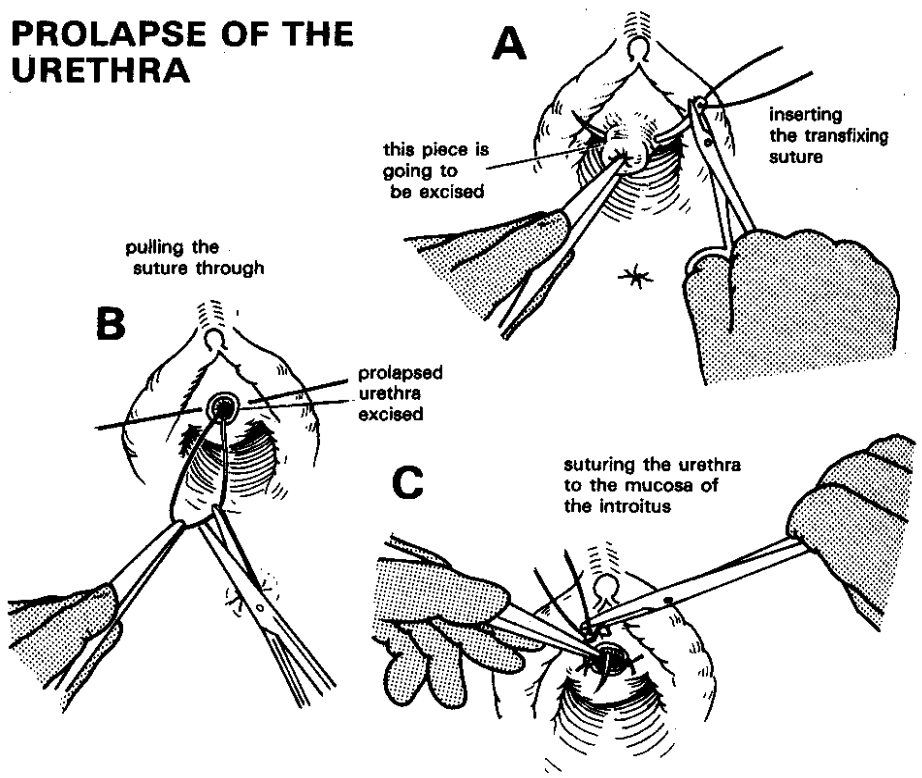


Fig. 23-6 PROLAPSE OF THE URETHRA. A, pass a suture through the prolapsed urethra. B, cut the suture so as to make 2 separate sutures at either side. C, insert further sutures as needed.
After Bonney V. Gynaecological Surgery, Baillière Tindall 1964 Figs 92-4 with kind permission.

Pull the strands down as two loops, cut them, and then tie each of the four pieces, so as to join the edge of the urethral mucosa to the skin. Insert a Foley catheter for ≥24h. Warn the patient that her vulva will be sore for at least 1wk.

23.7 Fibroids (Uterine myomata)

Fibroids, or myomata, are uncommon in young, but common in older, particularly black, women. There is also certainly a relation between low parity plus not having used hormonal contraception and fibroids; women with blocked tubes, women with infertile husbands, nuns, women sterilized at a young age, and women postponing their first pregnancy till they are >35yrs have fibroids more often.

They present with:
(1) Infertility or subfertility.
(2) Recurrent miscarriage.
(3) Abnormal bleeding.
(4) An abdominal swelling.
(5) Lower abdominal pain, usually pre-menstrual or, more rarely, acute, if a pedunculated fibroid twists.
(6) An intra-vaginal mass if a pedunculated fibroid prolapses (23-7). The cervix dilates to allow it to pass, and remains partly dilated around it.
(7) Chronic infection similar to PID where the fibroid has degenerated and itself becomes septic.

The mass may be large, necrotic, and infected. The severity of the symptoms depends less on the size of the fibroids than where they are; a small submucous fibroid can cause severe bleeding, whereas a huge interstitial one may hardly be noticed. Chronic bleeding may result in severe anaemia.

Ultrasound (38.2k) scan shows a heterogeneous hypo- or iso-echoic mass attached to the uterus, maybe with cystic or calcific changes.

Decide whether myomectomy (just removing the fibroid) or hysterectomy, ideally total, removing the cervix also, is best. You can perform myomectomy occasionally *per vaginam.* Total hysterectomy will remove the later risk of cervical cancer.

CAUTION! Don't simply amputate a prolapsing fibroid because it may pull the peritoneum down with it.

Surgery can be difficult, because of associated subacute or chronic PID, the need to avoid a hysterectomy, and the technical difficulties of performing a myomectomy.

(a) Differential diagnosis is that of a pelvic mass:
(1) Pregnancy.
(2) A full bladder.
(3) An ovarian cyst (23.9).
(4) A chronic ectopic gestation (20.7).
(5) PID with an inflammatory mass (23.1).
(6) A miscarriage.

CAUTION!
(1) A centrally placed fundal fibroid may feel like a pregnant uterus, but is much harder, mobile and lumpy.
(2) Pregnancy *can* occur in a fibroid uterus: so check for pregnancy before doing a hysterectomy!

(b) Indications for surgery

The rate at which a fibroid grows varies greatly. If it causes no symptoms, leave it alone unless it is the size of a ≥12wk pregnancy. At this size it will probably cause symptoms, so if the family is complete, recommend hysterectomy.

The indications for removing a fibroid depend more on symptoms (bleeding, anaemia, and premenstrual pain) than on its size. *Many patients don't need surgery,* especially when menopause is a few years off because the fibroids then stop growing or even reduce in size.

If the woman is older and does not want children, consider performing a total hysterectomy (23.15).

If the uterus and the mass seem fixed and tender, and especially if there is fever, this is more likely to be PID, with or without fibroids. Treat with antibiotics first, and reassess in 3-4wks.

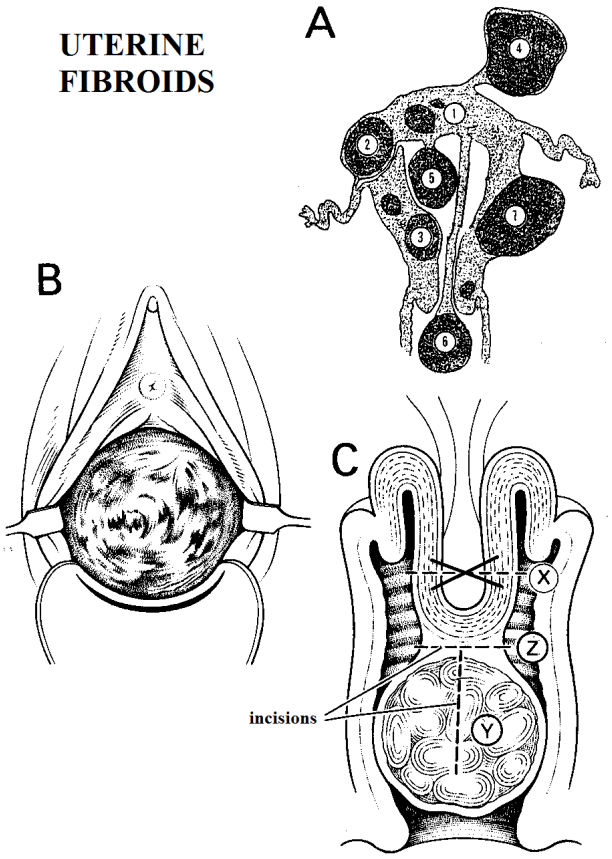


Fig. 23-7 UTERINE FIBROIDS. A, sites of uterine fibroids: (1) intramural. (2) subserous, distorting the tube. (3) submucous. (4) subserous and pedunculated. (5) intra-cavity. (6) prolapsing percervical. (7) parametrial. B, a submucous fibroid polyp has brought the fundus of the uterus down with it. C, the correct site for incision. First, incise the fibroid longitudinally (Y) to find the level of its pseudo-capsule. Then cut or twist off and transfix the pedicle transversely just above (Z), with no danger of entering the peritoneal cavity. *N.B. Don't incise at level X*!

If the temperature does not settle after a reasonable time, and the uterus remains tender, repeat the examination under GA. There may be:
(1) A tubo-ovarian abscess which fluctuates and needs draining. If so, leave the fibroids until later.
(2) Mobile degenerating fibroids that you can operate on.

N.B. 'Red degeneration' can occur in a fibroid most often during pregnancy, and can cause pain and a tender mass, but not the degree of fever that is common with pelvic sepsis.

If the patient is younger and wants children, **or has suffered with infertility and repeated miscarriages**, consider doing a myomectomy with a tuboplasty (if needed). Make sure she understands that:
(1) If it is found to be impracticable, she may have to have a hysterectomy, or to have the abdomen closed with nothing being done.
(2) She may grow more fibroids later, especially if she does not conceive.

MYOMECTOMY (GRADE 3.3)
INDICATIONS
A woman with fibroids who wants children.

N.B. Fibroids may be the result of infertility rather than its cause. Myomectomy is hazardous, and has more complications than hysterectomy. Most patients are better off with a hysterectomy (subtotal if necessary), or with no surgery at all.
If you are inexperienced, *don't attempt it unless there is*:
(1) A single fibroid <10cm in diameter.
(2) A fibroid which is subserous (pedunculated into the peritoneal cavity) or submucous (pedunculated into the uterine cavity, and coming through the cervix into the vagina: 23-7).

CONTRAINDICATIONS
(1) Active sepsis.
(2) Dense adhesions of both tubes which make pregnancy anyway impossible.
(3) A large posterior fibroid in the pouch of Douglas. Leave it unless you are an expert: removing this without damaging the bowel or ureters is difficult, and can be bloody.

METHOD FOR INTRAMURAL FIBROIDS
Bleeding is the great danger: cross-match 2 units of blood. Use tourniquets to prevent bleeding. Wind a rubber sling around the uterus, and push it down as far as you can in the direction of the bladder; *don't encircle the Fallopian tubes and ovaries, making sure they stay above the tourniquet.* Pull the rubber sling (or Foley catheter) tight and fix its tension by applying a clamp across both ends just above the bladder.

Make an incision over the fibroid which exceeds its diameter by 2-3cm. The correct plane to remove it in may not be easy to find. Cut into the fibroid and you should see it. Shell it out. If necessary, remove some of the wall of the uterus to reduce the size of the dead space.

Be very careful not to remove too much normal uterus; if you have to repair the outer layer of the uterus under tension, the sutures will cut through.

See if you can remove more fibroids through the same incision; grasp a small fibroid with a towel clip or vulsellum forceps to make it easier to remove and extract from surrounding tissues.

Try to prevent incisions in the lower posterior wall of the uterus. Dense adhesions fixing the adnexa and the uterus in the pouch of Douglas can easily result: there is then little chance of pregnancy and frequent dyspareunia.

Repair the uterus with at least 2 rows of #1 absorbable mattress sutures. Remove the catheter and sling. If the uterine incisions bleed, insert more mattress or Z-sutures.

N.B. Bleeding in the uterine wall will always stop once the pressure within the wall becomes great enough.

Close the abdomen without drainage. Make sure the patient knows what you have removed, and understands that she must nearly always have an elective Caesarean section for a subsequent pregnancy: *these are usually elderly women and their last chance of pregnancy must not end in uterine rupture and foetal death!*

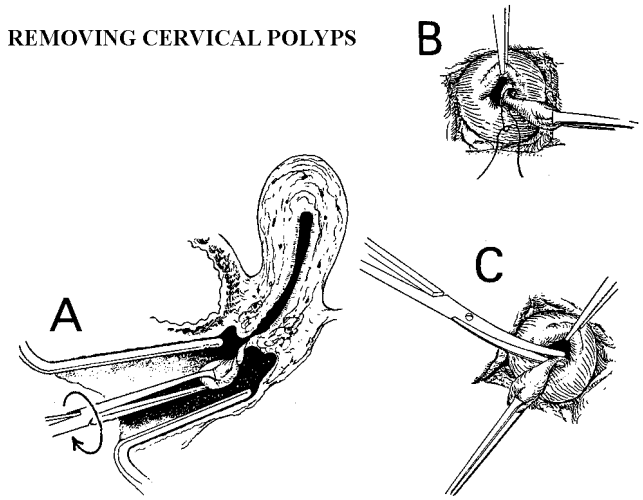


Fig. 23-8 THREE WAYS OF REMOVING MUCOSAL POLYPS. A, by twisting. B, by ligation. C, by section. These mucosal polyps are much more common and are more easily removed than fibroid polyps. *Kindly contributed by Jack Lange.*

METHOD FOR A SUBMUCOUS FIBROID POLYP

There may only be a single vaginal fibroid. If there are others, they can be removed later by myomectomy or hysterectomy.

If the pedicle of the polyp is thick and is attached well within the cavity of the uterus, *be careful*. Incise it if you have access to find the level of the capsule of the fibroid first (23-7). You can then cut and tie the pedicle just above this, with no danger of entering the abdomen.

Transfix the pedicle as far as you can away from the uterine wall, and divide it distal to the ligature, so that you minimize the risk of opening the peritoneal cavity. If you don't remove it completely, it will recur, but *don't cut a broad-based pedicle too near the uterus leaving a defect that you cannot close.*

If there is a large fibroid prolapsing through the dilated cervix, define it as well as you can with your fingers first and then twist it by rotating it with 2 vulsellum forceps in the same direction till it comes loose and then remove it vaginally. If it does not yet prolapse through the cervix, you can soften the cervix by inserting 600µg misoprostol PV; wait ≥4h and then grasp the fibroid and twist it.

N.B. Chronic bleeding may result in severe anaemia, so use iron medication but rarely blood transfusion pre-operatively. If there is post-operative bleeding, pack the vagina. The alternative is a hysterectomy, *but don't do this till you have administered antibiotics.*

(c) Difficulties with fibroids

If the fibroid is painful, either spontaneously or on palpation, with perhaps mild fever, this is due to aseptic necrosis (red degeneration), or associated torsion of a pedunculated fibroid.

If you discover a small submucous fibroid when you are doing a D&C for abnormal vaginal bleeding, you may be able to twist it off with the curette or ovum forceps.

If there is an endometrial mucosal polyp, you may only find it when you do a D&C if you haven't done an ultrasound before.

If during a Caesarean section, you find a fibroid, *don't remove it*, unless it is pedunculated.

If a woman just before menopause with moderate-sized fibroids has heavy bleeding, try medroxy-progesterone 300mg stat, then 150mg after 2 months, and then again 3-monthly till the expected time of menopause. Bleeding may stop and the fibroids shrink or stop growing, so you may avoid a hysterectomy. Medicated IUDs/implants probably inhibit the growth of fibroids also, or at least reduce the associated bleeding.

23.8 Cervical and endometrial carcinoma

(a) Cervical carcinoma

Cervical carcinoma is among the commonest tumours in the world, and causes much suffering. Most tumours are squamous carcinomas but a few are adenocarcinomas arising from the endocervix. Treatment is the same for the two subtypes. Carcinoma of the cervix is more common in grand multipara, in those with a history of many sexual partners, in partners of the uncircumcised, in women who smoke, and those with HIV. Even with ARV therapy, cervical carcinoma is 10 times as common in HIV+ve women as HIV-ve (5.6).

Human papilloma virus (HPV) is the cause of squamous cervical carcinoma. Many women become infected with this virus which has different subtypes, of which 16, 18, 31 and 52 are the ones most often causing carcinoma. Other subtypes are non-oncogenic and cause genital warts, for example.

Some women are not able to conquer the initial infection of HPV, which then becomes chronic, changes her DNA and can cause cancer after many years. Smoking, pregnancy, HIV infection, and probably malnutrition inhibit the ability to overcome HPV.

There are now vaccines against the most common subtypes of HPV which could have a dramatic impact on the incidence of cervical carcinoma if given to girls (and boys, so they don't spread the virus) before they are sexually active. Tragically this vaccine is too expensive for widespread use in many low-income settings.

Presentation is with:
(1) inter-menstrual bleeding, often bright red,
(2) post-coital bleeding,
(3) postmenopausal bleeding,
(4) vaginal discharge, which is not always blood-stained, and is usually watery.

The pre-malignant phases of cervical cancer can be identified in a woman without symptoms by examining a smear of cervical cells on a slide: a Pap smear.

N.B. Pap smears are nearly impossible in the presence of cervical bleeding!

Unfortunately, a screening programme needs good laboratories and a system to recall and monitor patients with abnormal cells. A cheap and effective screening method is to apply Lugol's iodine or 4% acetic acid to the cervix and examine it after 1min. Look for the squamo-columnar junction, where the outer pink squamous and inner reddish columnar lining of the cervix meet. The presence of dense, opaque, well-defined 'aceto-white' lesions or growths here is highly suggestive of a pre-malignant lesion (severe dysplasia). If this is present, a cone biopsy would prevent this lesion becoming malignant (see below). However, carcinoma is usually recognized later by an irregular, ulcerative lesion of the cervix, which bleeds easily. Cervical carcinoma is less common in those who use condoms, so encourage patients with multiple sexual partners, or those with partners who themselves have multiple partners, to use them.

DIAGNOSIS. Take a careful history and establish the timing and appearance of the bleeding; a patient may confuse vaginal bleeding with haematuria. Examine the abdomen and the groins. Examine the vagina and cervix first digitally, then with a speculum *using a good light source*. Perform a rectal examination and a bimanual examination of the pelvic organs. Do all this as an outpatient procedure. You may see:
(1) An ulcer on the cervix, often extending into one or more fornices.
(2) A cervical polyp (less common).
(3) An enlarged barrel-shaped cervix which may look relatively normal.
(4) Erosion into the bladder or rectum causing a vesico-vaginal or recto-vaginal fistula.

N.B. In all these situations, take a biopsy if you are unsure of the diagnosis. *A Pap smear is for lesions invisible to the naked eye.*

DIFFERENTIAL DIAGNOSIS includes a simple cervical ectopy, a cervical or endometrial polyp, a submucous fibroid, various stages of miscarriage, irregular bleeding near the menopause, carcinoma of the endometrium, senile vaginitis, urethral caruncle (23.17), syphilis and a tuberculous or schistosomal granuloma.

N.B. This last may look exactly like cancer!

Suggesting carcinoma: the lesion feels hard, it is friable (bits break off easily), a raised edge, you can feel it rectally extending into her parametrium.

Suggesting cervical ectopy (normal and physiological): an area of glandular columnar epithelium (usually found inside the cervical canal) around her external os surrounded by normal squamous epithelium with *no raised edge and which does not feel hard and gritty*. There is usually no contact bleeding.

If an acetic acid test is suspicious, freeze it with the help of liquid CO_2 (available via cool drink producers). This will cure 80-90% of patients (with some innocent over-treatment). Nurses can easily learn to do this. Otherwise you need to take an excisional cone biopsy, but this is not feasible if your patient cannot return for follow-up, pay for pathologist fees etc.

N.B. D&C is not necessary for investigating carcinoma of the cervix. This does occasionally reveal carcinoma of the body of the uterus or of the endocervix. *Invasion of the body of the uterus by a carcinoma of the cervix is not one of the staging criteria for this tumour. Officially, staging includes a cystoscopy but staging is usually quite possible without it.*

STAGING AND MANAGEMENT
If you suspect the carcinoma is in one of the earlier stages, with some hope of cure or palliation, take care to stage the disease under GA in the lithotomy position. Use a speculum to inspect the vagina for Stage IIA or III disease and for fistulae. Do a rectal examination to assess spread beyond the uterus. Do a vaginal examination at the same time, and feel the rectovaginal septum between your fingers.

If there is an advanced lesion, an examination under GA is unnecessary, and is possible as an outpatient if you are gentle, and remove a small piece of tissue for histology. You may however need to suggest admission on social grounds, especially if home is far away, but beware if you are dealing with terminal disease. Death in your hospital might result in very steep transport costs for the family, *and no improvement to your reputation*. Death caused by cervical carcinoma is usually not accompanied by pain, and caused by renal failure or blood loss. Arrange that there is access to analgesia/sedatives as needed (37.1,2).

Stage 0, carcinoma *in situ*. This is a histological diagnosis, made from either a cone (or wedge) biopsy, or a +ve Pap smear. The cells look malignant, but have not yet invaded the surrounding tissue, and may not do so for many years, if ever. Freezing (cryosurgery, see above) or cone biopsy (see below) is curative at this stage.

Stage I. The tumour is confined to the cervix (IA, <4cm; IB, >4cm in size) and can be cured by radical hysterectomy, *i.e.* with the parametrium (the round, uterosacral, broad and cardinal ligaments), the upper ⅓ or ½ of the vagina, and pelvic lymph node dissection. There is an 85-90% chance of surviving 5yrs. A radical cervicectomy (cervix, 2cm of upper vagina, and paracolpos) is useful if the patient still wishes to conceive. Follow-up is essential as you may still be able to treat recurrences. Beyond Stage I, however, cure is unlikely with surgery alone.

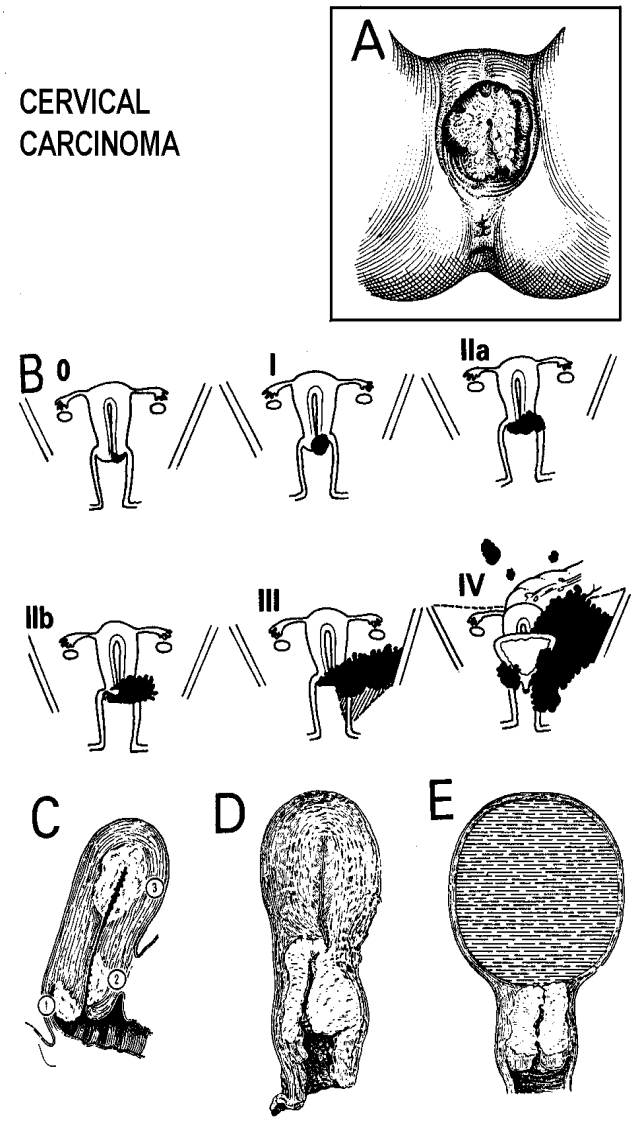


Fig. 23-9 CARCINOMA OF THE CERVIX.
A, a woman with a neglected carcinoma of the cervix involving the vulva (Stage IV). B, staging the disease. Stage 0, carcinoma *in situ*. Stage I, tumour is confined to the cervix (this can easily be seen and felt). Stage IIA, it extends into the upper vagina or uterus, but not into the parametrium. Stage IIB, it extends into the parametrium, but not to the pelvic wall. Stage III, it involves the side wall of the pelvis, or the lower ⅓ of the vagina or produces hydronephrosis. Stage IV, it involves the mucosa of the bladder, or rectum, or there are distant metastases. C, sites of uterine carcinoma: (1) Squamous carcinoma of the vaginal cervix. (2) Adenocarcinoma of the endocervix. (3) Adenocarcinoma of the endometrium. D, carcinoma of the cervix (Stage I or II). E, pyometra; the body of the uterus is filled with pus above a Stage I carcinoma of the cervix.
After Pinter and Roberts; Young J. A Textbook of Gynaecology A&C Black, 5th ed 1939.

Stage IIA. The tumour extends out from the cervix into the upper vagina or uterus, but not into the parametrium. Stage IIa is uncommon because progress to Stage IIb is so rapid. Radical hysterectomy should be combined with radio- or chemotherapy. You may achieve >50% 5yr survival. Radiotherapy to a dose of 75-80Gy is necessary or use cisplatin with or without fluorouracil (37.4). Complications of radiotherapy are common: look out for adhesions, proctitis (26.11), cystitis (27.36), and vesico-vaginal fistula (21.18). Radiotherapy implants are useful.

Stage IIB. The tumour extends into the parametrium but not as far as the pelvic wall. Radical surgery is unhelpful, but radio- or chemotherapy may be of benefit.

Stage III. The tumour involves the lower ⅓ of the vagina (IIIA) or the side wall of the pelvis (IIIB), or is causing hydronephrosis. Management with radio- or chemotherapy can achieve 5yr survival rates of 30-40%.

Stage IV. The tumour involves the mucosa of the bladder or the rectum, or has spread beyond the pelvis. Palliate the patient with chlorpromazine, diazepam and analgesics as the pain increases (37.1,2).

CAUTION!

(1) A Pap smear –ve does not exclude invasive carcinoma, nor does a +ve smear prove it (there may be carcinoma *in situ*, *i.e.* cells with cancerous changes only within the surface layer of the cervix which with time can develop into cancer).

(2) *Carcinoma* in situ *does not cause abnormal bleeding or other symptoms*.

CONE BIOPSY (GRADE 2.2)

INDICATIONS

(1) To confirm a +ve or suspicious acetic acid test or Pap smear. If the cervix is clinically normal, repeat the acetic acid test before you take the biopsy.

(2) As treatment for carcinoma *in situ*, as shown by a +ve Pap smear.

(3) As primary treatment for a lesion considered to be carcinoma *in situ* or possibly Stage I, in order to obtain reliable distinction between the two.

CAUTION!

(1) *Don't do a preliminary cervical dilation*. If it is necessary, do it after you have taken the biopsy.

(2) *Don't use cutting diathermy*: it spoils the specimen.

METHOD. Put the patient in the lithotomy position. A cone biopsy is notorious for post-operative bleeding, both reactionary and secondary. Minimize this by inserting a preliminary absorbable suture to prevent bleeding, exactly as with McDonald's cervical suture (20.5).

Either, start by inserting a #0 absorbable suture (23-10B), and tie it. Put it all round the cervix, as high up as you can, and pull it tight to occlude the descending cervical vessels.

Or, use vulsellum forceps to grasp the anterior aspect of the cervix away from the lesion. Transfix the descending cervical vessels (23-20) on each side with #0 absorbable suture, leaving the ligatures long as stays.

N.B. Schiller's test (optional) helps to define the extent of the atypical epithelium, but is not absolutely reliable. Apply 1% iodine to the lesion and the surrounding epithelium. Normal epithelium stains brown, atypical epithelium may not.

Incise the normal epithelium c.5mm from the abnormal epithelium, and extend your incision all round the cervix.

If you have cutting diathermy, cut (with your cold knife) in small steps while in every stage of the procedure arresting the haemorrhage. Apply vulsellum forceps to the lips of the cervix at 12 and 6o'clock positions.

Deepen the incision to remove a cone, with its apex at the cervical canal, keeping its edge 3-5mm away from the abnormal tissue. Use diathermy for haemostasis if possible. Leave the raw surface open.

Cut the cone open in the 12o'clock position (23-10E) and send it intact for histology.

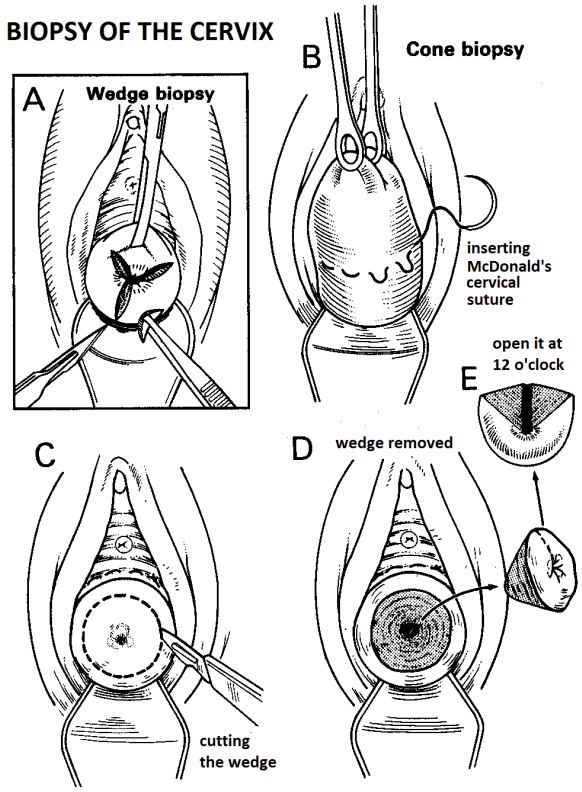


Fig. 23-10 BIOPSY OF THE CERVIX.
A, a wedge biopsy. B, insert McDonald's suture before doing a cone biopsy. C, cut the cone. D, remove the cone. E, open the cone out with an incision in the 12o'clock position.

CAUTION! For a pathologist to examine a cone biopsy:
(1) it must be big enough,
(2) its orientation on the patient must be identifiable (hence the 12o'clock cut),
(3) it must be in one piece,
(4) it must be injured as little as possible.
The pathologist must be able to report whether the edges of the cone are normal, and if not, where the suspicious tissue is situated.

WEDGE BIOPSY (GRADE 1.2)
INDICATIONS
To check for malignancy in an ulcer of the cervix.

METHOD. This is similar to but not the same as a cone biopsy. At ≥3 sites on the ulcerated area, excise ellipses ≥3mm deep, crossing the margin between the ulcerated and the normal area (23-10A).

(b) Endometrial carcinoma
Endometrial carcinoma occurs mostly in postmenopausal women, and presents with vaginal bleeding. *The older the woman, the larger the chance that vaginal blood loss is a sign of malignancy*. When disease is advanced, abdominal pain with or without bowel and bladder symptoms dominates.

STAGING
Do this either after a hysterectomy or pre-operatively when the disease is very advanced and you note involvement of the bladder or bowel. If the cervix shows macroscopic tumour of endometrial carcinoma, the stage is at least IIB.

Endometrial carcinoma	**Stage**
In situ	0
Confined to corpus Tumour limited to the endometrium Up to <½ of myometrium >½ of myometrium	I IA IB IC
Extension to cervix Endocervical glandular only Cervical stroma	II IIA IIB
Local and/or regional as specified below: Serosa/adnexa/positive peritoneal cytology Vaginal involvement Regional lymph node metastasis	III IIIA IIIB IIIC
Mucosa of bladder/bowel Distant metastasis	IVA IVB

DIAGNOSIS. Confirm the diagnosis by doing a suction curettage under LA, if possible. Otherwise, do a D&C and send scrapings for histology. If you have a vaginal ultrasound probe, measure the 2 layers of endometrium (the basal and functional layers, the latter being shed during menstruation). If they add up to >4mm, this is indicative of carcinoma, but you still need histology.

MANAGEMENT. This depends on staging and histological grade. If you have post-operative radiotherapy available, you don't need to take a vaginal cuff with the hysterectomy; otherwise this is advisable. You need to perform a *total* abdominal hysterectomy (TAH) with bilateral salpingo-oophorectomy (BSO), and add post-operative radiotherapy (POR) and lymph node ablation as well in certain cases.
Performing inadequate surgery will not benefit the patient.

Stage	**Grade**	**Age**	**Treatment**
IA, IB	1, 2		TAH+BSO
IA, IB	3	< 60yrs	TAH+BSO
IA, IB	3	≥60yrs	TAH+BSO + POR
IC	1, 2	< 60yrs	TAH+BSO
	1, 2	≥60yrs	TAH+BSO + POR
IC	3		TAH+BSO + POR
IIA	1, 2, 3		TAH+BSO + POR
IIB-III	1, 2, 3		Radical TAH+BSO + Lymphadenectomy + POR

Alternatively, especially with a well-differentiated tumour (grade 1) where operative cure is not an option, or there is recurrence, you can stabilize the cancer by high-dose progestogens, using 150mg medroxyprogesterone acetate od for 3wks, then weekly for 12wks, then monthly.

SURVIVAL
The 5yr survival after surgery and radiotherapy worsens for each higher stage: Overall under the best circumstances all stages combined, the survival is 75%. Recurrences occur mostly in the vaginal vault and can be temporarily controlled by radiotherapy.

23.9 Ovarian cysts and tumours

Many ovarian tumours are cystic, but many cysts are not malignant and some tumours are not cysts. Their classification is complex; this is a simplified scheme.

(a) Benign:
(1) Functional cysts of the follicles and corpus luteum.
(2) Benign serous or mucinous cystadenomas.
(3) Dermoid cyst (contains tissue derived from embryonal germ layers, such as hair or teeth).
(4) Unclassified benign cysts (simple cysts).
(5) Endometriosis ('chocolate, *i.e.* brown, cysts').

(b) Malignant:
(1) Serous or mucinous cystadenocarcinomas.
(2) Stromal cell or sex cord tumours (which can produce high levels of oestrogens or androgens).
(3) Germ cell tumours.
(4) Metastatic tumours (from bowel, or breast).
(5) Burkitt's lymphoma.
(6) Other, rarer tumours.

(c) Borderline: tumours that look benign but metastasize or recur 20yrs later.

(d) Pseudocysts are post-inflammatory collections of fluid between adhesions in the pelvis (23.1), and are not true ovarian cysts; but the distinction is not always easy, even at operation.

Most common ovarian malignancies are epithelial carcinomas. They arise in the ovaries or tubes. They metastasize in the peritoneal cavity, most commonly on and around the ovaries, in the pelvis, in the omentum and eventually in the whole peritoneal cavity. They also metastasize to the retroperitoneal lymph nodes. Finally they metastasize to the lungs. They rarely metastasize to the liver. If liver metastases are obvious, consider another primary malignancy.

The greatest challenge when dealing with an ovarian tumour is to know whether it is malignant or not. Manage a benign cyst conservatively *e.g.* by cystectomy in young women or adnexectomy in older women. Expectant management is also justifiable.

Try to drain very large cysts through a small incision and then remove the cysts. The final diagnosis (pathology) will only become clear after the operation.

An ovarian cyst can be of any size, from 1-30cm or more in diameter, and may:
(1) Present as a mass, or as abdominal distension, which may be massive.
(2) Cause abdominal pain due to torsion.
(3) Be confused with pregnancy.

Very large cysts, the size of term pregnancies, are usually benign and cause no pain. Cysts <5cm are usually benign. Postmenopausal tumours are more often malignant. Pre-pubertal tumours are usually germ cell malignancies.

Before the operation, make a malignancy risk assessment. The most useful diagnostic tool is ultrasound (38.2k). If ascites is already obvious clinically, then most often the disease has spread through the whole abdomen.

EXAMINATION bimanually reveals a round, solid or cystic mass, which is dull to percussion and separate from the uterus.

ULTRASOUND (38.2k, especially with a vaginal probe) will usually clinch the diagnosis, although if the cyst is very large, it may be difficult to be sure where it originates.
A benign cyst is usually: unilateral, single, and echo-translucent (just like water). Completely smooth cysts are usually benign functional, or endometriomas. If white marks (solid parts) are also present, they are dermoid cysts or occasionally cystadenocarcinomas.
Malignant cysts are usually: bilateral, multicystic, showing solid and cystic areas, especially with papillary formations inside.
Ascites is a strong sign (in the absence of suggestions of tuberculosis or cirrhosis) of malignancy. A cyst of >5cm diameter is significant. Solid ovarian tumours are more likely to be malignant, and produce early metastases.

A RADIOGRAPH may show teeth inside a dermoid cyst.

DIFFERENTIAL DIAGNOSIS varies according to the presentation:

As a simple cyst:
(1) Pregnancy.
(2) A distended bladder, which may contain 5L of urine.
(3) Pseudocysts.
(4) Hydrosalpinx (blocked Fallopian tube full of liquid).
(5) Fibroids.
(6) A chronic ectopic gestation (haematocoele).
(7) A broad ligament cyst arising from the Wolffian ducts (which contribute to male genital development but regress in females).
(8) An appendix mass, or a small-gut mass.
(9) Mesenteric cysts.
(10) An enlarged spleen with a long pedicle.
(11) Hydronephrosis.
(12) Hydatid cyst.

As torsion:
(1) Appendicitis or an appendix mass (14.1).
(2) Acute ectopic gestation (20.6).
(3) Degeneration, bleeding, or infection in a fibroid (23.7).
(4) A mass due to PID (23.1).

As a huge cyst:
(1) Ascites (dullness to percussion in the flanks, rather than in the centre of the abdomen).
(2) Obesity (fat is usually generalized).
(3) Pregnancy.
(4) An enormous bladder from chronic urinary retention.
(5) Haemato-colpos/-metrium from an imperforate hymen, so the blood of menstruation cannot escape (23.17).

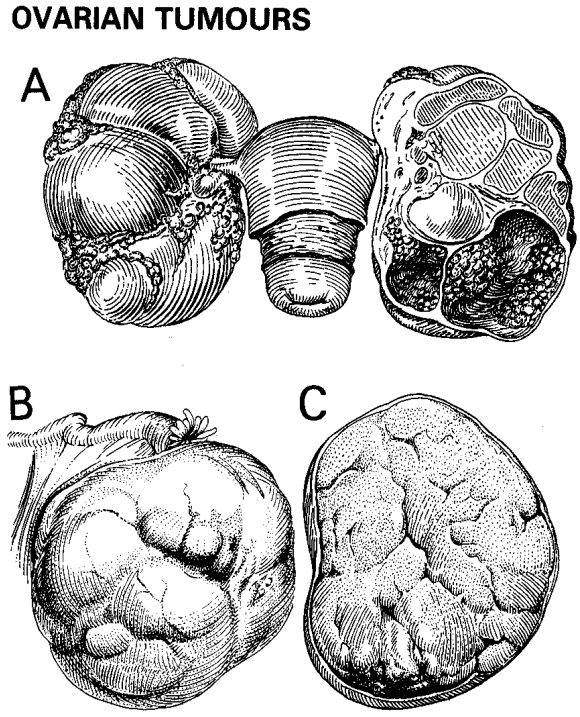


Fig. 23-11 OVARIAN TUMOURS.
A, a pseudomucinous papillary cystadeoncarcinoma shown in cross-section on the right. B, a solid primary carcinoma. C, the same carcinoma in cross-section.
Adapted from a drawing by Frank Netter with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

MANAGEMENT OF AN OVARIAN CYST

If a cyst is <5cm diameter, **and the patient is <50yrs**, it is usually a functional (follicular or luteal) cyst, and may be associated with dysfunctional uterine bleeding (23.3). The simple rule is that a cyst like this need not come out. Review the patient in 8-12wks, and only operate if the cyst persists. Most functional cysts will have disappeared. If you find such a cyst at laparotomy for some other condition, leave it. If it looks benign, open it.

If the patient is <15yrs (before menarche), many cysts are benign, but there is an increased risk of malignancy, which is sometimes low-grade. At operation the decision to remove the ovaries is particularly difficult. Only remove large (>10cm), solid ovarian tumours, and be sure to send them for histology. If ovarian cystectomy is not possible, remove only one ovary!

If the patient is 15-35yrs, and the cyst is >5cm diameter, it is probably a dermoid, especially if it is firm. Perform an oophorectomy. If it is bilateral (15%), try to leave some ovarian tissue by doing ovarian cystectomies if there are no signs of malignancy.

If the patient is 30-55yrs and the cyst is >10cm diameter, it is likely to be a cystadenoma, which may be bilateral (20%). The contents may be serous, and there may be papilliferous growths inside its wall (a sign of dysplasia or frank malignancy) or outside (virtually diagnostic of malignancy). Large cysts are more likely to be malignant than small ones.

If the cyst is very large, its contents are likely to be mucinous. Malignant change is unusual. If however the mucin spills into the peritoneum, further benign tumours may re-grow all over the abdomen (myxoma peritonei) and cause dense adhesions. Some large cysts are serous cystadenomas; some are cystadenocarcinomas.

Try to remove a cyst without spilling the fluid, because if you do, you may spread a malignant tumour and harm the patient greatly. If a tumour has not spread through the wall of a cyst, its removal intact without spilling will usually be curative. Aspirating a cyst (using a purse-string suture around the suction device) before you try to remove it, makes it easier to remove, requires a smaller incision lower in the abdomen, *but may not make dissection of adhesions any easier.* However, this is likely nevertheless to cause some spillage, whose effects can, though, be minimized by packing off the rest of the abdomen.

If you decide not to aspirate a cyst, you will obviously cause no spillage unless it bursts or you cut into it: this is then a much worse spillage than if you had aspirated it in the first place! Even large cysts are, however, not too difficult to remove intact, if you make an incision large enough. However, *don't make a huge incision to avoid the minor risk of spillage, for example in a frail elderly lady*. Ultrasound will give good information whether a cyst is likely to be malignant (38.2k).

Most ovarian cysts have few adhesions. If adhesions are dense, you may be dealing with:
(1) Old PID.
(2) A malignant cyst in which the growth is already spreading into the peritoneum.
(3) Previous peritonitis that has left adhesions which have stuck the cyst to the peritoneum.
(4) A cyst which has previously undergone torsion.
(5) Endometriosis.

If there are dense adhesions, it might be possible to shell out a benign cyst leaving the extreme outer layer, thus preventing damage to the surrounding tissues. Sometimes you are forced to empty a large cyst first because otherwise it is impossible to reach the blood vessels behind it.

N.B. Don't ruin a suction machine by letting the drainage bottle overflow and allowing foam or fluid spill into the mechanism while you attempt to drain a huge ovarian cyst!

If the other ovary is also cystic, and the patient is relatively young, perform an ovarian cystectomy, unless there is a suspicion of malignancy. Avoid removing both ovaries on a woman <40yrs for bilateral benign tumours (usually dermoids containing hair or teeth). *Remember that operating on a pseudocyst, or a cyst in the broad ligament, is particularly hazardous*, because of bleeding.

N.B. If you have to remove both ovaries, the cheapest hormone replacement therapy is the contraceptive pill.

If the patient is postmenopausal, the risk of malignancy is increased. Be prepared to perform a hysterectomy, at the time that you remove the cyst.

N.B. Suspect malignancy on the combination of these factors:
(1) a woman >40yrs,
(2) a solid or lobulated tumour,
(3) papillary excrescences (23-12A) on the ovarian surface (especially) or inside it,
(4) ascites,
(5) metastatic deposits on the peritoneum,
(6) a fixed and immobile cyst.

If the patient is pregnant, delay surgery till between 16-24wks gestation or later at a Caesarean section if obstructed labour is likely.

MANAGEMENT OF AN OVARIAN TUMOUR

If there is a bilateral, papilliferous, or obviously malignant ovarian tumour, what you should do depends on your skills, and how far the tumour has spread:
(1) Perform a bilateral adnexectomy (removal of the cyst with the ovary and tubes) if there is no peritoneal spread, and no tumour on the uterine surface.
(2) Perform a bilateral adnexectomy if you can clear limited localized tumour spread.
(3) Biopsy a peritoneal deposit if there is wide peritoneal spread. *Don't attempt heroic pelvic surgery*, because you will not be able to cure the patient thereby.

If there is a palpable mass, ascites, or oedema of the legs (due to lymphatic obstruction from peritoneal deposits), consider the possibility of a solid adeno- or undifferentiated carcinoma of the ovary, which typically presents like this. It is often bilateral, and the prognosis is poor whatever you do.

If the tumour is solid, remember the possibility that it may be a fibroma, which is benign, but can cause ascites and a pleural effusion (Meig's syndrome). Perform an oophorectomy. If you remove the tumour, the fluid resolves. It may be a thecoma, an oestrogen-producing generally benign tumour producing lipid-rich, yellow-orange fluid, or granulosa cell tumour, which may be malignant and present with abnormal uterine bleeding and in 20% with endometrial cancer. Most patients are >40yrs.

If you are in an endemic area and the patient is 10-25yrs, remember Burkitt's lymphoma (17.6), which is often bilateral, or hydatid cyst (15.12).

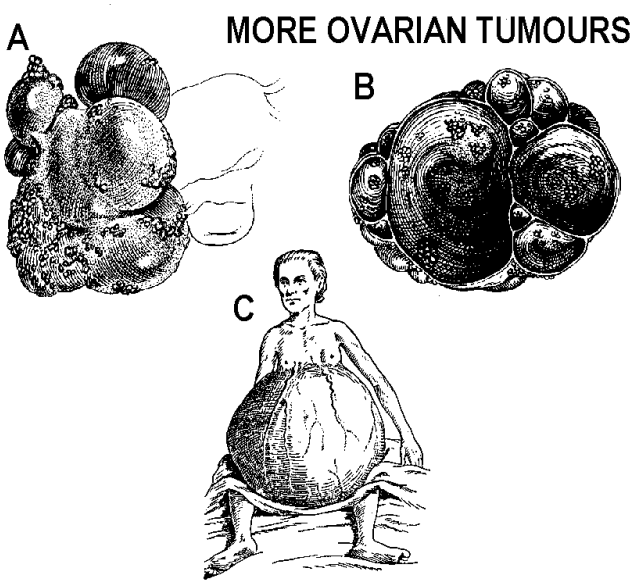


Fig. 23-12 MORE OVARIAN TUMOURS.
A, probably a papillary serous cystadenocarcinoma. B, the same in cross-section. C, a very large ovarian cyst showing dilated veins on the abdominal wall.
A,B, adapted from drawings by Frank Netter with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland. C, after Young J. A Textbook of Gynaecology A&C Black 5th ed 1939.

INDICATIONS FOR SURGERY

(1) Treatment or prevention of complications: torsion, bleeding, or infection.
(2) Suspected malignancy.
(3) Discomfort due to size.
(4) Causing obstructed labour.
CAUTION! Infertility is not an indication.

OOPHORECTOMY FOR SMALLER CYSTS (GRADE 3.2)

INCISION. Make a midline or Pfannenstiel incision, big enough to allow you to insert your hand, and to remove the cyst intact.

If you are sure the cyst is benign, make an incision just small enough to aspirate the cyst, and perform an ovarian cystectomy (see below). If not, feel its whole surface for adhesions. Search for metastases in the rest of the peritoneal cavity, over the surface of the liver, and under the right diaphragm.

When the cyst is free of adhesions, deliver it through the abdominal wound, and hand it to your assistant, taking care not to pull on its pedicle, which may be so thin that it easily tears, causing the proximal end to slip into the pelvis and bleed (23-13C).

CAUTION! Before you remove the cyst with the ovary, examine the other ovary.

The pedicle of an ovarian cyst consists of:
(1) the infundibulo-pelvic ligament and ovarian vessels superiorly (21-18, 23-20, 23-21),
(2) the ovarian ligament, which connects it to the uterus,
(3) part of the broad ligament, and
(4) frequently, the Fallopian tube.

BEWARE OF THE URETER, which runs under the infundibulo-pelvic ligament more deeply and posteriorly.

If the pedicle is wide (often it is not), clamp it with several clamps, taking a bite of ≤2cm in each of them. Cut through the pedicle at some distance from each clamp; it will be less likely to slip off if you do this.

Transfix the pedicle in each clamp with double '2' absorbable sutures, taking care to avoid the plexus of veins as you insert the needle. Finally, ask your assistant to hold the clamps, and pass a further ligature round the entire pedicle. This will tie any veins which may have escaped the other ligatures. Swab the stump, and, if bleeding has been controlled, cut the ligatures. Remove the cyst from the operation site, and ask an unscrubbed assistant to open it. If it looks malignant and she is >40yrs, perform a bilateral adnexectomy (see below). If the patient is younger, wait for histological confirmation of malignancy, and advise more radical surgery later if necessary.

OOPHORECTOMY FOR A VERY LARGE CYST (GRADE 3.2)

POSITION
Lay the patient with a sandbag under the right buttock. This prevents the supine hypotensive syndrome, if she lies on her back.

INCISION
Make a midline incision. If you hope to remove the cyst intact, make it ≥5cm longer than the diameter of the cyst.

If you are not sure if you can remove the cyst intact with the ovary, or whether this is really necessary, make the incision just long enough to examine it properly, separating such adhesions as you can see, without too forceful traction on the wound. If you cannot dissect further safely, enlarge the incision to see the outline of the cyst and any adhesions. Aspirating fluid may help you to deliver it through the abdominal wall, but seldom helps in dissecting adhesions. A flabby cyst has an edge which is difficult to define, so that vital structures, such as the ureter, are more easily cut. If you do decide to aspirate fluid, use a powerful sucker attached to some tubing you can secure to the cyst wall; *don't contaminate the operation site with the fluid you have aspirated!*

CAUTION! A wound which is too small is dangerous because you cannot dissect safely, and you are obliged to exert excessive traction. A large wound is more likely to dehisce or give rise to other complications.

Remove the cyst by clamping its pedicle (23-13). Be careful not to pull it so hard that you tear this.

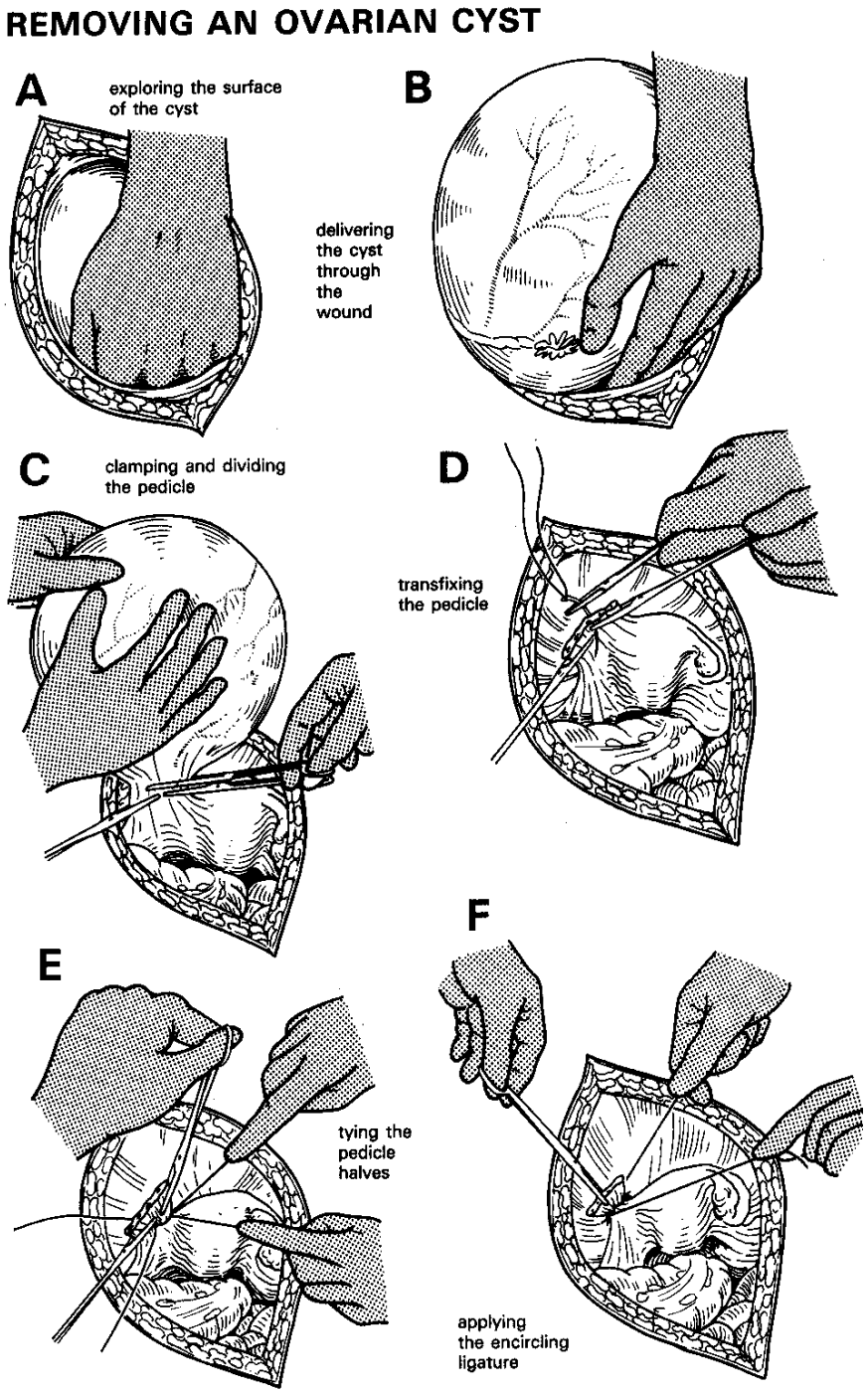


Fig. 23-13 REMOVING A LARGE OVARIAN CYST.
A, explore the surface of the cyst. B, deliver the cyst without rupturing it. C, clamp and divide the pedicle. D, transfix the pedicle. E, tie the pedicle in halves. F, apply the encircling ligature.
After Bonney V. Gynaecological Surgery, Baillière 1964 Figs 4.29-34 with kind permission.

OVARIAN CYSTECTOMY (GRADE 3.2): this removes the cyst but leaves behind the tissue of the ovary.

INDICATIONS
(1) The patient is <40yrs, and the other ovary is also damaged.
(2) The cyst is >5cm.
(3) The cyst must be benign, a reasonable amount of normal ovarian tissue should be present.

N.B. If the cystic ovary is the only remaining one, it is not important if its tube is intact or not. The patient needs its endocrine function, regardless of possible fertility. You may be able to shell out even quite large cysts, and retain some ovarian tissue. The cyst lies in the substance of the ovary and is covered by the ovarian capsule.

N.B. Don't remove an ovarian cyst early in pregnancy: the corpus luteum needed for its progesterone to prevent miscarriage may be in that cyst or ovary!

INCISION

Make a midline or Pfannenstiel incision. Cut around the edge of the cyst, well away from the remaining mass of the normal ovary. Using scissors or fingers, dissect between the cyst and the ovarian tissue. Control bleeding with 2/0 absorbable, and close the outer layer of the ovary with continuous locking sutures. Cover any raw surfaces with omentum, to avoid adhesions.

SALPINGO-OOPHORECTOMY (ADNEXECTOMY) (GRADE 3.3): this removes the Fallopian tube and ovary.

INDICATIONS

(1) Both tube and ovary have been affected by torsion, bleeding, or infection.
(2) A possibility of malignancy.
(3) Extensive adhesions between tube and ovary.
(4) Combined with a hysterectomy in a woman >50yrs (usually means a bilateral adnexectomy).

N.B. The tube and ovary receive their blood from 2 sources which anastomose with one another, the ovarian vessels in the infundibulo-pelvic ligament and the ascending branches of the uterine vessels (21-18, 22-14, 23-20, 23-21).

INCISION

Make a midline or Pfannenstiel incision. Carefully divide any adhesions between the ovary and broad ligament, approaching them from below and behind. Raise the tube and ovary, find the infundibulo-pelvic ligament, and identify the ureter, so that you can avoid it. Clamp, divide, and tie the ovarian vessels in the infundibulo-pelvic ligament. Clamp, divide, and tie the ovarian ligament. Clamp, divide, and tie the tube.

CAUTION! Be sure not to tie the ureter. This is not a problem if the structures are mobile. But if there are adhesions, and especially if the ovary and tube have stuck to the back of the broad ligament, be sure to identify and mobilize them before you resect them.

Difficulties with ovarian cysts

If there are extensive adhesions, this may be a post-inflammatory cyst. *Don't try to deliver the tumour until you have divided the adhesions*, or you may lacerate the bowel or tear large veins. Separate them using your hands, swabs, or scissors (*not a scalpel!*). Gently pass your hand between the cyst wall and the floor of the pelvis. *Don't mistake the parietal peritoneum for the cyst wall. Don't tie off any colon when you tie off adhesions.*

CAUTION! It is safer to leave a little cyst wall on the bowel or the bladder, than to remove a little bowel or bladder with the cyst wall.

There may be many of these cystic collections; just aspirate them rather than trying to remove them all.

If the cyst is not freely mobile, but seems to be embedded in the broad ligament, it may be arising from the remains of the Wolffian duct. Removing it may be difficult. It may be stuck to the broad ligament or inside it. The distinction is usually unimportant. If it is inside the ligament:

(1) *Be sure to avoid the ureter,* which may run anywhere over the cyst.
(2) *Don't damage the venous plexuses in this region*. Study the anatomy carefully before you start.

If the cyst does not shell out easily, and extends down close to the ureter, you would be wise to remove as much as you can, and leave the remains open to the peritoneal cavity (marsupialization). Take a biopsy to exclude malignancy.

If you can define the cyst clearly by finger dissection, and are able to push the ureter out of the way, you may be able to remove it completely. It is covered by peritoneum which you need to dissect off. Divide the round ligament on the same side, to open up the broad ligament. Then dissect off the peritoneum posteriorly, until you reach the ovarian vessels in the infundibulo-pelvic ligament. Tie them. Then dissect anteriorly and medially, and divide the tube and ovarian ligament close to the uterus. Finally, slowly and carefully dissect the cyst from the posterior leaf of the broad ligament, so as to avoid the ureter.

If the infundibulo-pelvic ligament is grossly thickened, so that the ovarian vessels are difficult to distinguish from the ureter, open up the peritoneal tissues lateral to them, and extend the incision towards the pelvic brim. Grasp the ovarian vessels and draw them medially. You will then see the ureter attached to the peritoneum, crossing the common iliac artery.

Difficulties with giant ovarian cysts

If the patient develops cardiac failure, which may be delayed for a day or two post-operatively, treat her with a diuretic.

If the patient develops respiratory failure, due to the paradoxical movement of the diaphragm, which is lax and overstretched, now that the cyst has been removed, treat her with oxygen and sit her up.

If the abdomen distends post-operatively, it is probably due to ileus. Mobilize the patient and if this fails, insert a nasogastric tube for 1-2 days and infuse IV fluids till bowel function returns.

If the abdomen is abnormally lax, apply an efficient binder post-operatively.

CAUTION! Don't be tempted to resect any redundant abdominal wall. This will make the operation much more extensive and bloody. Abdominal compartment syndrome may result (11.10).

CHEMOTHERAPY without a complete debulking operation is only palliative for ovarian malignancy. Chemotherapy may be useful however in treating ascites, and may prolong survival in patients in Stages II (peritoneal spread within the pelvis) and III (peritoneal spread throughout the abdomen). It also delays the onset of this distressing problem. Up to 40% of patients respond to cyclophosphamide $1{\cdot}5 g/m^2$ od every 21 days for up to 6 courses, and more to cisplatin (37.4).

23.10 Gestational trophoblastic disease (GTD)

One of the most important advances in oncology was the discovery that many cases of choriocarcinoma could be cured with methotrexate. In a normal pregnancy many placental (trophoblastic) cells are carried to the lungs, but don't grow there: they have a normal DNA structure. Trophoblast is only malignant when it grows outside the uterus, or abnormally within it. There are various possibilities:

(a) Benign GTD is an overgrowth of the trophoblast, in which the chorionic villi form fluid-filled, grape-like vesicles, up to 1cm in diameter. Such a MOLE can be *complete* without an embryo (more common), or *partial* (less common), when some foetal tissues are present.

A complete hydatidiform mole develops mainly because a sperm has fertilized an 'empty' egg (contains no nucleus or DNA). All the genetic material comes from the father's sperm. Therefore, there is no foetal tissue. Up to 20% of patients with complete moles will need additional surgery or chemotherapy after initial evacuation of the mole because of the presence of persistent trophoblastic material. A small percentage of complete moles may develop into choriocarcinoma, which is a malignant form of GTD.

A partial hydatidiform mole develops when 2 sperms fertilize a normal egg (triploidy). These contain some foetal tissue (most often blood vessels containing immature nucleated red cells, as opposed to mature red blood cells in adults, which appear as denucleated discs). But this tissue is often mixed with the trophoblastic tissue. No viable foetus is formed. Only a small percentage of patients with partial moles need further treatment after initial evacuation. Partial moles rarely develop into malignant GTD.

Moles of either kind can present as a miscarriage, or an ectopic gestation. In a binovular (non-identical) twin pregnancy, one twin may be normal and the other a mole.

Moles vary widely in incidence from 1:120 to 1:2000 pregnancies, and are more common in Asia than they are elsewhere. Thus haemoptysis in a female in East Asia may be more likely to indicate GTD than TB.

After an evacuation of GTD, there may be
(1) complete recovery (80-95%),
(2) non-metastasizing trophoblastic neoplastic change (invasive mole) (5-15%), or
(3) metastasizing trophoblastic neoplastic change (choriocarcinoma) (0-5%), of either high or low risk. Subsequent new gestation will be a mole in c.1·5% of cases.

Early diagnosis, effective treatment, and energetic follow-up are essential. GTD is treatable without too much difficulty, but a choriocarcinoma is different. Try to make sure choriocarcinoma patients get treatment in a recognized centre in your country where enough experience is present to monitor patients who have a potentially fatal disease but which can be cured with potent drugs.

(i) History and examination
Enquire about the times of bleeding, and ask about the passage of tissue ('grapes') vaginally. Note the size and feel of the uterus, especially changes after 2wks. Measure the fundal height. Listen for the absence of the foetal heart (you should hear it at 18wks in a normal pregnancy). Doppler foetal heart monitoring is useful. The presence of a foetal heart reduces the probability of a mole, but does not exclude the much rarer occurrence of a mole in a twin pregnancy. Look for signs of gestational hypertension.

(ii) Presentation
Commonly <20 or >40yrs, usually presenting before 18wks gestation with:
(1) Rapid enlargement of the uterus, which feels abnormally soft.
(2) Excessive nausea and vomiting.
(3) Vaginal bleeding which starts at the 6^{th}-8^{th} wk.
(4) Passage of vesicles though the cervix.
(5) Enlarged, often palpable cystic ovaries (50%).
(6) Signs of gestational hypertension.
(7) Hyperthyroidism (occasionally)
or any combination of these signs.
If gestation exceeds 18wks, also the inability to palpate foetal parts and the lack of perceived foetal movements.

(iii) Ultrasound (38.2k) gives a classic appearance of multiple cysts (or 'snowstorm') in the uterus but even with the best machines with a vaginal transducer and in the best hands, the diagnosis is missed in 50% of cases and confused with an incomplete or missed miscarriage. Invasion of the myometrium can sometimes be seen and cystic ovaries are often present (23-14).

In Asia histological examination should, resources permitting, be performed of every evacuated incomplete/missed miscarriage in case an embryo was never seen. In Africa that should also be the case ideally, but you should insist on it for women who need a re-evacuation with the diagnosis of incomplete evacuation.

(iv) Tests for gonadotrophin
The level of β-HCG in the blood is somewhat related to the prognosis but irrelevant on the whole in most circumstances for diagnosis. A mole needs evacuation; after that there will be a dramatic drop in HCG anyway.

(v) Differential diagnosis includes:
(1) a multiple pregnancy,
(2) a miscarriage, especially a missed miscarriage,
(3) acute polyhydramnios,
(4) retention of urine with a retroverted gravid uterus,
(5) a subacute or chronic ectopic gestation.

(vi) Management
ANAEMIA. Correct severe anaemia by blood transfusion. If the Hb is <50g/L, transfuse packed cells slowly, and precede each unit with furosemide 20mg unless there is rapid blood loss.

N.B. High levels of β-HCG may be difficult to detect except in diluted urine (see below).

N.B. High levels of β-HCG may have a thyrotoxic effect (25.2)!

EVACUATE THE UTERUS
Cross-match blood. It is advisable to use at least 2 drugs (3, if available) to limit blood loss. Start with high doses of oxytocin IV (10U/500ml, running at 30 drops/min), ergometrine 0·5mg IM and misoprostol 800μg PR.

You can then wait and see, but if you have some expertise, a suction curettage after you have felt the uterus become as hard as a stone is then best.

You are then present with the patient when it is most important to be so. If the patient has a serious tachycardia without severe anaemia, 8mg salbutamol orally 30mins before the procedure might prevent a thyrotoxic crisis.

As soon as you start suction or even dilatation (never use a sound), bleeding will start and it will only stop when the uterus is empty and very well contracted. You can sometimes use your fingers to check if it is empty.

Very important points: Make sure *before you start* that the suction machine works properly (at maximum capacity): you must be able to lift the tubing with your finger attached by vacuum suction to the end of the tube. It must also have a large diameter and high flow rate. An aspirator is no good because the syringe will fill up with fluid and blood in a second and you will not be able to empty the uterus. Blood will then collect faster in the uterus than you are able to evacuate it, let alone allow you to remove the mole. Use wide-bore tubing because the tube must not block halfway through the procedure when the patient is bleeding. Also, the Karman cannula should be large, preferably 12mm in a 16wk-sized uterus or otherwise ≥10mm if in a uterus <14wk size.

You should be able to empty, change, and reconnect the bottle fast or have 2 excellent suction machines in theatre.

N.B. This procedure can be done slickly (emptying the uterus in 30secs), *but it can also become a disaster if you have not checked the equipment!*

CAUTION! Take great care not to perforate the uterus.

If you think that evacuation is incomplete, or if bleeding continues slightly, accept this, and repeat the curettage in 3 days. Torrential bleeding can occur, so have 2 units of blood available, but don't transfuse unless forced to do so.

After you have evacuated the uterus, you may feel cystic ovaries: leave them alone. Repeat the evacuation if there is still bleeding after 7-10 days.

If you still cannot control the bleeding, take the patient to theatre to apply a B-Lynch suture (22-3). This will stop most bleeding and not interfere with a pregnancy later.

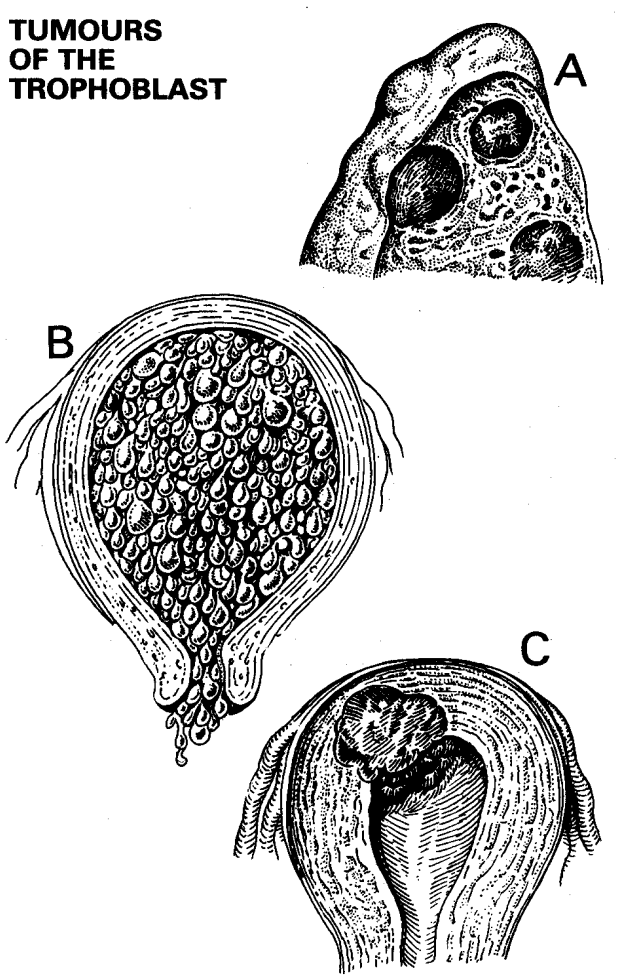


Fig. 23-14 TROPHOBLASTIC TUMOURS.
A, a choriocarcinoma has already metastasized to the lungs (typical 'cannonball' lesions). B, a hydatidiform mole. C, a choriocarcinoma invading the wall of the uterus.
Adapted from drawings by Frank Netter with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

If even then, bleeding persists, tie a tourniquet round the base of the uterus (as for a myomectomy: 23.7) and leave it for 12-24h. Then re-open the abdomen to remove the dead uterus; this transforms a horrendous procedure into an easy one!

N.B. Avoid a hysterectomy without the tourniquet as the GTD may have invaded the large vessels. *During a hysterectomy clamps and sutures will cut through and you might be unable to stop the haemorrhage.*

(vii) Follow-up is essential to identify those patients (up to 20%) who need cytotoxic drugs. However, >98% of women who become pregnant following a cured molar conception will not have a further GTD and these pregnancies are at no increased risk of other obstetric complications. You can follow the expected drop in β-HCG by routine urinary pregnancy tests. If a routine modern monoclonal sensitive pregnancy test is -ve (can take 2-3 months), then the trophoblastic disease has most likely virtually disappeared. You should however repeat it then 2wkly for 3 months (while the patient is using proper contraception) and then monthly for 1yr. If it is still -ve, you can consider her cured. She can stop contraception if she wants, and become pregnant.

If the pregnancy test stays +ve, then try a 2nd suction evacuation carefully (even if an ultrasound scan indicates a normal empty uterus) and send the material for histology. If after that, the pregnancy test stays +ve, it is then time for a work-up (chest radiograph, ultrasound of uterus, kidneys, liver, laboratory) and a quantitative β-HCG test because the patient will probably need chemotherapy. You need a baseline β-HCG level to see later how successful the chemotherapy is. The β-HCG value should preferably be reduced by ≥90% after every course.
Commercial kits measuring the quantity of β-HCG in serum are easiest but expensive. You could try pregnancy tests on serial dilutions of a 24h urine specimen (keeping it cool during collection) to detect the greatest dilution showing a +ve result. This is an inexpensive way of doing a quantitative test, and although it is not ideal, *you have to do it carefully.*
A modern urine test is just +ve with 25mIU/ml (check the leaflet in the box). This means that if the test is just +ve and you have collected 1½L of urine, there are 37·5IU in a 24h collection. If x10 dilution is just +ve, there are 375IU present.

You must insist on a reliable form of family planning method for a year. If the husband cannot be relied on to use a condom (unfortunately usual), either insert an IUD, even though its side-effects may be confused with choriocarcinoma, or use medroxy-progesterone.

Suspect a trophoblastic neoplasm if during follow-up:
(1) dark vaginal bleeding continues,
(2) the uterus remains large after evacuation or delivery,
(3) you see a haemorrhagic nodule near the urethral meatus, vaginal vault or cervix,
(4) amenorrhoea continues.
Establish the diagnosis by ultrasound and by measuring serial β-HCG levels as above.

(b) Persistent/invasive GTD (invasive mole) is a tumour-like process which invades the myometrium and arises from GTD, more commonly from a complete mole.

The risk of this developing is greater if:
(1) there was a long time (>4 months) between the last menstrual period and evacuation,
(2) the uterus has become very large,
(3) the woman is >40yrs,
(4) the woman has had GTD in the past. These lesions occasionally regress spontaneously.

Diagnosis
A titre of β-HCG of >200IU in a 24h specimen after 2-3 months, or a rising or unimproved titre after 2 months, is abnormal.
Look for absence of metastatic disease in the vagina, pelvis, liver, brain, and chest (23-14A).
The differential diagnosis includes the incomplete evacuation of a normal placenta, placenta accreta, or ectopic gestation.
If you make the diagnosis within 4 months of delivery, you have a 95% chance of cure.

Treat with oral methotrexate 15mg/m^2 od in courses of 4 days. Repeat the courses every 14 days for 2 courses after the β-HCG is normal, to a maximum of 6 courses.
If the β-HCG is still >2000IU/24h after 6 courses of methotrexate, start actinomycin D 2mg/m^2 (or 1mg on days 1 and 3), one course every 3wks. Repeat this every 3wks for 2 courses after the β-HCG test is -ve.
If the β-HCG test remains +ve, start therapy, as for choriocarcinoma (see below).

(c) A metastasizing trophoblastic neoplasm (chorio-carcinoma) arises from the trophoblast after a live birth (25%), a stillbirth or miscarriage (25%), an ectopic gestation, or a hydatidiform mole (50%). These may all present as heavy irregular bleeding, often mistaken for an incomplete miscarriage.

PRESENTATION
(1) A history of heavy irregular bleeding, sometimes following a miscarriage, especially with the need for repeated evacuation, particularly if an embryo was never seen by ultrasound or within the evacuated material.
(2) Persistent amenorrhoea following GTD.
(3) A haemorrhagic nodule on the urethral meatus, vaginal vault or cervix.
(4) Cough, chest pain, 'unresolved pneumonia' or a bloody pleural effusion.
(5) An acute haemoperitoneum like a ruptured ectopic gestation, due to perforation of the uterus by the tumour.
(6) Neurological symptoms.
(7) Gastrointestinal bleeding.
(8) Haematuria.

ULTRASOUND (38.2k) shows an irregularity in the uterine wall.

CHEST RADIOGRAPH may show round metastases.

CAUTION! If the choriocarcinoma arose from a hydatidiform mole, you will already have evacuated it.
If it presented in other ways, *don't do a diagnostic curettage*, because:
(1) A 'negative' one does not exclude the diagnosis.
(2) You can easily perforate the uterus, or cause catastrophic bleeding.
(3) You may spread the tumour.

Prognosis. All untreated patients die from multiple metastases. Try to diagnose this disease early. You may, if you cannot refer to a special centre, treat 'low risk' patients, but don't attempt to treat 'high risk' patients who need different complicated expensive chemotherapy.

Repeat the courses every 14 days (*i.e.* start day 15) as long as the β-HCG decreases by a factor of 10 by every course; repeat 2 more courses after a sensitive β-HCG test is negative.

Low risk, after normalization of β-HCG, 2 more courses	1. Index pregnancy was a mole or miscarriage. 2. Metastases only in vagina or lungs. 3. No previous chemotherapy. 4. Interval between evacuation and chemotherapy <12 months.
High risk, after normalization of β-HCG, 3 more courses	1. Insufficient success with previously administered chemotherapy. 2. Metastases in >1 organ outside the uterus. 3. Metastases in one of: liver, spleen, kidney, GI tract, brain, bones. 4. Index pregnancy was a term delivery. 5. Interval between evacuation and chemotherapy >12 months.

LOW-RISK PATIENTS:

Methotrexate (MTX)	1mg/kg	IM	day 1,3,5,7
Folinic acid (leucovorin)	15mg	oral	24h after methotrexate on day 2,4,6,8

If this is unsuccessful, and if β-HCG (in blood) is <100IU, use:

Actinomycin D	0·5mg	IV	day 1,3,5,7

If this is unsuccessful, the patient is re-classified as high-risk and needs treatment in a specialist centre.

(d) Placental-site trophoblastic tumour is a rare form of GTD that develops where the placenta attaches to the uterus. This tumour most often develops after a normal pregnancy or miscarriage. Most placental-site tumours don't spread to other sites in the body. Sometimes, though, these tumours penetrate the muscle layer of the uterus. Although most forms of GTD are very sensitive to chemotherapy, placental-site tumours are not and, therefore, they must be completely removed by surgery.

23.11 Uterine prolapse

Childbirth may so injure the pelvic organs that the uterus, the bladder or the rectum may prolapse, either singly or in combination. In nullipara, it also occurs after the menopause. There are large regional differences in the prevalence of prolapse, most likely related to the quality of the ligaments. In Southern Africa prolapse is rare but stress incontinence even more so, possibly related to the ubiquitous use of the squatting position, which strengthens pelvic floor muscles. Women working on the land with long skirts can easily squat frequently and keep their bladder content small.

Travelling long distances in buses or taxis might become embarrassing; this also applies to shopping in circumstances where there are few toilets for clients. However, absorbent 'makapads' can be made from dried crushed raw papyrus processed into a thick paste with waste paper and water, cut to size and placed in absorbent inserts.

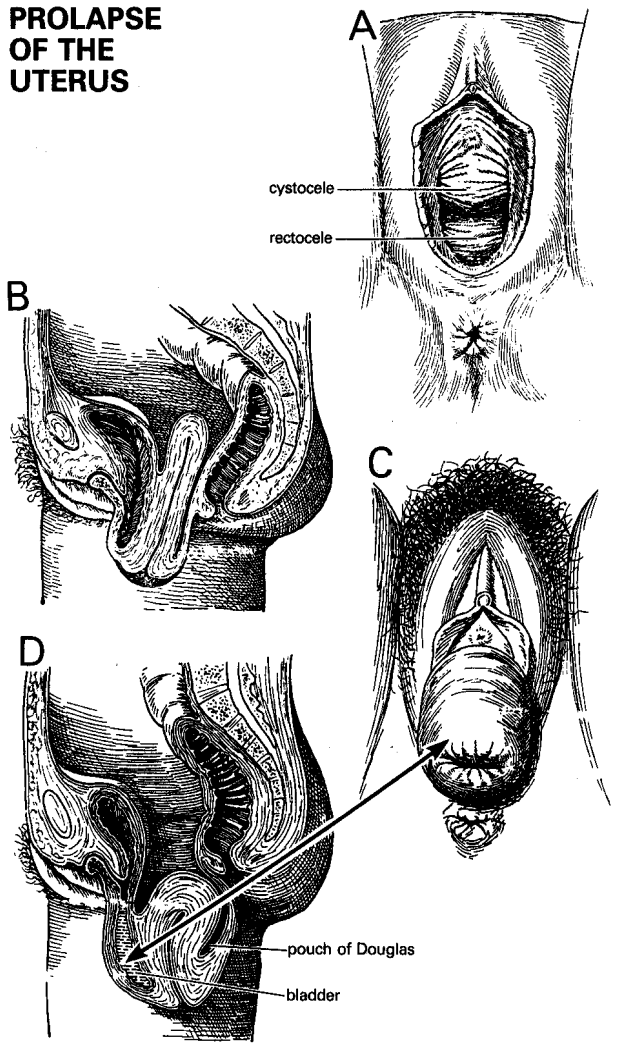


Fig. 23-15 PROLAPSE OF THE UTERUS.
A, a cystocoele and a rectocoele. B, 3rd degree prolapse. C,D, proccidentia; the fundus is outside the introitus. Ideally, all these patients need a vaginal hysterectomy and an anterior (23.13) **and posterior colporrhaphy** (23.14)**.**
After Young J. A Textbook of Gynaecology A&C Black, 5th ed 1939. permission requested.

If the bladder or urethra prolapses as a cystocoele, perform an anterior colporrhaphy (23.13).
If the rectum prolapses as a rectocoele, perform a posterior colporrhaphy (23.14).
If the uterus prolapses, arrange a Manchester repair (23.12) if it is to be left *in situ*, or a vaginal hysterectomy if it is to be removed. A simpler alternative is a ventrisuspension (23.12).

N.B. A vaginal hysterectomy, when the uterus is completely prolapsed out of the vagina, is much easier than an abdominal hysterectomy: the uterine arteries are far away from the ureters, so you can readily ligate them and amputate the vagina.

If there is ulceration of the prolapsed uterus, apply oestrogen cream (you can make this by crushing tablets in petroleum jelly).

MARY (80yrs) complained that her husband was accusing her of having given him an STI, because he was having pain in passing his urine. She wanted a letter she could take to the court saying that she was free of any STI. On examination the uterus was grossly prolapsed, ulcerated and stinking, but she had no evidence of any STI. A Manchester repair cured her completely. LESSON Patients' diagnoses are not always correct!

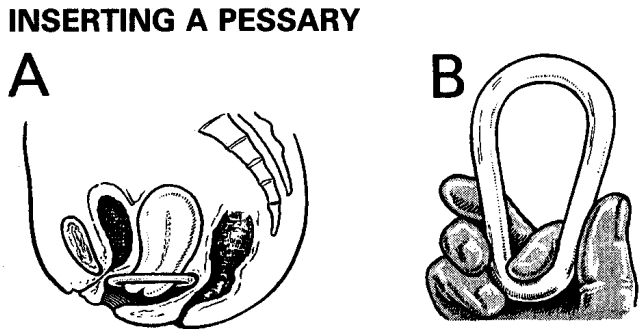


Fig. 23-16 A RING PESSARY is often very acceptable to an older patient with moderate prolapse. Choose its size *(40-120mm)* as you would a diaphragm, by measuring the depth of the vagina with your fingers. *After Garrey MM. Obstetrics Illustrated Churchill Livingstone 1974, permission requested.*

RING PESSARIES FOR PROLAPSE
Many old women prefer to avoid surgery, and can be treated with a pessary. Semi-rigid polythene ring pessaries are suitable. If they are comfortable, they can be left in indefinitely and may even not be noticed by the patient. Menstruation and sexual intercourse can take place as usual.

INDICATIONS
(1) Moderate prolapse especially in an older patient.

CONTRAINDICATIONS
(1) Ulceration of the prolapsed uterus.
(2) Weak perineal muscles; they will not hold a pessary and it will keep falling out. If too big a ring is required, the vaginal wall or cervix may prolapse through.

METHOD. Choose the size of a ring pessary as you would a diaphragm, by measuring the depth of the vagina with your fingers. It will usually be c.70mm. Lubricate it, compress it, and insert it like a diaphragm, with the posterior part behind the cervix, and the anterior part behind the symphysis.
It will resume its ring shape and take up a position in the coronal plane. If a 70mm pessary falls out, try a larger size in 5-10mm intervals. If it feels very tight and uncomfortable, so that the patient cannot pass urine, try a smaller size. Let her walk around, drink a lot, go to the toilet and return in c.1h to see if it is likely to be satisfactory. She should not feel it, and be able to pass urine. Make her also strain hard (when standing) to see if it comes out. Many women can learn to remove it and replace it themselves: it is not difficult and only needs some explanation/encouragement.
Teach the old ladies how to remove and insert it: it is probably better off for sexual intercourse and most women don't need it at night anyway.
This will help in reducing discharge and ulceration. Also sometimes the ring comes off (*e.g.* after defecation), so it is helpful if a woman can wash and replace it herself rather than visiting the hospital just for this.
Review her in 3 months; if all is well then, review annually and ask if the pessary is comfortable. Ideally, she needs a new silicone pessary each year. This may become permanently coated with solid material which you cannot wash off; then replace the pessary.
If the vagina is ulcerated at the annual check-up, leave the pessary out for 1-2 months and apply oestrogen cream nightly. When the ulcers have healed, insert a smaller pessary, and review in 3 months.

23.12 Ventrisuspension

In this operation the prolapsed uterus is sutured to the anterior abdominal wall. This relieves both the prolapse and the rectocoele or cystocoele, which will probably also be present. Ventrisuspension alone does not interfere with the bladder, the urethra, the rectum, or the vagina. It is not difficult and is a convenient operation if you are inexperienced; it does however sometimes fail.

Aim to make the anterior wall of the uterus, cervix, and bladder stick to the rectus muscles, and to make the peritoneum over the bladder, and the anterior wall of the cervix stick to the back of the pubis, so that there is no chance of bowel herniating between them.

INDICATIONS
(1) Prolapse involving a considerable descent of the uterus.
(2) Prolapse in old postmenopausal patients.

CONTRAINDICATIONS
A woman who still wants to become pregnant.

METHOD (GRADE 2.5)
Open the abdomen through a Pfannenstiel or midline incision, extending well down towards the symphysis pubis. The upper limit of the incision will depend on how far you can pull up the uterus, when you have examined it. Separate the uterus and adnexa from any adhesions, bring them into the wound, and examine them.

Identify the peritoneal reflexion of the bladder, so that you can avoid it. Separate the *rectus abdominis* muscles from the peritoneum, along their whole length on each side of the wound (23-17C).

Use a scalpel vigorously to excoriate the anterior surface of the body of the uterus, to within 1cm of its upper and lateral borders (23-17A). *Don't excoriate the cervix*. Instead, elevate and remove a strip of peritoneum about 2cm wide off the cervix, the bladder, and the anterior abdominal wall, to join up with the skin incision. Decide how high up the uterus should come behind the abdominal wall.

Pass three #2 parallel monofilament sutures through the outer surface of the rectus sheath on one side, through the rectus muscle, and then out of its bare posterior surface.

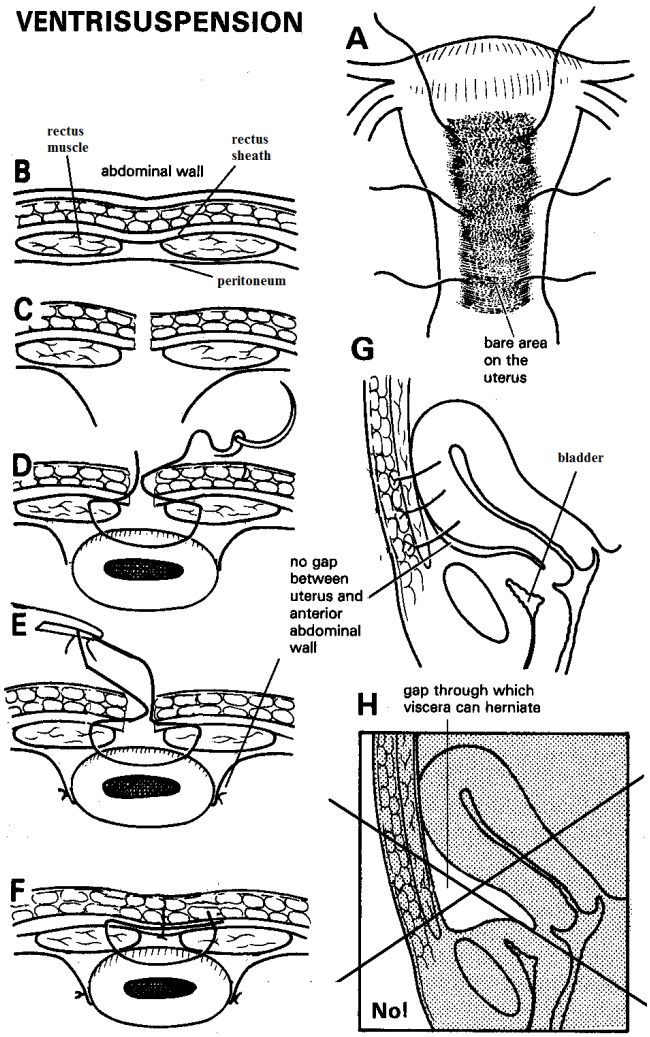


Fig. 23-17 VENTRISUSPENSION.
A, the bare area you aim to create on the uterus, with the sutures in place. B, the abdominal wall before starting the operation. C, open the abdominal wall and reflect the peritoneum. D,E, suture the rectus sheath and peritoneum to the uterus. E,F, tie the sutures. G, a side view of the completed operation, showing the uterus close up against the abdominal wall. H, the space needing closure to avoid internal bowel herniation.
Kindly contributed by Andrew Boddham-Whetham.

Then pass it deeply in and out of the bare area of the anterior wall of the uterus, across into the bare area of the other rectus muscle, and out through the anterior rectus sheath on the other side. *Leave no gap between the uterus and the anterior abdominal wall*.

As you do this, avoid the peritoneum, which will fold inwards (23-17D). Apply clamps to each suture, and leave them until later (23-17E).

Using 2/0 long-acting absorbable suture on a round-bodied needle, and starting at the apex of the bladder (but without penetrating it), suture the peritoneum continuously to itself along the line that you have previously excoriated. When you have closed this gap, suture the peritoneum to the edges of the excoriated area on the uterus.

In this way, you will have closed the peritoneal cavity, still leaving most of the uterus and all the adnexa intra-peritoneally, but with the excoriated area of the anterior uterine wall exposed in the open abdominal wall.

Now bring the anterior rectus sheaths lightly together with a continuous #1 monofilament suture, and tie the three large sutures which you previously passed through the anterior wall of the uterus (23-17F). The main strength of the suspension is the adhesions that are formed, *not these sutures*.

N.B. In a pre-menopausal woman, perform tubal ligation in addition.

If a ventrisuspension is not enough, add a simple diamond-shaped excision of the anterior or posterior vaginal wall to tighten up the vagina without doing a full Manchester repair. This consists of anterior and posterior colporrhaphy, amputation of the cervix, and plication of the transverse cervical ligaments, sutured to the front of the cervical stump.

23.13 Anterior colporrhaphy

The anterior vaginal wall, and with it the bladder, may bulge towards the introitus when a patient coughs or strains (cystocoele). The same thing can happen to the rectum (rectocoele) (23.14).

An anterior colporrhaphy mobilizes the bladder, returns it to its normal place, and fixes it there by exposing the peritoneum of the uterovesical pouch, and then suturing the fascia on either side, so as to make a supporting buttress from the urethra to the cervix.

EXAMINATION. Lay the patient on her side in the left lateral position. Insert a Sims' speculum posteriorly and anteriorly and ask her to cough and strain downwards. The cystocoele or rectocoele will then show its full size and the degree of uterine descent. Distinguish this from a urethral diverticulum (23.17).

If the cervix comes down to the vulva, she needs a Manchester repair (23.12), not just an anterior repair.

If she is postmenopausal, treat her with a course of oestradiol cream before operating.

If there is a rectocoele, usually accompanied by a deficient perineum, repair this at the same time.

ANTERIOR COLPORRHAPHY (GRADE 3.4)

INDICATIONS. Prolapse of the anterior vaginal wall which is troubling, especially if the patient has to push it back to micturate, provided there is little or no descent of the uterus. Preferably wait until childbearing is ended, because a prolapse may recur after pregnancy.

CONTRAINDICATIONS
(1) Ascites.
(2) A severe chronic cough.
N.B. Having a period is certainly not a contraindication.

PREPARATION. The tissues must be clean before you operate, so insist on a bath just before surgery. Clear the rectum with an enema.

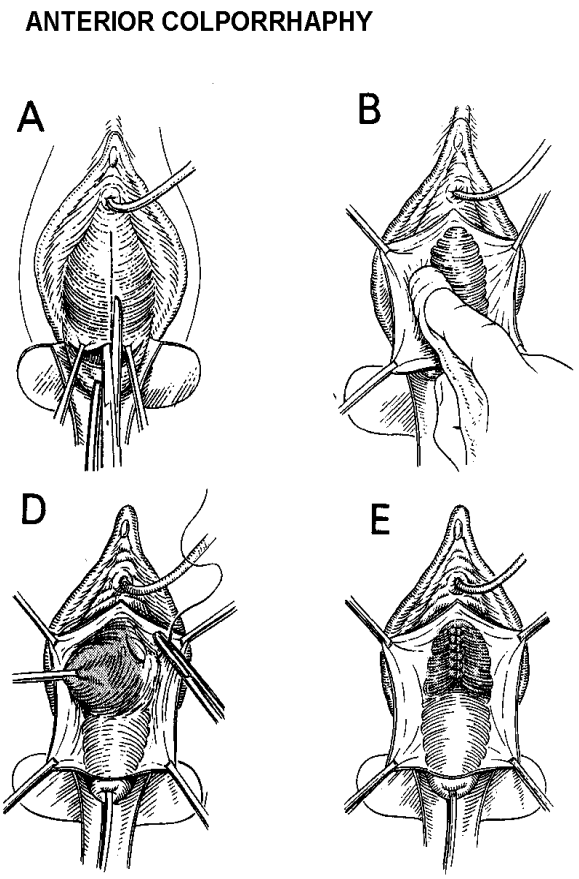


Fig. 23-18 ANTERIOR COLPORRHAPHY.
A, incise the anterior vaginal wall. B, mobilize the cystocoele. C, mobilize the cystocoele from the cervix. D, insert the tightening suture as far laterally as you can. E, obliteration of the cystocoele is complete. F, left anterior vaginal wall pulled to the right, and redundant wall cut off. *Don't cut off too much!*

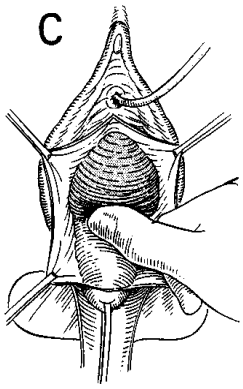
METHOD
Place the patient in the lithotomy position and clean the vulva and vagina. Suture the labia minora to the skin c.4cm from the vulva. Infiltrate the tissues, from the anterior urethral orifice to the anterior lip of the cervix, with 1/200,000 adrenaline; you will probably need 20-30ml.

Insert a catheter to identify the urethra. Put vulsellum forceps on the cervix and draw it down. Incise the vaginal wall covering the cervix about 1·5cm from the cervical os, and continue this laterally for 2cm on each side.

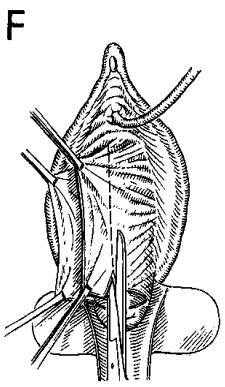
Undermine the edge away from the cervix, and continue to within 1cm of the urethral orifice, using the 'push and spread technique' with scissors (4-9).

CAUTION! Keep close to the vaginal wall to avoid injuring the bladder. Distending the tissues with adrenaline solution makes this easier. The key to success is to work in the right layer.

Cut the wall of the vagina in the midline (23-18A). Dissect the vaginal wall away from the underlying tissues with a combination of blunt and sharp dissection, until you expose the bulging bladder fully on both sides. Where possible, use a gauze-wrapped finger (23-18B). Take great care to separate the bladder from the vagina in the lateral part of the flap near the cervix. Dissection should be almost bloodless, until you reach the veins which lie well laterally.



Dissect the bladder away from the cervix (23-18C). If necessary, draw up the bladder with dissecting forceps and cut it from the cervix with Mayo's scissors. Separate the bladder from the cervix with a retractor and expose the peritoneum of the uterovesical pouch, but *don't open the peritoneal cavity*. Using gauze dissection, separate the lateral extensions of the bladder from the lateral border of the uterus.



Feel for a stout pillar of fascia on each side of the uterus. The secret of success is wide and courageous dissection to find the pelvic perivaginal (cardinal) fascia laterally. Use a series of interrupted simple or, better, mattress sutures of long-acting absorbable to pick up this fascia as far laterally as you can, starting superiorly (23-18D).

If this fascia is difficult to identify, insert the sutures into the fascial envelope of the bladder. When you reach the cervix, secure the fascia to it. Tying the sutures will suspend the bladder (23-18E).

Remove redundant vaginal wall (23-18F); this usually needs to have a diamond shape. If there is a large cystocoele, you will have to remove much vaginal wall, but if you remove too much, the vagina will be too narrow. Close it with interrupted sutures.

23.14 Posterior colporrhaphy

A posterior colporrhaphy reduces the gaping introitus, reconstitutes the perineal body, reinforces the pelvic diaphragm by approximating the *levator ani* muscles, corrects the rectocoele and eliminates the hernia of the pouch of Douglas. You can feel the *levator ani* muscles of a normal nullipara 5cm from the introitus. The key sutures in this operation bring the *levator ani* muscles together in this position.

If the cervix descends more than a little at the same time, a Manchester repair (23.12) or a vaginal hysterectomy is necessary.

POSTERIOR COLPORRHAPHY
(GRADE 3.4)
INDICATION
Prolapse of the posterior vaginal wall, with little or no descent of the cervix. Do this at the same time as an anterior repair, if indicated (23.13).

CONTRAINDICATIONS and PREPARATION are as for an anterior colporrhaphy (23.13).

METHOD. Infiltrate the subepithelial tissues with adrenaline solution. On each side place Allis forceps 2cm apart over the posterior termination of the labium minor, just inside the fourchette (where the labia minora meet posteriorly) at the level of the little skin tags remaining from the hymen, and retract them. If you place them lower than this, the repair produces a bridge of skin which may cause dyspareunia. Retract the forceps, and use scissors to remove a little ellipse of skin between them (23-19A).
Hold the posterior vaginal wall with forceps. Use blunt dissection, and the 'push and spread technique' with scissors (23-19B), to dissect to a point where the posterior vaginal wall bulges less. When you have established a plane of cleavage, you can use your index finger (23-19C).

CAUTION! Keep near the vaginal wall to avoid incising the rectum.
At this point you usually need to excise some posterior vaginal wall (23-19D,E). How much you remove will decide how tight you leave the vagina (23-19F: assumes that you have not removed any).
Use #1 long-acting absorbable sutures on a curved needle to pick up: (1) The *levator ani* muscles high in the wound on each side. (2) The fascial layer, which is rather thin, and tie it on each side. This will support the rectal wall (23-19F). Then pick up the *transversus perinei* muscles on each side to reconstitute the perineal body (23-19G).

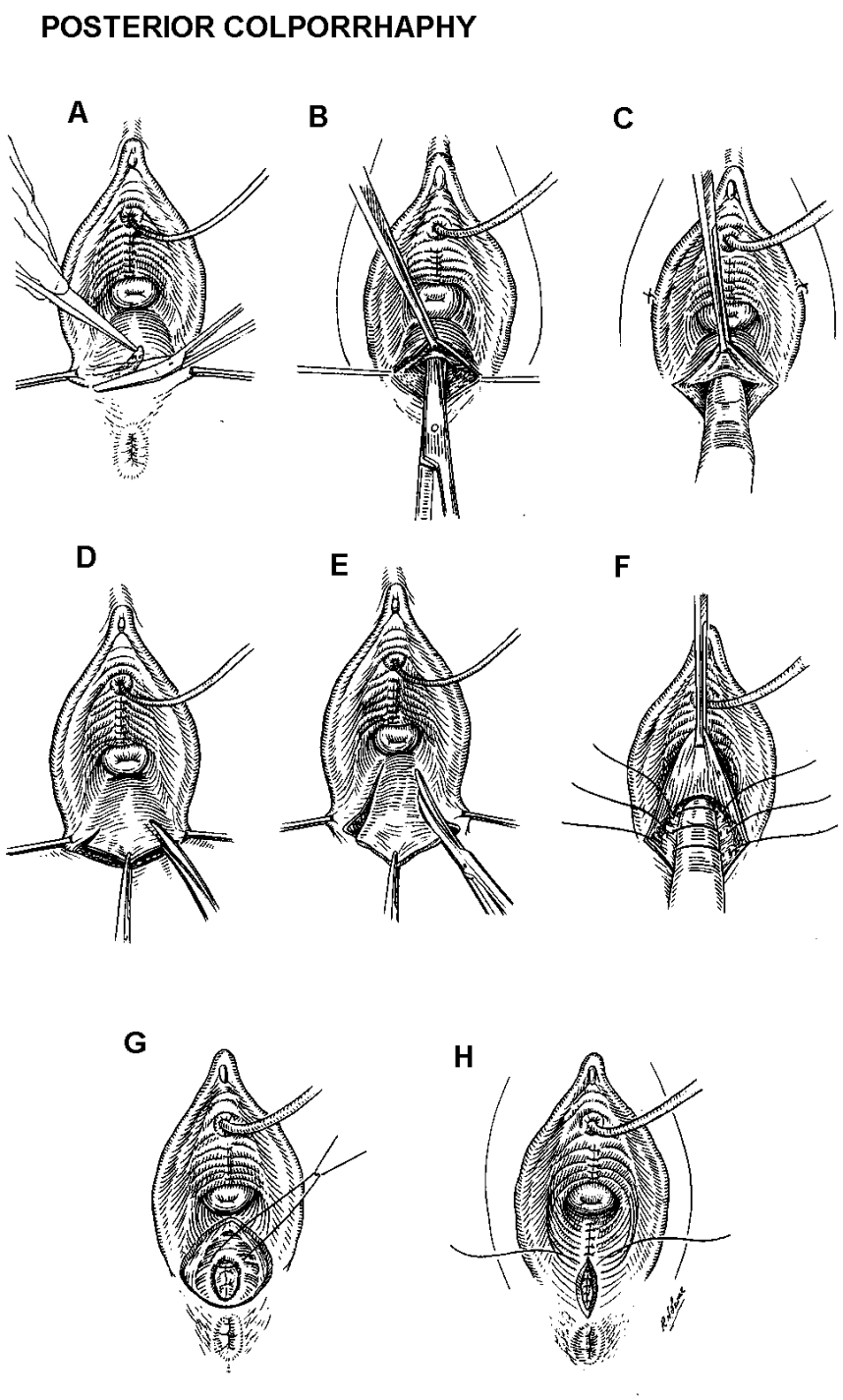


Fig. 23-19 POSTERIOR COLPORRHAPHY.
A, excise an ellipse of skin at the junction of the vagina and perineum. B, mobilize the posterior vaginal wall with a 'push and spread technique'. C, separate the rectocoele from the posterior vaginal wall. D, remove some skin from the posterior vaginal wall. E, carry the excision up to the apex of the freed vaginal skin. F, obliterate the rectocoele by tightening the fascial layer. G, suture the perineal muscles together. H, operation nearly complete.

Finally, close the posterior vaginal wall and perineum longitudinally in the sagittal plane (23-19H).

If you have done an anterior and a posterior repair together, the vagina should admit 2 fingers easily.

If you can only insert 1 finger, there will be some dyspareunia. Remove the outer 2 sutures, and reconstitute the margin (fourchette) transversely.

POST-OPERATIVELY, drain the bladder. Remove the catheter on the 5th day. About 6h later, ask the patient to pass urine and then re-catheterize her.

If the residual urine is <100ml on ultrasound (38.2i), let her pass urine normally.

If the residual urine is >100ml, reinsert the catheter for another 2 days and repeat the process.

DIFFICULTIES WITH COLPORRHAPHY

If there is much bleeding:
(1) If it is venous, inject adrenaline solution and wait 3mins. If necessary, pack the vagina. *Don't try to control venous bleeding with haemostats and ligatures*, because you won't find specific bleeding points.
(2) Undersew a bleeding artery with a suture.

If you open the bladder by mistake, repair it with a purse-string suture and reinforce it with a second layer of Lembert sutures (11-5). Leave a catheter in the bladder for 10 days.

If you open the rectum by mistake, this is not a disaster. If it is a large wound, close it transversely with long-acting absorbable sutures.

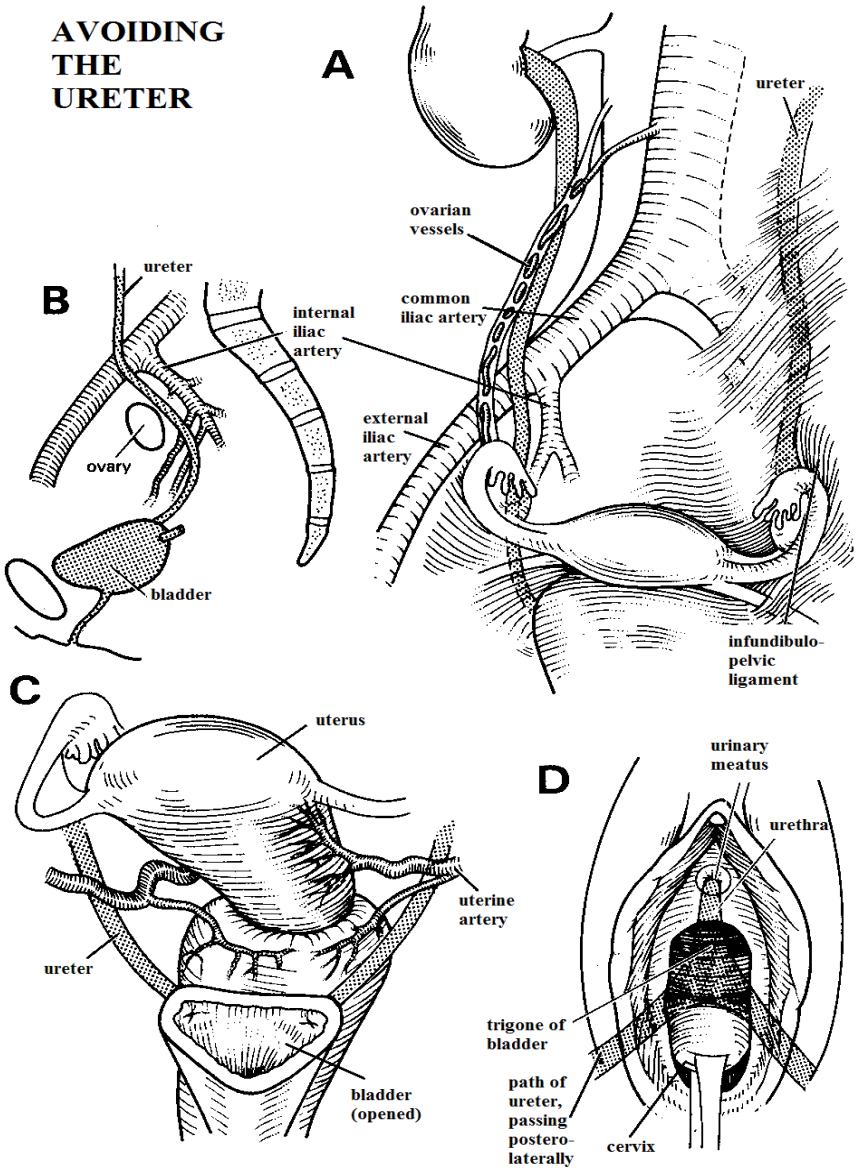


Fig. 23-20 AVOIDING THE URETER.
A, notice how the ovarian vessels pass in front of the ureter. B, the ureter passes over the pelvic brim (*i.e.* the pelvic inlet at the level of the promontory of the sacrum and the pubic symphysis, 23-21C) just after the common iliac artery has divided into its internal and external iliac branches. C, the ureter passes close round the vault of the vagina *under* the uterine artery *(remember this by 'water under the bridge')*. D, the relation of the urethra, the trigone of the bladder (a smooth surface delimited by the openings of both ureters and the urethra) and the ureters when you retract the cervix.
After Garrey MM. Gynaecology Illustrated Churchill Livingstone, 1977, p.308-9 with kind permission.

23.15 Hysterectomy

You may occasionally need to perform an *emergency hysterectomy* if a patient has:
(1) A ruptured uterus, and repair is impossible (not uncommon).
(2) Uncontrollable postpartum haemorrhage.

N.B. Hysterectomy for a ruptured uterus (21.17) differs in approach from the operation described below.

N.B. If possible, try to refer all elective cases. They may have disastrous complications, even in the hands of experts, and their patients even die occasionally. So *don't perform a hysterectomy, unless you are experienced*. Fibroids may cause disability, but they seldom threaten life.

(a) A total hysterectomy removes the entire uterus; the advantage being that you remove the cervix, which is a common site for carcinoma.

(b) A subtotal hysterectomy leaves a stump of cervix behind. It is contraindicated if there is any suspicion of carcinoma in either the cervix or the body of the uterus.

But it is an easier operation, because you can more easily avoid the ureters. It may also prevent a vaginal prolapse later, in populations prone to this complication.

If you are inexperienced, start by doing a subtotal operation, particularly if you are operating for fibroids. But even this can be difficult, if there are adhesions from chronic PID.

Don't attempt a radical hysterectomy which also removes the pelvic lymph nodes.

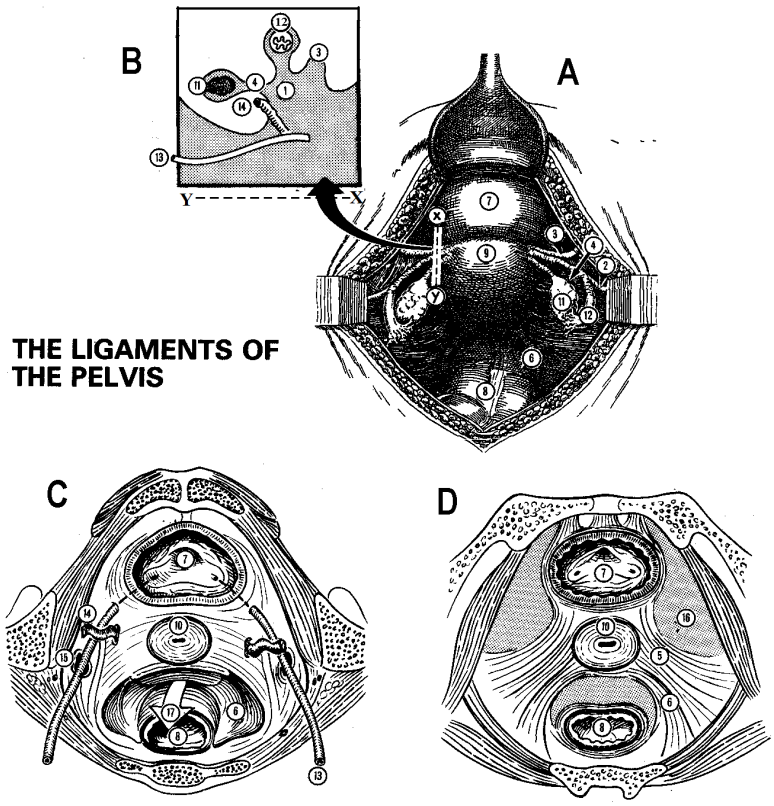


Fig. 23-21 THE LIGAMENTS OF THE PELVIS.
A, view through a laparotomy, looking down into the pelvis with the bladder at the top of the illustration. B, a sagittal section of part of the pelvis along line YX. C, a section through the pelvis, parallel with the pelvic brim (*i.e.* the pelvic inlet at the level of the sacral promontory and the symphysis). D, the main supporting ligaments of the pelvis viewed from above.
(1) the broad ligaments. (2) the infundibulo-pelvic ligaments. (3) the round ligaments. (4) the ovarian ligaments. (5) the cardinal (transverse cervical) ligaments. (6) the utero-sacral ligaments. (7) the bladder. (8) the rectum. (9) the fundus of the uterus. (10) the cervix. (11) the ovaries. (12) the Fallopian tubes. (13) the ureters. (14) the uterine arteries. (15) the veins of the pelvis. (16) fat filling the odd spaces in the pelvic connective tissue. (17) the arrow shows how an opening can be made from the posterior fornix into the pouch of Douglas.
A, after Young, J. A Textbook of Gynaecology, A&C Black, 5th ed 1939. C, after Last RJ. Anatomy, Regional and Applied.Churchill Livingstone 5th ed 1972. D, after Jeffcoate TNA. Principles of Gynaecology. Butterworths 3rd ed 1967.

ANATOMY. The most critical items of a patient's pelvic anatomy are the ureters (23-20). 'Ligaments' mean quite different things to gynaecologists and to orthopaedic surgeons. To gynaecologists, a 'ligament' is a fold of peritoneum, or a local thickening of the pelvic connective tissue. These are:
(1) The broad ligament, which is a fold of peritoneum running from the Fallopian tubes towards the floor of the pelvis. The ureter and the uterine artery lie in the base of the broad ligament; vessels run round its edge, and its middle is avascular (21-18).
(2) The infundibulo-pelvic ligaments are folds of tissue which run from the lateral ends of the tubes to the pelvic wall. Their importance is that the ovarian vessels run in them.
(3) The round ligaments are folds of tissue which run from the uterus close to its junction with the tubes, antero-laterally towards the brim of the pelvis. They are really anterior folds or leaves in the broad ligaments.
(4) The ovarian ligaments support the ovaries, and hang off the back of the broad ligaments.
(5) The cardinal (transverse cervical) ligaments are thickenings of the pelvic connective tissue which run laterally from the cervix to the sides of the pelvis.
(6) The utero-sacral ligaments run from the lower segment to the sacrum on each side of the rectum. They are, in effect, the posterior edges of the cardinal ligaments.

It is the only adequate surgical treatment for carcinoma of the cervix, but this really is a task for an expert with services of an expert anaesthetist and urologist available.

The great danger at hysterectomy is that you may damage the ureter by cutting, tying or clamping it. The ureter is at risk at several stages:
(1) when you tie the ovarian vessels. So, lift these clear of the ureters beforehand.
(2) when clamping and tying the exposed broad ligament, and where the ureter is displaced by a large uterine fibroid.
(3) when unexpected bleeding is controlled and clamps and sutures are placed blindly and too deep.
So before you do anything in this region which might injure the ureters, feel for them carefully. You can roll a ureter between your finger and thumb, and when you pinch it, it vermiculates (moves like a worm).

Bleeding can be severe, especially from the uterine vessels. Even when you have divided them, you are still in a bloody triangle at the sides of the vaginal vault. If you are not careful, you can also cause a vesico-vaginal fistula. This will be much less likely if:
(1) You develop a bladder flap.
(2) You carefully separate the bladder from the cervix.

Gentle continued traction is the secret of all pelvic surgery:
(1) It demonstrates the tissue planes.
(2) You are less likely to pick up structures that you don't want to cut.
(3) Vessels stand out more clearly.
(4) You are less likely to injure the bladder or the ureter.
(5) You can find the relation of the bladder to the cervix and vagina more easily.

The operation we describe is the classic extrafascial hysterectomy. After this operation, it is painful to bear down to defecate; so, make sure your patient is not constipated, and provide laxatives if they have such a tendency.

HYSTERECTOMY (GRADE 3.4)

INDICATIONS
(1) Severe anaemia or mechanical problems from large fibroids.
(2) Endometrial carcinoma (Stage I, IIA).
N.B. A total hysterectomy with adnexectomy is needed for cervical carcinoma (23.8).
(3) Severe dysfunctional uterine bleeding (23.3).
(4) Severe post-abortal or postpartum uterine sepsis.
(5) Chronic pelvic pain due to PID which fails to respond to medical treatment.
(6) Complete or nearly complete uterine prolapse.
N.B. In this last case, a vaginal hysterectomy is more appropriate (23.11).

CONTRAINDICATIONS

(1) An inexperienced operator.

(2) Active PID, but distinguish this from post-abortal or postpartum uterine sepsis.

(3) A uterus 'fixed' in the pelvis.

N.B. **Dense adhesions**, such as those due to PID, fibroids in the broad ligament, and obesity may pull the ureters out of place and make the operation very difficult and hazardous.

EQUIPMENT. A general set, a catheter, a uterine probe and sounds, a suitable self-retaining retractor, preferably Kirschner's, Gosset's, or Balfour's; also a Deaver's retractor and a tenaculum forceps. At least 4 and preferably 6 long curved uterine clamps, either Hunter's or Maingot's. 2 large packs with cloth tapes, T-tube for ureter repair.

PREPARATION

Make sure that consent is signed and the patient understands that she will have no more children and no periods. Give laxatives or an enema the evening before surgery because passing a stool immediately afterwards is very painful. Set up an IV infusion, and have blood cross-matched.

Start prophylactic metronidazole 1g PR together with gentamicin 240mg or chloramphenicol 1g IV.

Find yourself a competent assistant, who, if inexperienced, should go through this account with you first.

Place the patient in the lithotomy position, perform a vaginal examination (with non-sterile gloves) to reassess the size, position and mobility of the uterus. Then clean the vagina with aqueous iodine solution. Catheterize the bladder. Compress it suprapubically to make sure it is empty, and leave the catheter in for continuous drainage.

Then lay the patient supine on the table and remove the lithotomy poles. Tip the table slightly head-down to let the bowel fall away from the pelvic cavity. Provided the angle is not too steep, it will not make anaesthesia difficult. Ideally, adjust the break in the table so that the knees are slightly flexed. Stand on the left if you are right-handed.

INCISION. If you are inexperienced, make a midline incision from the symphysis to the umbilicus. If you are skilled, and the uterus is not >15cm high (equivalent to a 14-16wk pregnancy), a Pfannenstiel incision gives the best cosmetic result and avoids incisional hernias.

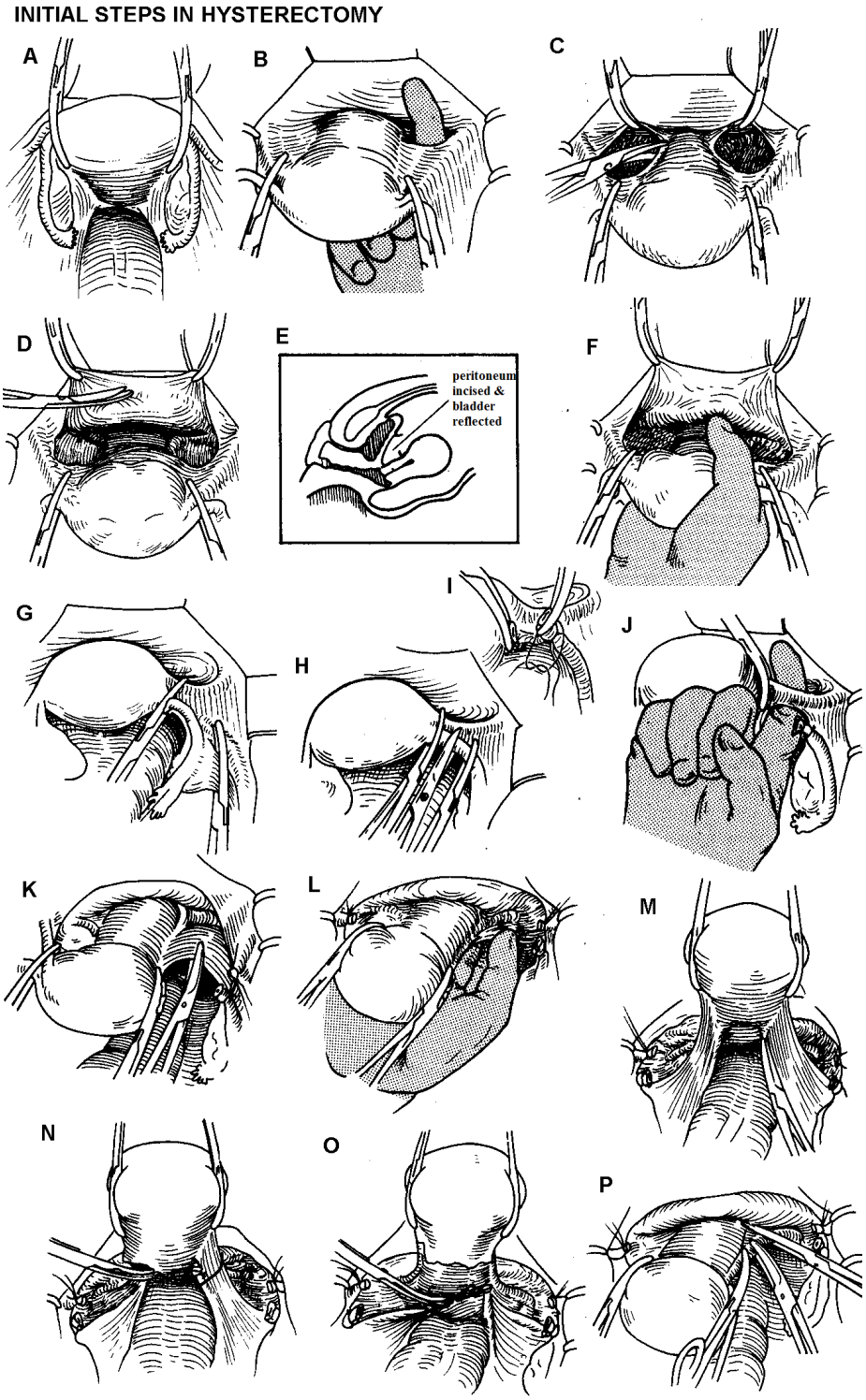


Fig. 23-22 INITIAL STEPS IN HYSTERECTOMY.
A, put clamps on either side of the fundus. B, put clamps on the tubes and round ligament and make a hole in it with your finger. C, reflect the bladder. D,E, incise the peritoneum in front of the cervix. F, feel for the tip of the cervix. G, clamp the ovarian pedicle laterally if you are removing the ovary or, H, clamp it medially if you are retaining the ovary. I, divide and tie the stumps. J,K, isolate and tie the round ligaments. L, find the uterine arteries and cut the posterior leaf of the broad ligament almost as far as the artery. M, lift up the uterus. N, divide and tie the utero-sacral ligaments. O, reflect the peritoneum off the back of the cervix. P, doubly clamp the uterine arteries.
After Parsons L, Ulfelder H. An Atlas of Pelvic Operations. WB Saunders 1968 p.21ff with kind permission.

N.B. The illustrations here assume you are standing on the patient's left, which most right-handed surgeons find easier.

CAUTION! Make sure your incision is long enough, and that you have divided the rectus sheath and muscles as far as the symphysis pubis (an extra 1cm at the bottom is worth 5cm at the top). If necessary, extend the incision generously above the umbilicus.

With a Pfannenstiel incision, arrest all the bleeding before you enter the abdomen; you might otherwise forget later and the result will be an enormous haematoma.
Open the peritoneum with your finger in the middle of the incision, firstly upwards, so as to avoid the bladder more easily.

N.B. An improvement on the Pfannenstiel is the transverse Joel-Cohen incision (11.2). The incision is not curved and c.3cm below the level of the umbilicus but *not below* the upper limit of the pubic hair. Cut only the skin and make an incision down to the rectus sheath. Make a small transverse incision in the middle portion of the sheath and open the *rectus* muscle (vertically) by stretching laterally with two fingers of each hand. Make a small hole in the peritoneum and stretch it vertically with both index fingers. Then make the opening in the abdominal wall as large as that in the skin by manual traction. Do this *after* the peritoneum is opened (to prevent disrupting vessels between posterior rectus sheath/peritoneum and *rectus* muscle). This approach is very fast, works also excellently with a Caesarean section and minimizes blood loss from the abdominal wall.
Exploration is the first step: inspect the pelvic cavity. *If you find an inflammatory lesion, don't proceed to explore the upper abdomen*, because you may spread the infection. Otherwise, put your left hand into the wound to feel the organs in the abdominal cavity quickly and thoroughly. Follow a set pattern: look particularly for metastases in the liver.
Clear the operative field. This is often the most difficult part of the operation. *Don't start removing any organs until you have cleared the site of operation*.
Carefully pack the bowel out of the way with large damp packs, attached to a cloth tape, to which a haemostat is fixed. Protect the wound edges with moist gauze, and insert a 3-blade self-retaining retractor. You can put the crossbar towards the head or towards the feet, and use the 3rd blade to retract the bladder. Make sure it does not compress the caecum, the sigmoid, the small bowel, or the iliac vessels. When necessary, use Deaver's retractor.
Clear any adherent bowel or omentum from the pelvis. Use blunt dissection to free any loose adhesions between the uterus and its surrounding structures: the sigmoid colon, the ovaries, or the walls of the pelvic cavity. The tubes and ovaries may be stuck down behind the broad ligaments; get your fingers under them and free them from below upwards. You may have to divide denser adhesions with scissors, or if you think they are likely to contain blood vessels, clamp, divide, and tie them. Divide any adhesions between the fundus of the bladder and the fundus of the uterus.
If you can deliver the uterus out of the abdomen, especially if it is very big, this will help greatly.
If you restore the proper anatomy first by removing adhesions, you are far less likely later to damage ureters, bladder or bowel.

Put clamps on either side of the fundus of the uterus, (23-22A) and over the tubes and round ligaments (23-22B). Use them to exert traction, and control arterial bleeding.
Alternatively, if these structures are friable, use a myomectomy screw or traction sutures to hold the fundus. Ask your assistant to pull on the clamps, so as to demonstrate the thin avascular part of the broad ligament more clearly. Push your finger through this thin part near the uterus, from behind forwards, to make a hole (23-22B). Do the same on the other side.
Reflect the bladder. Incise the peritoneum on the front of the cervix, near to its vesico-cervical reflexion (23-22C).
Dissect the bladder off the front of the cervix and upper vagina (23-22D,E), until you can feel the tip of the cervix (23-22F). Do this bluntly with your gloved finger or a gauze while your force is exerted in the direction of the uterus and cervix, *not the bladder*. If there was a previous Caesarean section, you often need sharp dissection: so cut even into the cervix superficially, rather than into the bladder. Feel the cervix from in front and behind. Separate the bladder from the underlying tissues somewhat laterally also.
Find the ureters. They enter the pelvis at the bifurcation of the iliac vessels. Trace them distally to beyond the tip of the cervix; recognize them by their feel: they are rather hard, they don't pulsate, and you can roll them between your finger and thumb (23-20).

CAUTION! Ureters are apt to be easy to find when they are in no danger, and almost impossible to find when you need to find them!

If you cannot find the ureters, these steps will protect them:
(1) Free the adnexa from adhesions before you remove them.
(2) Lift the infundibulo-pelvic ligament and find the ovarian vessels before you clamp them.
(3) Very carefully dissect the bladder away from the cervix, and the adjoining part of the broad ligament.
(4) Cut and mobilize downwards the posterior peritoneal leaf of the broad ligament from the posterior surface of the cervix and somewhat beyond, and a tiny bit laterally so that it is possible to apply a clamp from lateral just under the cervix at the last stage of the hysterectomy without having any peritoneum in the clamp.

Now deal with the ovaries. You must now decide if you want to retain them or not. If they have multiple large cysts, remove them, but try to retain at least one ovary if the patient is pre-menopausal or <5yrs postmenopausal. If there are any cysts, it is better to remove the ovaries in an older postmenopausal woman.

To remove an ovary, going lateral to it, but very near it, clamp its vessels, taking care not to clamp the ureter at the same time (23-22G).
You don't need a counter clamp if you have already placed clamps on either side of the fundus (see above): this makes it possible to ligate very near to the ovary. Otherwise place the other clamp medial to the ovary.

Divide the ovarian pedicle medial to the lateral (*not the counter*) clamp, and tie it with a double transfixion suture using #1 absorbable. Ease and squeeze the clamp while tightening the suture to make sure the tissues can be compressed properly by the suture.

If you remove the ovary, be sure also to remove the Fallopian tube with it.

If you want to retain an ovary, apply a clamp across the Fallopian tube and its pedicle, 1cm lateral to the first clamp that you applied to these structures near the uterus (23-22H). Divide the tissues between these clamps (23-22I). Ease and squeeze and then remove the lateral clamp and tie its pedicle as above. Do the same thing on the other side, removing or retaining the ovary, as you decide.

Define, tie, and divide the lateral end of the round ligament. Do this by pushing your finger under it and tying it (23-22J,K).

Find the uterine artery (23-22L). Cut the posterior leaf of the broad ligament with the loose areolar tissue inside it, almost as far as the artery (23-22K,L). If your assistant stretches the broad ligament well by pulling on the clamps, you may see the artery through the tissues you are going to cut. Repeat this on the other side.

Ask your assistant to lift up the uterus again (23-22M). This will demonstrate the utero-sacral ligaments. Clamp, divide, and tie them (23-22N). Dissect the peritoneum off the back of the cervix (23-22O), if it is not too adherent, otherwise leave it. The uterus will now be much more mobile.

Feel for the uterine arteries again. There is no need to dissect them out. Next feel for the ureters on each side of the distal cervix. Again, identify them by their feel: firm cords which you can roll between your finger and thumb. Doubly clamp the pedicle containing the uterine artery (23-22P), well away from the ureter, with the tip of the clamp biting the side of the cervix, and leaving little or no tissue on the uterine side.

Use scissors, or a knife. Cut as near, or even just in the uterus, as possible. If you don't use 2 clamps on each side, apply bilateral single clamps before you start cutting because the uterus will start bleeding on one side when the uterine artery on the other side is not clamped.

Using, in this way, 2 clamps instead of 4 makes it possible to divide and clamp nearer to the uterus/cervix decreasing the risk to the ureters; there is also less clutter in the operative field.

Put the convex side of the clamp near the uterus so that it is easier to get the clamp very near it.

If the bladder is well down and the posterior leaf of the broad ligament out of the way and the clamp (and suture) very near to the uterus, then the ureters should be out of harm's way. The clamp is then not on the slippery peritoneum. Sometimes it needs 2-3 steps to clamp the tissues on each side of the uterus. Place the suture 1mm medial and 1mm distal from the point of the clamp while laterally, tie it c.1cm under the clamp. This will prevent oozing later.

Complete the task of pushing the bladder down the cervix, if you have not already done so (23-23A). Blunt dissection is usually enough.

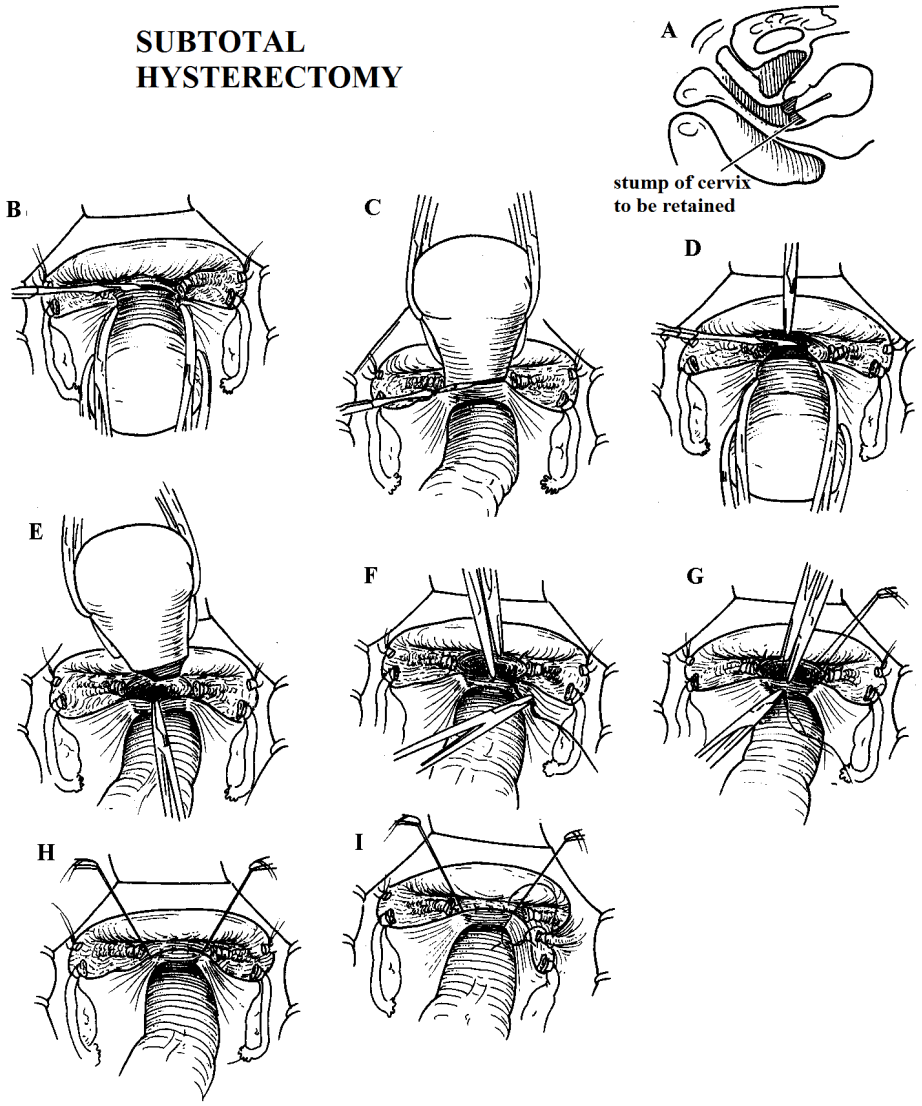


Fig. 23-23 SUBTOTAL HYSTERECTOMY.
A, the part of the uterus to be retained. B,C, incise the anterior and posterior walls of the cervix. D,E, grasp the cervix stump and make a cone-shaped cut. F,G,H, close the cervix and control bleeding by placing sutures through the posterior peritoneal reflection deep into both lips of the cone. I, suture the round ligaments to the cervix.
After Parsons L, Ulfelder H, An Atlas of Pelvic Operations, WB Saunders 1968 p.45,47, with kind permission.

Now decide if you want to proceed with a subtotal or total hysterectomy. If you are inexperienced, do the former.

SUBTOTAL HYSTERECTOMY

If you have placed 2 clamps for the uterine vessels with their points 1·5-3cm above the distal end of the cervix on the lateral uterine wall, release each slightly in turn, squeeze again and tie them individually after the ligating needle has gone *through* the uterus just under the point of the clamp. In this way, you will be sure to have tied all the vessels lateral to the uterine part you are going to remove.

When you are sure you have reflected the bladder adequately (23-23A), pull on the clamps attached to the uterus and incise the anterior wall of the cervix, above the reflexion of the bladder and the stump of the uterine vessel (23-23B). Then draw the uterus sharply forwards towards the symphysis, and incise the posterior wall of the cervix (23-23C). Place a clamp on its anterior incised edge (23-23D).

Place a clamp on the posterior cut edge of the cervix (23-23E), so that you can maintain traction. Bring the two cut edges of the cervix together to control bleeding. Use a cutting Mayo half-circle needle, and place the first stitch in the edge of the cervix, close to the point where you tied the uterine arteries. Control bleeding by placing the sutures through the posterior peritoneal reflection, deep into the muscle of both lips (23-23F,G,H). Suture the round ligaments to the cervix if you can do this easily (23-23I).

TOTAL HYSTERECTOMY

Cut through the cardinal ligaments flush with the cervix, until you feel their ends on each side (23-24A). You should now be able to feel the cervix abdominally through the wall of the vagina from in front and behind with your finger and thumb (23-24B).

Often it is possible with a total hysterectomy to have the cardinal ligament including the utero-sacral ligament in the same clamp and hence in the same pedicle as the uterine artery. So, on one side, you should have the following pedicles:
(1) the round ligament;
(2) the doubly ligated infundibulo-pelvic ligament;
(3) the doubly ligated uterine artery including the cardinal ligament;
(4) tissue *just* lateral to the distal cervix plus the top ⅓ of the vagina.

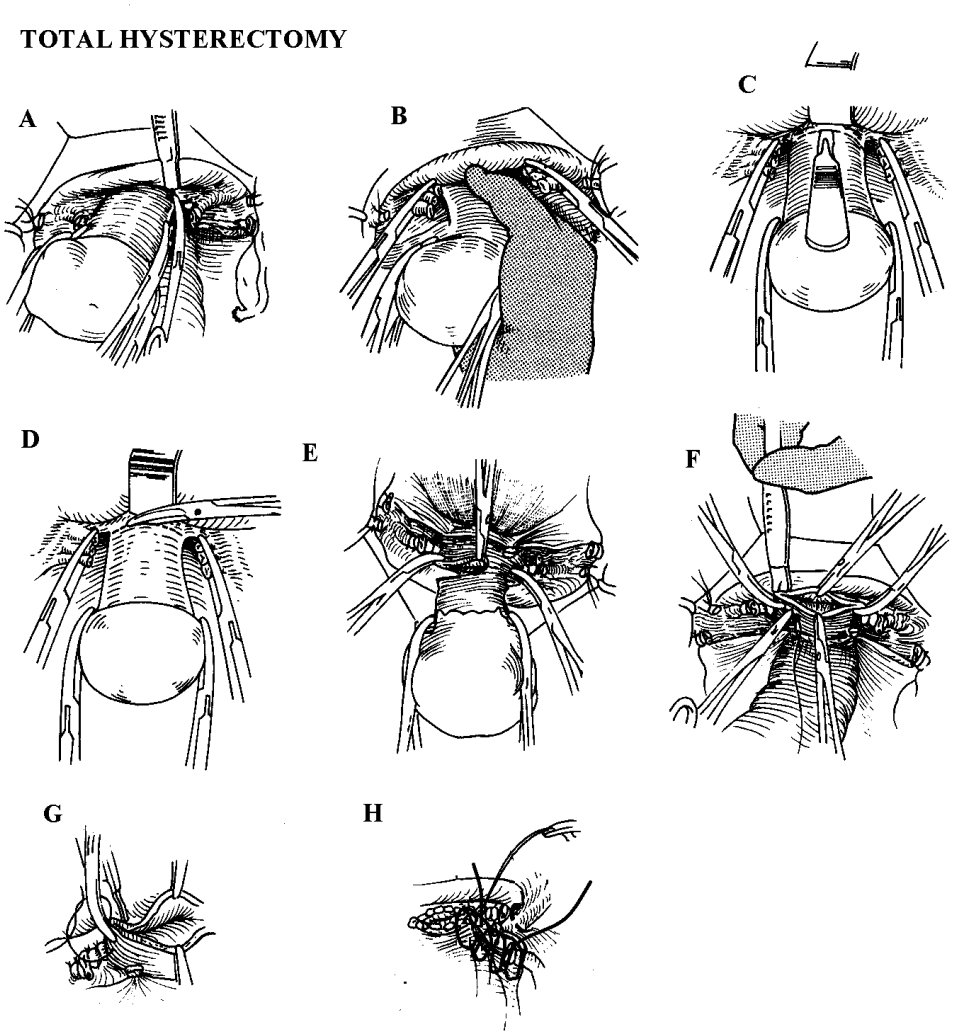


Fig. 23-24 TOTAL HYSTERECTOMY.
A, reflect the bladder. B, feel for the cervix. C,D, incise the fornices of the vagina. E, cut the uterus free. F,G, close the vagina. H, suture the round ligaments to the plicated vaginal vault.
After Parsons L, Ulfelder H, An Atlas of Pelvic Operations. WB Saunders 1968 p.33,34, with kind permission.

Before you open the vagina, insert clamps on the vaginal angles immediately below the cervix. Ask your assistant to pull up the uterus. Make absolutely sure no bowel or bladder is in these 2 clamps placed below the cervix. Use a broad-bladed or right-angle retractor to pull back the bladder carefully.

Cut the vagina above the clamps as near as possible to the cervix. If you can see easily, complete the cut with curved scissors (23-24D).

CAUTION! To avoid damage to the ureters, always make sure you find them. Clamp the uterine pedicles away from them, and cut the vaginal wall very close to the cervix.

Use #1 long-acting absorbable transfixion sutures to tie the vaginal angle and top pedicles bilaterally, *making sure that you don't include the ureters*, keeping the ends long in order to tie the uterine artery on each side, once again for extra security. If there is some oozing from the open part of the vagina, control it with mattress or figure-of-8 sutures (4-9H).

If you can easily do so, suture the round ligaments to the ends of the vaginal vault (23-24H). This will help to prevent prolapse, but is not essential.

Remove the swab holding the bowel, and close the abdomen in the usual way. There is no need for a drain if the operation was not for an infection in the first place and the bowel or urinary tract was not damaged. Otherwise leave the vagina open to help drainage. In serious infection, leave a large tube draining into the vagina, fixing it from inside the abdomen with a thin absorbable suture.

POST-OPERATIVELY, check the vaginal pads, pulse, BP and urine production to make sure there is no bleeding.

THE SPECIMEN. Open the uterus to see if there is a carcinoma of its body. Do this after the operation, to avoid contaminating the wound with tumour cells if any are present.

DIFFICULTIES WITH HYSTERECTOMY

If adhesions from old PID or endometriosis prevent you starting, begin by dividing the round ligaments. Then put your hand behind the uterus and push a finger through the broad ligament under the tube and out through the divided round ligament. You now have the tube and ovarian vessels and can clamp and divide them safely.

If the uterus is so large that it obstructs your access to the pelvis, perform a subtotal operation first, and cut across the cervix quite high up. When you have removed the body of the uterus, you will have plenty of room to complete the operation.

If you cannot find the ureter, but must proceed with the operation, keep extremely close to the uterus. You will nearly always be safe there. Perform a subtotal hysterectomy only.

If a fibroid extends into the broad ligament, this may be: (1) growing out from the uterus and displace the uterine vessels and ureter downwards, laterally or upwards; (2) separate from the uterus and arise *de novo* from the connective tissue in the broad ligament. Both are difficult to dissect out.

In the 1st case, divide both the ovarian vessels and dissect out the upper part of the fibroid. Then proceed with the operation as usual on the normal side of the uterus only. Clamp and tie the uterine artery and utero-sacral ligament. Cut across the vagina. As you reach the affected side of the vagina, you will see the uterine artery on that side. Clamp and divide it (it may be large) and shell out the remainder of the fibroid.

In the 2nd case, open the broad ligament by dividing the round ligament as you would for a broad ligament cyst. The ureter will be attached to the posterior edge of the broad ligament above; lower down it will be displaced downwards and medially by the fibroid.

If there is a fibroid in the cervix, removing it can be very difficult. Limit yourself to a subtotal hysterectomy.

If there is a fibroid low in the posterior uterine wall, make a transverse incision over it and shell it (partly) out with your finger: this will help mobilize the uterus. If the uterus is thus mobilized, a hysterectomy will be routine. You may then be able to ligate the vessels leading to the fibroid and can then close the resulting cavity, so that the hysterectomy is no longer necessary.

If you divide the ureter and recognize that you have done so, repair the ureter with continuous 3/0 or 4/0 absorbable over a fine feeding tube which you have inserted into the proximal and distal ends of the divided ureter. If you can perform a cystoscopy, you will be able to withdraw the feeding tube after 10 days. If not, make a small cystostomy and find the distal end of the tube: *don't pull on it!* Attach its distal end through the eye of a Foley catheter that you have placed in the bladder. You can then remove the feeding tube simply by removing the catheter.

Otherwise, fix the feeding tube in place in the proximal ureter, and lead the other end of the tube out of the abdomen through a separate stab incision, and allow urine to collect in a sealed bag. This will preserve kidney function till you can refer the patient for ureteric re-implantation later. Place a tube drain into the abdomen.

If you open the bladder, repair it in at least 2 layers with long-acting absorbable. Leave a catheter in for 10 days. The tear is likely to heal uneventfully.

If you have injured the colon, repair the tear in 2 layers. Fashion a defunctioning colostomy if there is severe soiling, or if there is severe scarring, and you are uncertain of the reliability of your closure.

If there is bleeding at the end of the operation, try packing the pelvis with warm packs and tie off any arterial bleeding vessels. If this fails, *don't close the vaginal vault*. Instead, insert a purse-string suture in the vaginal vault around a tube drain and pull it tight. This will leave a central hole in the vagina through which any fluid can escape.

If there is post-operative retention of urine, it is likely to be due to *detrusor* failure, and to be difficult to treat. First of all, make sure urine is being produced. Try 4wks of catheter drainage and urethral dilatation. If this fails, teach intermittent self-catheterization, which is effective and safe. Use a clean but not sterile simple plastic catheter, which she can use for at least a week. *A retentive bladder is much more comfortable than a leaky one, and easier to manage.*

23.16 Vulval carcinoma

If a patient has an ulcerating lesion of her vulva, this may possibly be a carcinoma which may need a wide and mutilating excision of the primary, with a margin of normal tissue of ≥2cm all round, and 1cm deep to the lesion. If the groin nodes are involved, she needs a bilateral groin dissection. This is a major intervention. There is often an offensive discharge, and dyspareunia, as well as dysuria.

Differential diagnosis includes:
(1) vulval warts or condylomata (23.17),
(2) syphilis (2^0 or 3^0: 23.17),
(3) chancroid (23.17),
(4) tuberculosis (23.17),
(5) donovanosis (granuloma inguinale: 23.17)
(6) lymphogranuloma venereum (23.17),
(7) *Schistosoma haematobium* (23.17),
(8) extensive anorectal cancer, particularly in HIV+ve patients (26.7).

CAUTION! Before contemplating a radical operation on the vulva, be sure to take a biopsy: it is tragic to perform a mutilating operation for an innocent lesion. *Inguinal lymphadenopathy does not necessarily mean cancer!*

23.17 Other gynaecological problems

(a) Congenital abnormalities of the genital tract

If a girl 12-16yrs has low abdominal pain and an abdominal mass, examine the vagina and vulva. If you find a bulging membrane (the hymen), the first menstrual discharges have distended the vagina (haematocolpos) and perhaps the uterus (haematometra) (23-25). The distended vagina may cause retention of urine by compressing the urethra.

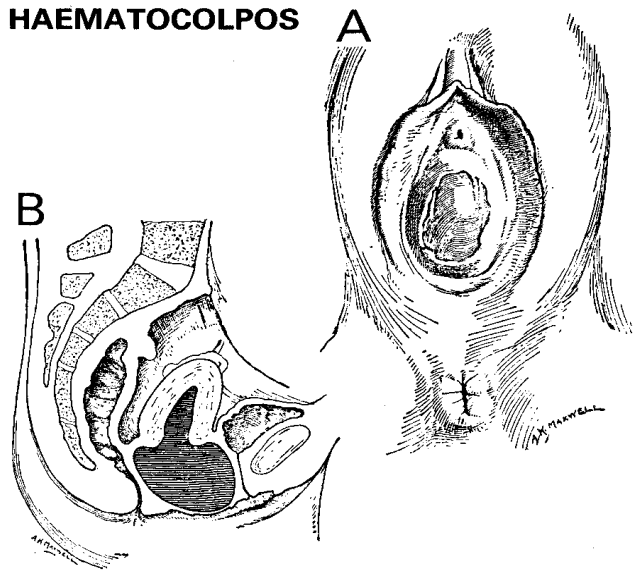


Fig. 23-25 HAEMATOCOLPOS. A, the bulging membrane retaining a girl's first menstrual discharges. B, a cross-section shows that there is also some degree of haematometra.
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Make the diagnosis by inspecting the introitus and when in doubt, by a finger in the rectum. If the membrane feels thin, incise it with a cross-shaped incision. *Don't do anything more than make a cruciate incision in a thin membrane. Don't insert a drain*; you risk introducing infection.

If the gap between the upper and lower vagina is more than a membrane, the operation to establish patency is not easy. Re-stenosis is common. The problem is not urgent: you would be best to refer to a specialist.

If a girl 6-24 months after puberty has a lower abdominal mass, one of the possibilities is a haematometra in the horn of a uterus didelphus (double uterus with double cervix) with one cervix stenosed. You can usually manage this by repeatedly dilating the stenosed cervix. Rarely, an isolated horn is not connected at all to the vagina; then a laparotomy is needed. *Beware: abnormalities of the ureters are likely as well.*

If there is a swelling in the anterior vaginal wall behind the urethra, especially before the reproductive years, consider the possibility of a URETHRAL DIVERTICULUM, and *don't confuse it with a cystocoele or a urethrocoele*. If you can squeeze its contents into the urethra, the diagnosis is confirmed. If you are not sure, perform a urethrogram (38.1). Consider excising the diverticulum, which is usually not difficult. Operate with a urethral catheter in place. Repair the small defect in the urethra which was the neck of the diverticulum.

(b) Other abnormalities

If there is a vaginal stricture from chemical irritants (including crushed beetles) or trauma (including FGM, 57.6), or existing congenitally, consider inserting a skin graft on a large mould, as soon as the vaginal cavity is clean. A dentist may have suitable material for the mould. Make it otherwise round a syringe barrel with the distal end cut off and made smooth, so as to make a passage for the menstrual fluid to escape. Hold the mould in place with sutures through the flanges (wings) of the syringe. Remove these 21 days later, and immediately re-graft any raw areas. You can use Hegar's dilators of different sizes to dilate a narrow vagina.

If there are condylomata acuminata, normally these are small and look like warts and are caused by a virus. When women are pregnant, they tend to grow and they can become huge if she is also HIV+ve (5.6). *Don't operate on them in pregnancy*: there will be much bleeding, and topical cytotoxics (like podophyline) are contraindicated in pregnancy. You will reduce transmission of HIV by performing a Caesarean section in such cases. Check if a tubal ligation is also desired.

Outside pregnancy, ARV therapy is indicated if there are huge condylomata. Repeated diathermy is successful and it is surprising how the large 3rd degree burns created by the treatment re-epithelialize within 1wk. Wear a mask and make sure the operating theatre is well aerated, to prevent aerosol spread of the condylomata.

If there is a spontaneous recto-vaginal fistula, this may develop in baby girls without a congenital or traumatic reason, related to HIV infection (5.6). Differentiate this from a congenital lesion where the anus is usually closed (33.6). *Don't be tempted to operate*. The baby does not know there is a problem! The best treatment is the use of a seton (as well as ARV therapy).

If there are papular nodules in the lower genital tract, consider SCHISTOSOMA. These take various forms:
(1) Frond-like (fern-like) lesions with a narrow base or plaques developing on the vulva, usually from 6-15yrs. These are often single, cause no problems, seldom bleed, and can be removed easily.
(2) Multiple granulomata of the vagina and cervix in the reproductive years and after them. These also seldom bleed, but they may be so extensive that they distort the bladder/urethral angle and cause incontinence.
(3) Cervical ulceration or papillomas, closely resembling carcinoma.
(4) Granulomata might present as infertility by blocking the tubes.
Look for ova in the urine, stool, vaginal discharge, and tissue scrapings or biopsies.
CAUTION!
(1) If schistosomiasis is endemic, *don't think all suspicious vulval or cervical lesions are carcinoma*.
(2) Carcinoma may co-exist with some other pathology. *Don't excise large vulval lesions without a biopsy first*.
(3) There is an increase in bladder carcinoma in these areas. These might present as supposed vaginal bleeding.

If there is a profuse vaginal discharge or post-coital bleeding, one cause is CERVICAL ECTOPY. The normal columnar endothelium of the endocervix bulges out onto the ectocervix, visible when you do a speculum examination. Cervical ectopy usually causes no symptoms. Exclude *Chlamydia* cervicitis, cervical carcinoma, severe trichomonal infection or a foreign body. Use diathermy to make 6-8 radial burns from the external os to the junction of the glandular ectopy (the erosion) with the normal squamous epithelium. Or, using a stick of silver nitrate, just touch all the glandular epithelium. Warn that the discharge will get worse for 1wk before it improves. No anaesthesia is necessary. Cryotherapy also needs no anaesthesia and is very effective.

If there is chronic or recurrent vulval ulceration, the differential diagnosis includes:

(1) **Small and usually ulcerated granulomata** arising in a perineum that is permanently wet from a vesico-vaginal fistula (salt baths will improve this temporarily, but a barrier petroleum jelly/silicone cream will be best).

(2) **Cellulitis, furunculosis, folliculitis, candidiasis:** Test the blood or urine for glucose. High humidity related to obesity or nylon underwear might also be involved.

(3) **Secondary syphilis:** Painless, greyish, moist, flat-topped broad-based raised swellings (condylomata accuminata). You may also see these around the anus in the patient's baby.

(4) **Tuberculosis:** Ulcers, sinuses and gross thickening are the most common types of vulval TB, which is usually transmitted sexually. Histology will help if there is a suspicion, especially if the woman is HIV+ve. Chronic skin TB is notorious for developing into cancer.

(5) **Donovanosis** (granuloma inguinale): Beefy, red, angry, destructive, irregular, sometimes tender lesions develop with a raised edge. It is caused by *Klebsiella granulomatis.* Initially no swollen groin nodes are present, but these may appear after secondary infection. The inguinal nodes can also become involved by extension of the original infection. Donovanosis can cause a pseudoepitheliomatous hyperplasia, which may be mistaken histologically for carcinoma.

(6) **Lymphogranuloma venereum** (LGV, 26.11): The initial vulval lesion, caused by *chlamydia*, is painless and small and may be missed. Later there are enlarged, matted, firm, painful nodes (more often in men than women) which can suppurate and cause several sinuses. A 'groove' caused by the separation of the inguinal and femoral glands (the bubo) by the inguinal ligament is typical. A so-called genital syndrome can develop, an oedematous swelling (elephantiasis) of the genitalia combined with destructive painless hypertrophic lesions involving often urethra and/or rectum. This might result in fistula (watering-can perineum) and strictures.

Distinguishing between (6) and (7) can be difficult, and both may be present. Fortunately, they both respond to tetracycline or chloramphenicol over 3wks.

(7) **Herpes simplex** (type 1 or 2): The first manifestation can be very painful and can even cause urine retention. Vesicles soon rupture and become skin lesions which can become secondary infected. Repeat infections appear in a subgroup of patients. These are far less painful and start with itching, pain or a 'funny' feeling. Small, itchy, painful vesicles lasting for 2-3wks appear. These lesions are typically grouped. If you treat with acyclovir cream at the earliest suspicion that a new attack is coming, an attack can be shortened.

(8) **HIV** infections might facilitate all sorts of chronic ulcerations (5.6) of the vulva (and penis). Micro-organisms which normally are not a problem or only a minor nuisance, now grab the opportunity like the different herpes infections, molluscum contagiosum, candidiasis, vulvar warts, chancroid, and LGV to cause huge and chronic sores, erosions, necrosis and warts. It is best to restore immune competence with ARVs before intervening surgically.

(9) **Amoebiasis** (rare): Painless ulcers which may mimic carcinoma and usually respond dramatically to metronidazole (14.5).

(10) **Chancroid:** Single or multiple, painful/tender, soft, bleeding shallow ulcers with minimal to no surrounding

induration arising within 1wk of a sexual contact. The inguinal nodes are enlarged, tender and may suppurate.

(11) **Schistosomiasis** (see above).

(12) **Carcinoma** (23.16).

If an old woman complains of sudden severe vaginal bleeding, suspect a vaginal tear usually in the posterior fornix as the result of sexual intercourse, especially after a period of abstinence. You will see the tear on speculum examination:
(1) If bleeding has stopped, do nothing.
(2) If bleeding continues, insert 1 or 2 mattress sutures.
(3) If the tear has penetrated the posterior fornix, replace the bowel and repair the laceration. Use antibiotic prophylaxis.

If there is a small round red lump on the posterior margin of the urethral orifice, it is probably a URETHRAL CARUNCLE. Usually, it needs no treatment; if it is pedunculated and bleeding, excise it (23.6). Distinguish this from a choriocarcinoma, if it appears after pregnancy.

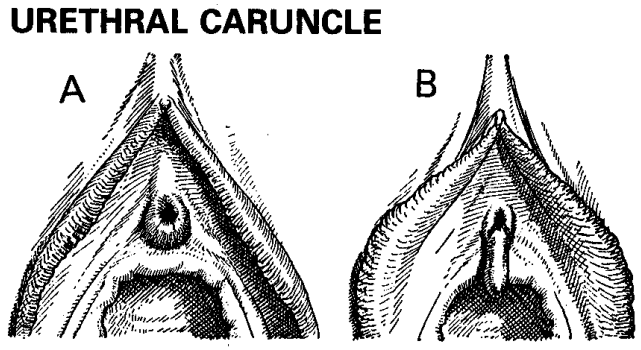

Fig. 23-26 URETHRAL CARUNCLE.
This usually needs no treatment (A); if it is pedunculated (B), excise the excess.
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If there is lymphoedema of the vulva, think of:
(1) tuberculous lymphadenitis in the groin (17.4),
(2) lymphogranuloma venereum (LGV: 26.11),
(3) donovanosis (see above),
(4) filariasis (27.34, 34.14),
(5) 2º or 3º syphilis.

N.B. Vulval oedema, especially in donovanosis and filariasis, can sometimes be so gross as to mimic elephantiasis of the scrotum. *Don't excise associated lymph nodes*; this will only make the condition worse.

Suggesting LGV (26.11): a fistulated inguinal adenitis with a sour smell, a concealed indolent sore of the vaginal vault, vesico-vaginal or recto-vaginal sinuses and rectal strictures; absence of pain. Histology is often non-specific.

Suggesting donovanosis or hydradenitis: extensive destruction with oedema with scarring.

Suggesting filariasis or chronic cellulitis: oedema without scarring. Treat any local sepsis. A large swelling of the vulva may need excising. Use prophylactic antibiotics. Excise a wide area of skin, so that the incision goes through healthy skin; this will assist healing, and make recurrence less likely. Catheterize the patient for 1wk. Apply a well-padded dressing of petroleum jelly gauze.

If an IUD is lost or neglected (but has not been removed), you should search for it because there is significant risk of complications, including:
(1) Uterine bleeding (23.3)
(2) Uterine sepsis (23.1)
(3) Ectopic gestation (20.6)
(4) Recurrent urinary infection and bladder stone formation.

Ideally, try to look for the IUD threads in the vagina or within the uterus. You may have to grab the IUD itself with appropriate forceps at colposcopy. Very rarely, the IUD can migrate into the bladder (or leave a vesico-uterine fistula, which will result in urinary incontinence) and produce a stone.

24 The breast

24.1 Introduction

There are two kinds of inflammatory lesion in the breast:
(1) Acute abscesses, which mostly occur during lactation, but may be a sign of HIV disease in a non-lactating woman, and occasionally occur during pregnancy (6.13).
(2) A varied group of subacute or chronic infections, including tuberculosis, which you need to distinguish from tumours of the breast.

24.2 Breast lumps

Most breast diseases make the breast lumpy. Sorting out these lumps can be difficult. The important decision is whether or not a patient has a carcinoma.
Consider all lumps in the breast as malignant unless you are *sure* they are benign. No woman should be left with a lump in the breast if she can have it removed by aspirating a cyst or by excision. After the menopause, lumps in the breast are more likely to be malignant.
A normal breast is slightly and uniformly nodular, especially before the menopause; this nodularity is maximal before menses. At the menopause the nodularity becomes less, and more fat is deposited.
The classical signs of malignancy are:
(1) fixation of the lump to the skin or to *pectoralis major* muscle,
(2) enlarged nodes in the axilla,
(3) '*peau d'orange*' (24-1),
(4) disease of the nipple (resembling eczema, unrelated to Paget's disease of bone).

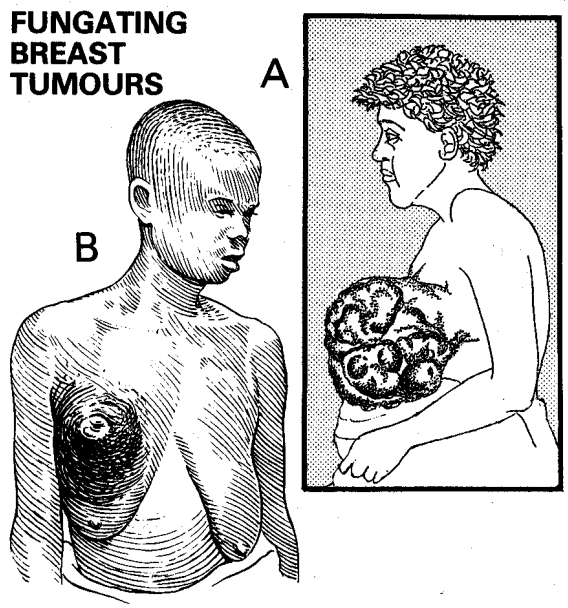

Fig. 24-1 TWO FUNGATING TUMOURS OF THE BREAST.
Unfortunately, many patients may present late when their tumours are already fungating like this. A, phylloides tumour (uncommon: see below). B, fungating carcinoma (very common); note the '*peau d'orange*' appearance of the breast skin, resembling an orange peel, and the malignant ulcer.

The absence of these signs does NOT exclude carcinoma. Their presence increases the chances of it, but they are not confirmatory, because they can also be caused by tuberculosis or fat necrosis.

(a) History. How long have the symptoms been present? Is there any pain? Is it associated with menses? If there is pain, is it in one breast or both? Is there any discharge from the nipple? Is it watery, bloody, or like thin pus?

(b) Inspection. Ask for permission, and if you are male, ensure you have a chaperone present, *i.e*. a person who acts as a witness during any intimate examination. First examine the patient sitting up undressed to the waist, then lying down. Examine both breasts, starting with the normal breast, regional lymph nodes, chest, liver, and the skeleton.
(1) The nipples for position, retraction, and cracking.
(2) The areolae for pigmentation, swelling of Montgomery's tubercles (the external orifices of the areolar glands), a rash or eczema ('Paget's disease').
(3) The skin for prominent veins, sinuses, ulcers, and '*peau d'orange*' (orange-peel skin), which is thickening due to lymphoedema under the skin.

(c) Palpation

(1) Each of the 4 quadrants, then the subareolar area.
(2) For lumps, note their size and site, and whether they are discrete and well-defined, single or multiple; also their consistency, warmth, tenderness, mobility, and surface.
(3) For tethering to the skin, or to *pectoralis major*. Test for this by asking the patient to place her open hand on her waist, and then ask her to press downwards to tense this muscle, while you try to move the lump.
(4) With your finger and thumb behind each nipple, and look for any discharge.
(5) The axillary nodes: medial (pectoral), lateral, anterior and posterior. Note their number and size, and if they are fixed to the skin, or to deep structures. Although the number is actually not so relevant, the presence of a metastasis in the sentinel node is, *i.e*. the first 'guard' node where lymph spreads.

If you are not sure if there is a lump or not, examine it again 2 or 6wks later, at the opposite phase of the menstrual cycle. Write down your findings clearly on the 1st occasion!

(d) Diagnosing cysts in the breast

If a lump is deep and spherical in all directions, it is probably a cyst; it may or may not be fluctuant, and is usually benign. Distinguish cysts from solid lumps by aspiration with a wide-bore needle, or using ultrasound.

(1) **Fibrocystic disease** (or fibroadenosis) is the commonest cause, especially between 25-50yrs, and makes both breasts abnormally granular, usually with premenstrual pain and some tenderness. One or more of these granular areas may be sufficiently obvious to be palpable as a cyst, and there may be a clear discharge from the nipple; rarely, this is blood-stained.

(2) **Breast abscess** may present by finding pus on aspiration, especially in HIV disease, when there is not much inflammatory response: this still needs drainage (6.13).

Other, rarer causes are:
(3) **Galactocele**: a residual milk-containing cyst, the contents of which may solidify.

(4) **Intracystic papilliferous carcinoma**: aspiration yields blood-stained fluid, and does not make the cyst disappear entirely.

(5) **Carcinoma of the breast with colloid degeneration**: aspiration yields only a little thick fluid, and does not make the cyst disappear.

(6) **Cystadenoma phylloides**: a rapidly growing benign giant fibroadenoma, which becomes partly necrotic and fluctuant. The skin over it may ulcerate, but is not infiltrated.

(7) **Hydatid cyst** (15.12): a possibility in endemic areas: look for cysts elsewhere, especially in the liver.

(e) Diagnosing solid lumps in the breast

Careful palpation will give you a good idea of the nature of a lump, but even experts are misled. These are the classic features:

(1) **An 'antibioma'** is the result of treating an abscess with antibiotics, and not draining it. The lump is usually tender, but not always so. It is indurated but smooth; aspiration may produce pus. There may be tender axillary nodes and even '*peau d'orange*'.

(2) **A fibroadenoma** (common) is a smooth, well-defined, usually painless firm lump 2-5cm in diameter (but which may be much larger) that moves freely in the breast (a 'breast mouse'). There may be several fibroadenomas in one or both breasts (24-2). From its firmness such a lump could still be a carcinoma; mobility is the important sign, so is lobulation. Be careful to distinguish a fibroadenoma, which is an isolated lump, from an area of nodularity due to fibroadenosis, which is a different disease, featuring pain and nodularity increasing before and during menstruation. The patient is almost always 15-45yrs, and usually 18-30yrs.

(3) **A giant fibroadenoma** (uncommon) presents as a large breast filled with a large, deeply lying, firm, smooth, lumpy mass (24-6). If it is untreated, it may fungate (24-1) or develop into a sarcoma.

(4) **A neurofibroma** (rare) feels hard, like a fibroadenoma, but may be soft, and may be one of many similar tumours elsewhere (neurofibromatosis).

(5) **A lipoma** (uncommon) feels like breast tissue, but has an indistinct outline separating it from the surrounding normal breast.

If there is a serous, dark or blood-stained nipple discharge, it may herald the presence of an intraduct papilloma, adenoma or carcinoma. If a lump is palpable, it is more likely to be a carcinoma. The prognosis after local resection is usually good.

(6) **An organizing haematoma** or **fat necrosis** may occur after trauma as fairly discrete, mildly tender lumps.

(7) **Tuberculosis** is less often seen in the breast than in the axillary lymph nodes (17.4) but you should suspect it if there are signs of HIV disease; it closely resembles carcinoma. Suspect tuberculosis if there is a sinus and the swelling and '*peau d'orange*' are generalized. The mass is painless, and may be attached to the skin or the muscles of the chest wall. The nipple may be retracted. Look for signs of tuberculosis elsewhere. The breast itself may be swollen due to enlargement of axillary nodes, and may not be the seat of the primary pathology.

(8) **A carcinoma** (usually squamous) may be ductal or lobular (both common). It is a hard, painless, fixed mass often with tethering of the skin or attachment to *pectoralis major*, overlying localized '*peau d'orange*' and fungation of tumour through the skin.

(9) **Mastitis carcinomatosa** (rare) is a highly malignant form of carcinoma seen during pregnancy. It is more generalized, and more like inflammation, or Burkitt's lymphoma, than the hard, fixed mass of a typical carcinoma.

(10) **Burkitt's lymphoma** (17.6) is rare, usually bilateral, occurring at 14-25yrs. It may simulate mastitis carcinomatosa, but is not particularly associated with pregnancy. The skin is stretched, and may ulcerate; there are usually other tumours elsewhere.

(f) Managing cysts in the breast

First exclude a hydatid cyst (if that is at all likely), by looking for hydatid cysts (15.12) elsewhere.

Aspirate the cyst with a wide-bore needle as completely as you can.

If the fluid you aspirate is blood-stained, explore and excise the lump.

If a lump remains after you have aspirated the cyst, excise the lump completely.

If the fluid is clear and the lump disappears, as is usual in fibrocystic disease (the commonest cause), no further treatment is necessary. Do a regular follow-up. If the cyst appears again, or other cysts appear, aspirate again. If at any time lumps don't disappear, remove them as above.

(g) Managing solid lumps in the breast

Consider any lump as potentially malignant, until you are sure it is benign.

If it is an obvious fibroadenoma, shell it out completely unless it is <1cm in diameter, when you can review it at 3-monthly intervals (24-5).

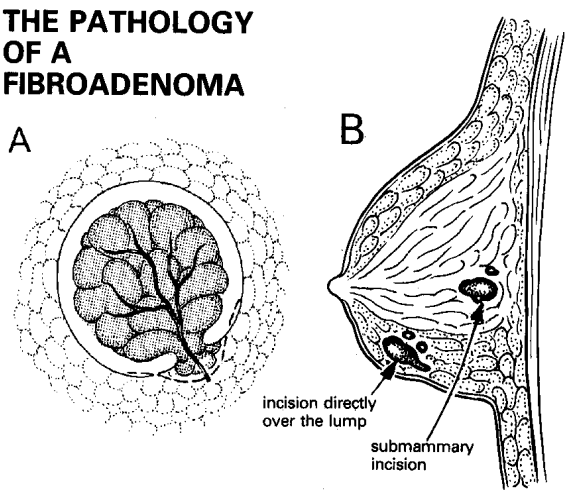

Fig. 24-2 PATHOLOGY OF A FIBROADENOMA.
A, tumour is attached to the capsule by a stalk carrying its blood supply. Sometimes the tumour extends into the capsule near the stalk. B, remove a superficial fibroadenoma by incising directly over it. If it is deeper, a submammary incision may be best.
After Rob C, Smith R. Operative Surgery Vol 1, Butterworth 2nd ed 1981 with kind permission.

If you are not sure but think it is benign, remove it completely together with a 2cm margin of tissue around the lump and send it for histology. *Don't try to perform a trucut biopsy on a small lump, especially if it is mobile*; you may well miss it or just biopsy unrepresentative tissue!

If there is a lump and a discharge from the nipple, it is likely to be a duct papilloma, adenoma or carcinoma. Excise the duct involved (24-7), as well as the lump with a 2cm margin.

If you suspect tuberculosis, get an aspirate for AAFB (or PCR) from the breast and axillary nodes. Otherwise do an open biopsy; it is better to do this on the nodes because the breast may not heal well. Then treat (5.7) for TB; there is no need for mastectomy.

If you suspect malignancy, try to get a diagnosis first to plan your treatment. Fine needle aspiration cytology is the best method but needs careful immediate expert examination of the slides. It is almost essential if you suspect Burkitt's lymphoma. Do cytology on axillary nodes: if you find breast cells, you have proved metastases. Or, do a trucut biopsy if the lump is big enough and you can hold it firmly in the hand (24-3). Otherwise, excise the lump fully with a 2cm margin of normal tissue *and remember to orientate it properly for the pathologist, by marking it with indelible inks or coloured threads*. If this is not feasible, take a small incision biopsy. If the result is carcinoma, proceed as in 24.4.

If there is a fungating mass, perform a 'toilet' mastectomy (24.5); it is cruel on the patient to waste time getting a histology result when it will not affect the management.

(h) Special tests. Ultrasound is useful to distinguish between cystic and solid lesions, and between discrete lumps and lumpy breasts. It is unnecessary for an easily palpable lump which you can aspirate.

TRUCUT BREAST BIOPSY

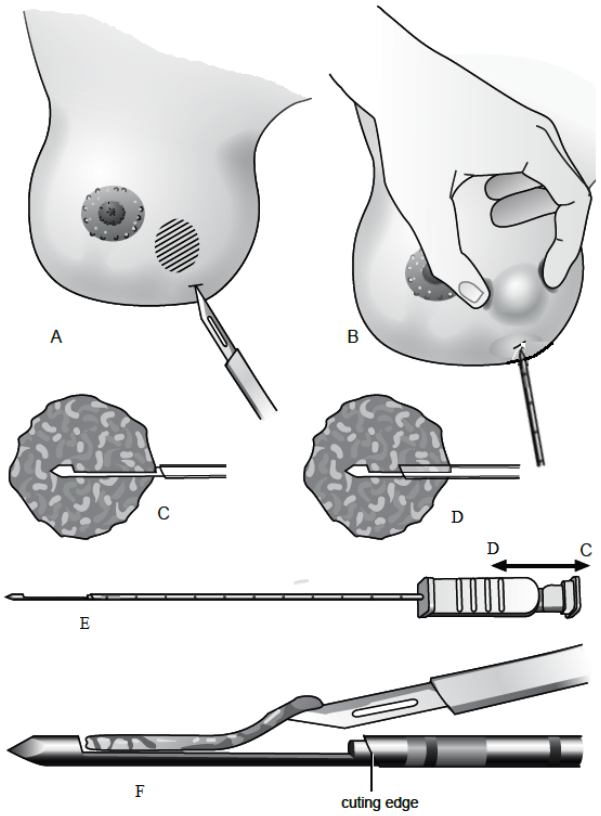

Fig. 24-3 TRUCUT BIOPSY OF THE BREAST (GRADE 1.2).
A, using LA, make a small cut with a blade. B, introduce the closed biopsy needle into the lesion, with an assistant steadying the lump. C, open the trucut biopsy device. D, close the trucut device by pushing the handle, and withdraw the needle. E, positions of trucut needle open and closed. F, examine the specimen obtained to see if there is adequate tissue sampled.

Mammography needs special equipment where the breast is squeezed between two plates and X-rayed; it is sometimes painful and may miss some carcinomas.

Try to screen women with a strong family history of breast cancer before the age of 40yrs, or contralateral breast cancer, especially of the lobular type.

EXCISION OF A BREAST LUMP (GRADE 2.3)

ANAESTHESIA. LA is only really feasible if the lump is very small and superficial. Regional anaesthesia using intercostal nerve blocks works well (but a pneumothorax may occasionally result); otherwise use ketamine or GA.

INCISION. If you are removing a benign lump from a woman, try not to disfigure the breast or compromise future lactation. Use a circumareolar (24-4A), circumferential (24-4B) (less satisfactory), or submammary incision (24-4C).

N.B. Avoid a radial incision.

N.B. With all incisions, use a sharp knife.

If you suspect malignancy, excise the lump *with a margin of at least 2cm of normal breast, and orientate the specimen carefully for the pathologist.*

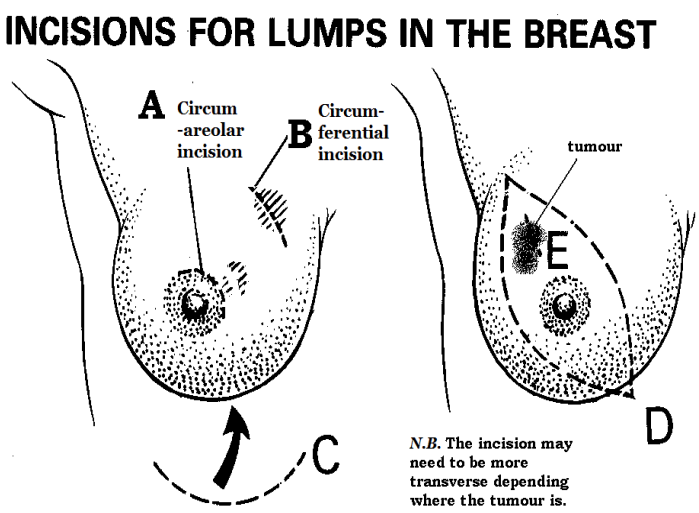

Fig. 24-4 INCISIONS FOR REMOVING LUMPS FROM THE BREAST. A, if the lump is within 5-8cm of the nipple, make a circumareolar incision, not longer than ⅔ the circumference of the areola. B, if the lump is further away, make a curved circumferential incision over it, parallel to the areola. C, if the lump is deep in the breast, you may be able to use a submammary incision. D, slant a mastectomy incision as transversely as possible towards the axilla. E, if your histology services are good enough to justify taking a biopsy, make a radial incision within the area of a possible later mastectomy, so that you can excise the scar.

By a circumareolar incision (24-4A), remove a lump up to 5cm from the nipple. Gently dissect radially through the breast from the areola, in line with the ducts. You can cut ⅔ round the circumference of the areola without compromising its blood supply.

By a circumferential incision over it (24-4B), remove any lump if an inframammary incision is too far away. This produces an obvious scar, but will be much more aesthetic than a radial scar.

By a submammary incision (24-4C), approaching the lump from the underside, remove deep inferiorly placed lumps: this will be less easy than the circumareolar or circumferential incisions. Cut round the infra-lateral quadrant of the breast: in light-skinned women this follows a pigmented line. Hold the breast up while you make your incision in this line, and free the breast from the pectoral fascia. Continue to hold the breast up. Incise the posterior surface of the breast, until you have exposed the lump. Grasp it with forceps, and then free it from its bed with a scalpel or curved scissors.

Bleeding is not usually much of a problem. If it is difficult to control immediately, pack the wound with swabs, apply pressure for 5-10mins, remove the swabs, and then either transfix and tie the bleeding vessels, or control them with diathermy.

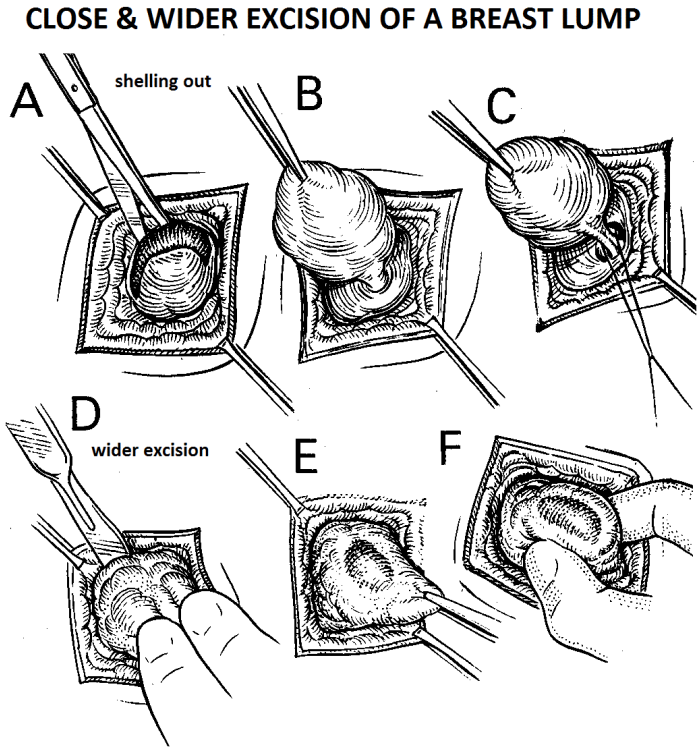

Fig. 24-5 CLOSE AND WIDE EXCISION OF A BREAST LUMP.
A-C, remove a benign lesion through a small incision with minimal disturbance to the surrounding tissue. D-F, WIDER EXCISION for the removal of a suspicious lump: expose the lesion, *but don't cut into it,* and remove the lump with a small part of the surrounding breast.
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Close the cavity with interrupted sutures of absorbable suture on a half-circle needle. If the cavity is too large to be completely obliterated by sutures, and bleeding is troublesome, insert a Penrose drain.

Close the skin with 3/0 or 4/0 subcuticular monofilament. Post-operatively, apply a tight binder, which is uncomfortable, or better, a pressure dressing of adhesive strapping.

(i) Difficulties with tumours of the breast

If there is a giant fibroadenoma (24-6), simple removal may not be possible, and you may have to remove it piecemeal with the risk of seeding cells resulting in recurrent fibroadenomas. Avoid a mastectomy unless the tumour is fungating. If it only occupies part of the breast, you may be able to shell it out. If you preserve normal breast tissue where you can, the breast may surprisingly return to its normal shape afterwards.

24.3 Other benign breast conditions

(a) Managing a discharge from the nipple

This can be:
(1) The normal action of breasts in pregnancy. Colostrum can be discharged from the 16th week of pregnancy, and even earlier in multipara.
(2) The normal, usually milky discharge after lactation stops. This may however still persist for months and occasionally years, especially if lactation is prolonged.
(3) The clear, or less often blood-stained, discharge due to an intraduct papilloma, adenoma or carcinoma.
Or rarely,
(4) The discharge associated with fibroadenosis.
(5) The discharge associated with periductal mastitis.

Discharge is more serious if it comes from one duct rather than from many, if it is bloody, or if it is associated with a lump. At the start of the examination, *don't palpate the breast in the normal way*, because this may squeeze out any secretion which has accumulated, and you want to see exactly where it is coming from.

Instead, with the patient supine, gently wipe the nipple clean. Then, press with one finger 3cm distal to the areola, and move it towards the nipple. Start at the 1o'clock position and move progressively all round the breast to the 12o'clock position. If there is any discharge, wipe it away and note its position. Then examine the breast in the usual way.

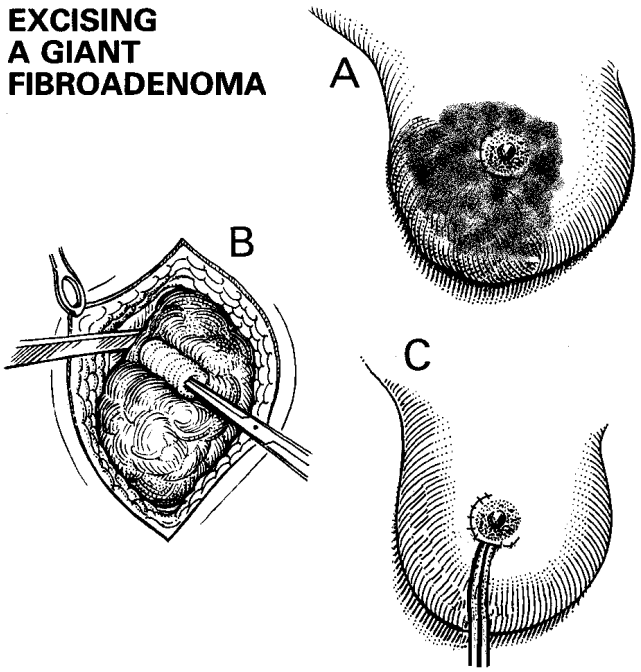

Fig. 24-6 EXCISING A GIANT FIBROADENOMA.
A, make a circumareolar (or for a very large mass, a circumferential) incision. B, shelling out the tumour. C, leaving a drain in place. *N.B.* Very rarely, only if there is gross pressure ulceration, you may need to perform a simple mastectomy.
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In a pregnant woman, if the fluid is clear and comes from many ducts in both breasts, reassure her.

If both breasts continue to discharge milky fluid from many ducts, this is galactorrhoea, which may be due to medications (sedatives and anti-depressants), or rarely a pituitary tumour raising prolactin levels. Jasmine is effective if you cannot get bromocriptine.

If there is a recurrent discharge from several points on the nipple, watery or viscid, green, white, black or occasionally bloody, suspect duct ectasia and/or periductal (plasma cell) mastitis, which is common in smokers. It can also present as a hard, tender swelling with redness of the overlying skin, which you can confuse with an acute breast abscess, or a rapidly growing carcinoma.

If there is fibrocystic disease, the retention cysts it causes may occasionally cause a discharge from one breast, seldom both. Aspirate the cyst if there is one. If it does not disappear, or if the fluid is blood-stained, perform an excision biopsy.

If there is a watery, or bloody, or dark discharge from one duct, usually without a lump, this is probably due to an intraduct papilloma or adenoma. If there is a lump, it is more likely to be a carcinoma; even so, the prognosis is usually good. Excise the lump with the duct (see below).

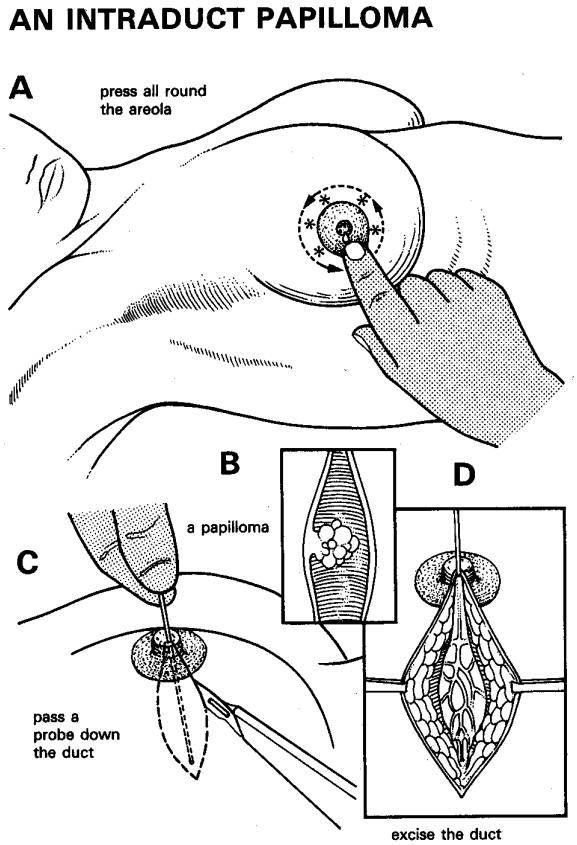

Fig. 24-7 BREAST MICRODOCHECTOMY.
A, carefully palpate all round the breast to find out which segment the discharge is coming from. B, lesion in the wall of a duct which might equally well be a duct adenoma or a carcinoma. C-D, pass a fine probe down the duct, and excise it with some of the surrounding tissue.

(b) Breast microdochectomy (GRADE 2.3)

Aim to excise a single duct system with its surrounding tissue. Try to make sure that neither the patient, nor anyone else, squeezes the breast during the 2-3 days before you do so. Under GA, find the orifice of the affected duct by squeezing the secretion out of it. You may be able to feel the lesion under the areola (24.2). Pass a fine probe or a hypodermic needle with a blunt end along the duct (24-7C). Ask your assistant to hold this, while you excise an oval of skin and breast tissue with the duct and the lesion. Make sure that you excise the probe with a margin of at least 2cm of macroscopically normal tissue horizontally and vertically all round the duct, except at the nipple (24-7D). Suture the deeper layers with plain absorbable suture to obliterate the dead space. Close the skin with 3/0 monofilament. There is no need for a drain. If haemostasis is not good, apply a pressure dressing. Send the specimen for histology. Remove sutures at 7 days.

At review, if it is a papilloma (75% chance), nothing further is needed. If there is an *in situ* carcinoma (15%), do a careful follow-up for at least 6 months.

The operation itself should be sufficient, but if there is an invasive carcinoma and the tumour is >2cm diameter, perform a mastectomy unless radiotherapy is available.

(c) Managing a sinus or fistula in the breast

A sinus or fistula may discharge milk, or a non-specific fluid. A milk fistula can follow a breast abscess, trauma, tuberculosis, a cancer of the breast, or occasionally the presence of a foreign body, or fungal infection.

If there is a milk fistula, and the patient is or should be breastfeeding, try to improve or re-establish breastfeeding soon. The fistula will probably heal. If it does not, it will probably do so when breastfeeding ceases at the normal time.

CAUTION! A milk fistula is not an indication to stop breastfeeding. Rather, it is an indication to re-establish breastfeeding soon, if lactation has stopped.

(d) Nipple diseases

(1) **Paget's disease of the nipple**, a localized eczema, is unilateral and, though rare, a sure sign that there is an underlying intraduct carcinoma. Biopsy the nipple skin and if you find 'Paget' cells, perform a mastectomy.

(2) **Chronic eczema** (uncommon) is bilateral. Clean the nipples frequently with soap and water. Apply zinc and salicylic acid paste (Lassar's) 1%, or hydrocortisone ointment 1%. Make sure you follow up the patient and exclude Paget's disease!

A painless ulcer on the nipple may be a syphilitic chancre if there is a history of nursing an infected child or kissing by an infected individual; check the RPR or VDRL and treat with penicillin.
(*N.B.* The mother of a syphilitic infant does not get re-infected by her own child.)

(e) Difficulties with other breast diseases

If there is evidence of acute inflammation – a recent history, throbbing pain, and tenderness – *don't wait for fluctuation*. Treat as for a breast abscess (6.13). Acute infection may be difficult to differentiate from mastitis carcinomatosa (24.2).

If both breasts are enlarged, with pitting oedema, suspect some generalized disease, such as cirrhosis, nephrotic syndrome, or heart failure.

If there are small fibrotic axillary nodes, not the typical enlarged matted tuberculous nodes, and no signs of tuberculosis elsewhere, there may be chronic non-specific infection following repeated infection of the hand and arm, usually from wounds, or filariasis (34.14).

If the nipple is chronically ulcerated, suspect that this is associated with an underlying duct carcinoma, unless there is a clear history of trauma. Get a biopsy to exclude rare causes such as syphilis and tuberculosis.

If there is one swollen breast with pitting oedema and no palpable mass, this may be:
(1) Primary TB of the breast, or
(2) Secondary to chronic axillary lymphadenitis.
The affected breast is larger than the opposite one, is not tender or only slightly so, and almost always shows '*peau d'orange*'. The axillary nodes (usually the lateral pectoral group) are commonly matted together, and may be attached to underlying structures and the skin. In TB, there may also be a discharging sinus. Look for signs of TB elsewhere, especially enlarged nodes in the other axilla, groins, and abdomen. Get a chest radiograph, and do an ESR. This manifestation of TB affecting the breast via the axillary nodes is more common than primary TB of the breast.
In endemic areas, think of filariasis (34.14).

If both breasts enlarge prematurely, associated with menstruation, there is precocious puberty. Look for an ovarian tumour, or an intracranial lesion, which may disturb the gonadotrophin releasing mechanism.

If one breast is very much larger than the other during puberty, but is otherwise normal, this is probably congenital GIANT HYPERPLASIA, and may affect both breasts. Later, the breasts usually become the same size but may enlarge again in the 3rd decade, especially following pregnancy. *Don't remove breast tissue in a child for a biopsy* because you will destroy the normal development of the breasts.

If both breasts enlarge hugely in an adult, this may cause backache; very large breasts can be made smaller by reduction mammoplasty but this is difficult plastic surgery.

If extra breasts develop, they may develop anywhere along the nipple line from the axilla, along the chest wall down to the thigh. Rarely, these breasts are functional. You should be able to excise them easily.

If there is a soft fatty lump in a lady >70yrs, which feels as if it might be a lipoma, suspect that it is in fact a carcinoma, which can be as soft as a lipoma at this age.

24.4 Breast carcinoma

Carcinoma of the breast is common and can occur at any age >20yrs, but is most common from 50-70yrs, particularly in nullipara and in women who started childbearing late; it is also common in the sisters of patients with the disease, and to a lesser extent in their daughters. It appears to arrive earlier in HIV disease, and may act more aggressively. It may be a consequence of the use of hormone replacement therapy which used to be advocated to prevent menopausal osteoporosis.

Carcinoma of the breast may also occur in males, *but is not related to gynaecomastia* (24.6).

Breast tumours arise commonly from the duct system or less commonly from the lobule. When confined and not invading the basement membrane, they are described as *in situ* carcinoma, and therefore potentially curable by surgical excision. Most invasive ductal carcinomas have no specific features, but medullary, tubular, mucinous, papillary and cribriform have better prognosis, whilst signet ring, clear cell and inflammatory types have worse prognosis. **Mastitis carcinomatosa** is of this last type. Lobular carcinomas are often bilateral but have better prognosis than invasive ductal carcinomas, unless they are of the pleomorphic type.

Sarcomas are rare and arise from fibrous stromal tissue. They occur in young girls <20yrs as **phylloides** tumours (24.2). These may be well circumscribed but tend to recur if a margin of excision is not included with the tumour, and rarely metastasize.

Lymphomas (rare) also occur, especially Burkitt's lymphoma (17.6).

Breast carcinomas form no capsule; they invade locally through the lymphatics, and spread widely through the bloodstream. The prognosis is related to:
(1) the stage at which treatment starts,
(2) the histological tumour type, and
(3) less significantly, the treatment.

The stage at which the diagnosis is made is critical, so persuade your staff to include breast palpation in every general clinical examination. A 1cm lump represents 30 doublings; growth however occurs in spurts and dormant periods are frequent but irregular. Occasionally you may detect a carcinoma as a suspicious lesion on a mammogram or ultrasound when it is still not palpable.

Carcinoma of the breast may present as a painless lump in the breast (80%), as enlargement of a breast, as ulceration, or as a discharge from the nipple which is usually but not always blood-stained.

Treatment is mainly surgical but as in any other part of the body, surgery can only cure if it remains localized and has not spread elsewhere. If radiotherapy is available, it is the preferred treatment for affected axillary nodes. Anti-oestrogens improve survival and chemotherapy can result in remission. Many patients present late with foul, stinking ulcers. Metronidazole often helps here to remove the odour, and a 'toilet' mastectomy, if it is possible (the growth may be fixed to the deep structures and make it not worthwhile), relieves suffering, and may make the last months more bearable.

(a) Staging and prognosis

The Manchester system of staging describes:
Stage I: growth is confined to the breast, skin involvement smaller than tumour
Stage II: growth is confined to the breast, palpable mobile axillary nodes
Stage III: skin involvement larger than the tumour, or tumour fixed to underlying fascia
Stage IV: distant metastases, fixed axillary nodes, palpable supraclavicular nodes, satellite nodules.

The TNM classification describes **t**umour size, **n**odal involvement and distant **m**etastases:
T0: no tumour,
T1: tumour <2cm diameter,
T2: tumour 2-5cm,
T3: tumour >5cm,
T4: tumour fixed,
N0: no nodes,
N1: mobile axillary nodes,
N2: fixed axillary nodes,
N3: supraclavicular nodes,
M0: no distant metastases,
M1: distant metastases.

The systems overlap as follows:

	T0	**T1**	**T2**	**T3**	**T4**
N0	Stage I				
N1	Stage II				
N2	Stage IIIA				
N3	Stage IIIB				
M1	Stage IV				

5-year survival should be >65% for stage I and 60% for stage II, but dropping to 15% for stage III and <5% for stage IV.

On microscopic examination the axillary nodes are involved in 10% of patients in Stage I, although they may not be obvious for 20yrs. Tumours in the lateral half of the breast have a better prognosis. If the tumour is Stage I in the lateral half of the breast, there is a 90% chance of cure surgically. If it is in the medial half of the breast (less common), the prognosis is worse, because it is more likely to spread to the internal mammary nodes.

Inadequate local removal of tumour will result in local recurrence; wide excision of a tumour >2cm diameter without radiotherapy will result in a 30% chance of local recurrence. However, the more radical operations (the standard Patey's, removing *pectoralis minor*, or Halsted's, removing *pectoralis major*) don't confer any survival benefit by virtue of their more guaranteed clearance of tumour.

(b) Management

(1) **Stage I**
Excise the lump with 2cm of normal tissue around it (24-5D-F) and send it for histology. If the result confirms a carcinoma <2cm diameter, do a follow-up every 6 months for a year, and yearly thereafter. If the carcinoma is >2cm diameter, or the excision margin is <2cm, perform a mastectomy.

You can try to map *sentinel nodes* especially in the axilla if you inject 0·5-2ml of blue dye around the lump, and then 10-15mins later explore the axilla (17.3). You may be able to increase your detection of axillary nodes by ultrasound. If you then remove *sentinel nodes* which have taken up the blue dye, as well as obviously palpable nodes, you will get a better definition of the spread of the disease, and be able to advise better about further treatment options.

*N.B. Nipple-sparing mastectomy may be feasible in a younger woman, with a view to later insertion of a prosthetic implant (*i.e. *silicone):* think about this if you can arrange to get it done.

(2) **Stage II**
Perform a mastectomy and remove as many obviously involved nodes as you can. This 'axillary sampling' is controversial, but axillary clearance is difficult and may result in damage to the axillary vein and nerves, and may not give any better result. If available, administer radiotherapy if axillary nodes are involved.

If the patient cannot contemplate removal of the breast, you could do a wide excision of the lump but this should include axillary node excision and radiotherapy if the nodes are involved. So it may not actually be the best option if radiotherapy is not available; also the risk of local recurrence is higher, and if the breast is small, a wide excision might not lead to a better oncological result.

Treat with tamoxifen 20mg od if nodes are involved, for 5yrs. Administer chemotherapy (cyclophosphamide 100mg/m² od for 14 days, and methotrexate 30mg/m² plus either 5-fluorouracil 500mg/m² IV on days 1 and 8, or doxorubicin 60mg/m² every 3wks for 6 cycles) if the patient is <40yrs old and HIV-ve.

Alternatively, as good an effect can often be achieved by bilateral oophorectomy.

(3) **Stage IIIA**
Perform a more extensive mastectomy to clear tumour with a 2cm margin and remove as many involved nodes as you find. Occasionally the tumour becomes fixed to the chest wall without significant lymph node spread; if you can remove all of the tumour the first time round, you may give her long disease-free survival, otherwise local recurrence is inevitable.

Advise radiotherapy to the chest wall and axilla if possible, and tamoxifen 20mg od up to 5yrs.

Alternatively, advise bilateral oophorectomy.

(4) **Stage IIIB**
Perform a mastectomy *only* if you think you can clear tumour with a 2cm margin (unlikely) or if there is fungating tumour eroding through the skin (a 'toilet' mastectomy). You probably will not be able to close the defect except with a skin graft; unless the tumour mass is very infected, it's best to do this at the time of the mastectomy rather than delaying.

Interference with fixed axillary nodes may damage nerves and blood vessels and is unlikely to be helpful.

If the tumour is firmly fixed to the chest wall, only very extensive surgery is likely to be successful. Radiotherapy is probably palliative at this stage, but tamoxifen 20mg od may help, and if you can administer chemotherapy it may benefit, but you probably will have more deserving cases for your valuable resources.

Alternatively, advise bilateral oophorectomy.

(5) **Stage IV**
No surgery is likely to be helpful here. Treat with tamoxifen 20mg od: occasionally it can produce dramatic results. Radiotherapy to bone metastases may remove constant pain, and bilateral oophorectomy in pre-menopausal women results in remission in c.20%.

(c) Difficulties with carcinoma of the breast

If you suspect Burkitt's lymphoma of the breast, take a needle biopsy, stain a slide preparation, and interpret it yourself (17-1). Or, less satisfactorily, send a biopsy. If you are not confident that you can interpret a slide preparation, do both; excise the lump, make a slide from it, and send the biopsy for histology. If the tumour is large and ulcerating, excise it, and skin-graft the exposed *pectoralis major* as for Stage IIIB tumours. If Burkitt's lymphoma is likely and histology reporting slow, start chemotherapy immediately (17.6).

If you find breast carcinoma in a pregnant woman, it may not necessarily be mastitis carcinomatosa (24.2). Treatment of the breast carcinoma takes precedence, however, over the pregnancy.

If no cytology or histology is available, *don't delay treatment inappropriately to seek confirmation*, but always consider TB as an alternative diagnosis.

The patient may not return, the report may be lost, and there will be too long an interval between the biopsy and the definitive operation. The biopsy scar may interfere with your proposed mastectomy incision.

If there is a suspicious impalpable lesion found on mammography or ultrasound, and you have no way yourself of localizing the lesion, repeat the ultrasound scan if you can, move the affected breast quadrant and make sure you get a histology report. If the lesion is malignant and >2cm, it is best to proceed to mastectomy.

24.5 Modified simple mastectomy

In this operation you always remove the nipple (24-4D).

PREPARATION
Check the side to be operated upon is correct when the patient is still awake and can confirm the side; mark it with indelible ink.

Cross-match blood if the breast is large, you are inexperienced or your diathermy is faulty.

Position the lady supine with the arm on the affected side abducted to 120º and carefully place sterile towels underneath the axilla. It helps to flex her elbow and place her hand under the head.

ANAESTHESIA
You can use intercostal nerve blocks instead of GA.

METHOD (GRADE 3.1). Infiltrate with 1:400,000 adrenaline subcutaneously to reduce bleeding. Make an incision as transversely as possible including the areola and 5cm around the tumour. Ask your assistant to stretch the skin as you cut. Excise a teardrop-shaped ellipse of skin (24-8A). Make it wide enough to let you dissect the breast adequately, and yet not so wide as to make closure difficult. Control bleeding by asking your assistant to press firmly with gauze as you cut.

Dissect back the superior (24-8B) and inferior flaps, in the plane between the subcutaneous fat (usually 1-2cm thick) and the breast fat. Hold the skin flaps *underneath the skin surface* in Allis forceps, and control bleeding of the flaps with haemostats or diathermy.

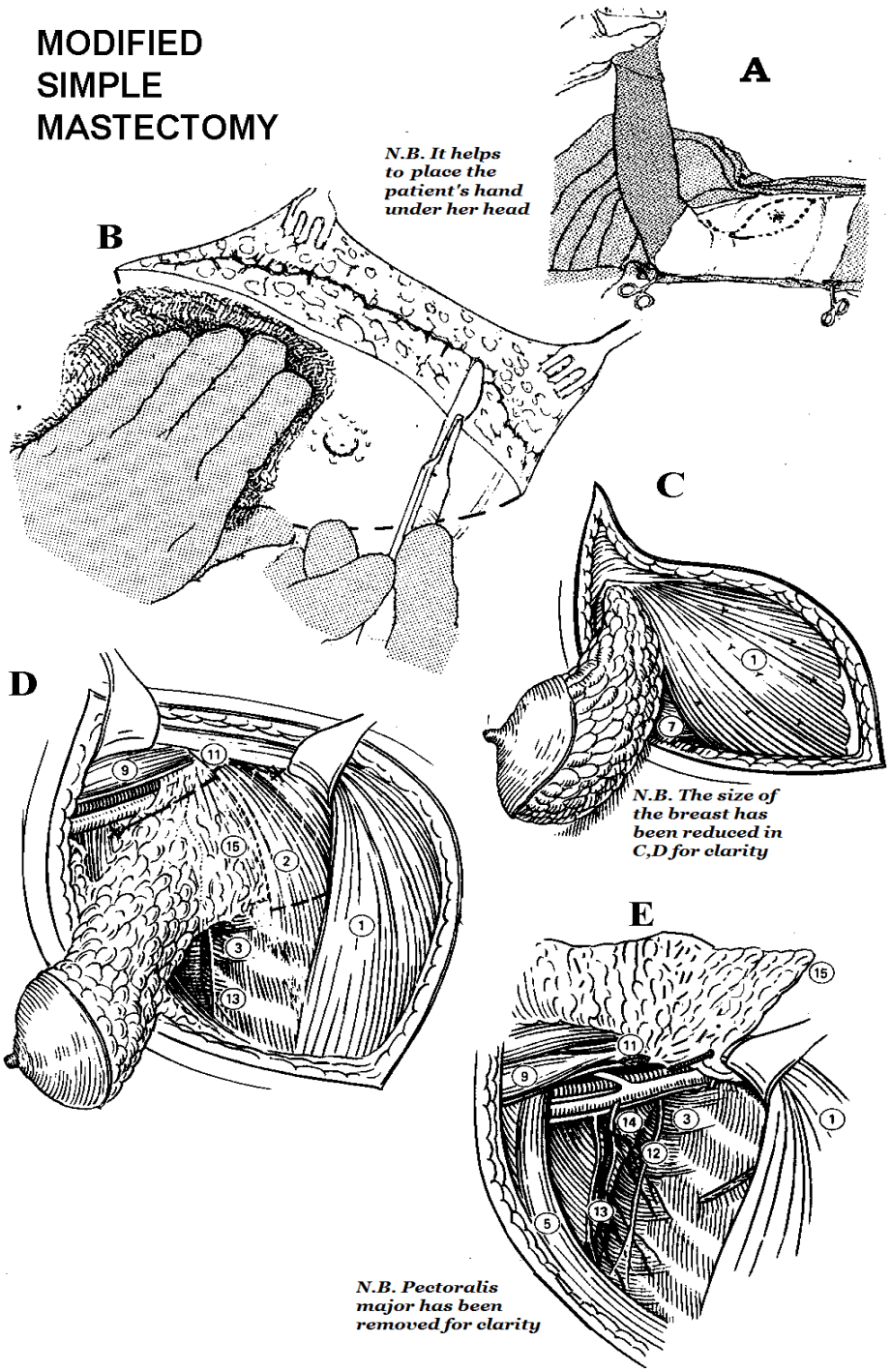

Fig. 24-8 MODIFIED SIMPLE MASTECTOMY.
A, make a transverse-oblique teardrop-shaped incision on the breast with its apex from its superolateral to its inferomedial margin, centred over the tumour mass and including the nipple. B, dissect upper and lower skin flaps. C, continue dissection to the edge of the breast, and dissect it off *pectoralis major*. D, dissect it further off *pectoralis minor*, leaving its axillary tail intact. E, clear the axillary nodes (*if necessary*). *You don't remove the* pectoralis minor. (Numbers as in 24-9)
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Continue the dissection to the periphery of the breast, where you will meet the *pectoralis major* muscle on the chest wall. Do this for upper and lower flaps in turn; when the flaps are raised fully, dissect the breast off *pectoralis major* (24-8C: usually using a knife), clamping bleeding points as you proceed, leaving the axillary tail attached. Laterally, dissect the breast off the *pectoralis minor* which will lead you to the fascia overlying the axillary vessels.

CAUTION!
(1) *Don't make the skin flaps too thin, or open up tissue planes more than is necessary*. The flaps should be ≥1cm thick. Move the Allis forceps lower on the flaps as you proceed.

(2) *Don't remove the pectoral fascia, or muscle,* unless the tumour is sticking to it.
(3) Make the flaps of even thickness.

Then enter the axilla. The axillary tail only extends a short way into the axilla, but you should be able to look at the axillary nodes. Carefully separate the axillary skin from its underlying fat, and try to remove any lymph nodes *en bloc* with the breast. This is easiest if you identify the axillary vein, and tease the axillary contents with small 'Lahey' swabs (3-3).

Take care not to damage the long thoracic nerve and the nerve to serratus anterior (24-8E, 24-9B).

Now control bleeding points by diathermy or ligatures. Irrigate the wound with warm water before you close it. Remove any redundant skin, so that the edges of the incision come together cleanly. If you cannot close the wound completely, cover the bare area with a split skin graft. Insert a suction drain inferolateral to the incision, placing the drain puncture wounds as inconspicuously as possible.

Close the wound with 2/0 absorbable and 3/0 subcuticular monofilament skin sutures. A useful device is a pocket sewn to the bra to carry the suction bottle (24-10).

POCKET ON BRA FOR SUCTION BOTTLE

Fig. 24-10 A pocket sewn to the bra to carry the suction bottle.
After Bradbury C, Parvaiz, MA, Sircar T. Ann Roy Coll Surg Engl 2014;96(3):241 with kind permission.

POST-OPERATIVELY, cover the wound with layers of gauze and cotton wool, and hold them firmly in place with adhesive strapping. Apply a pressure dressing for 3-4 days. Remove the drain when no more blood or serous fluid comes out, usually at 3-7 days. Remove the sutures after 7-10 days, the alternate ones first. Encourage active movement of the arm and shoulder.

DIFFICULTIES WITH MODIFIED SIMPLE MASTECTOMY

If you accidentally buttonhole the skin flap during dissection, use the hole for a drain.

If you damage the axillary vein, *don't panic!* Press firmly, arrange suction and extra light, and call for assistance. *Don't apply haemostats*. You can use bulldog clips if you have them. When you can identify the hole in the vein, try to close it with a round-bodied 4/0 or 5/0 continuous monofilament suture.

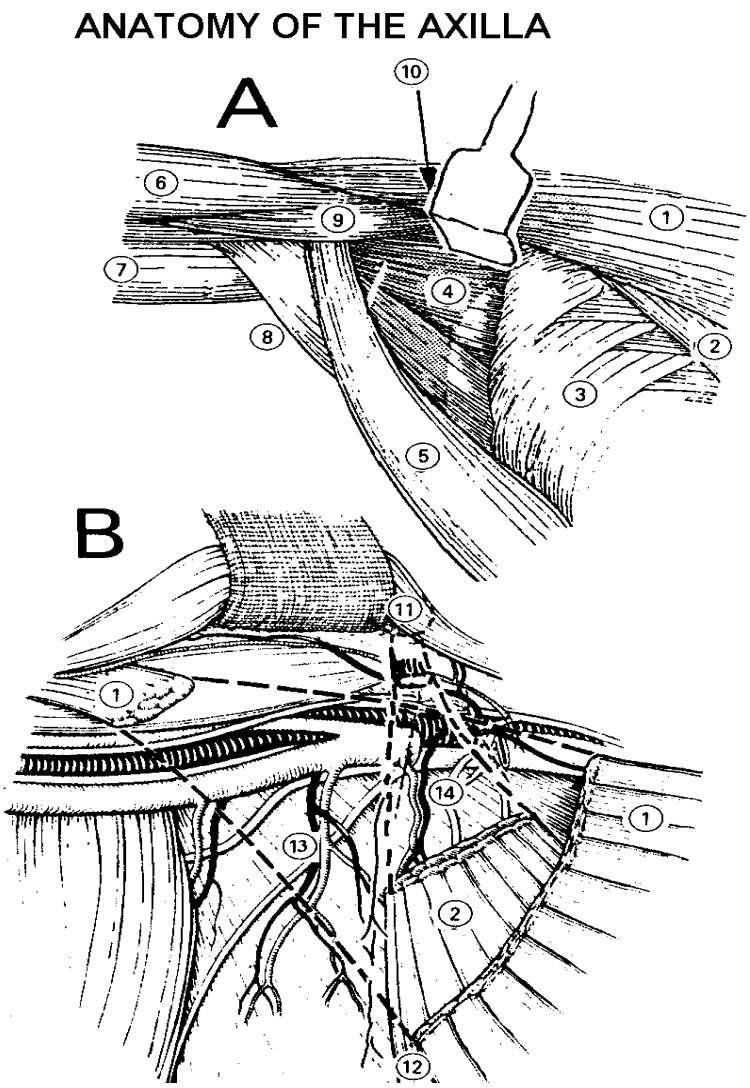

Fig. 24-9 ANATOMY OF THE AXILLA.
A, empty axilla showing its muscles, as if its contents were absent. B, *pectoralis major* shown partially cut away to reveal the structures under it. (*In reality it is retained.*)
(1) *pectoralis major*. (2) *pectoralis minor*. (3) *serratus anterior*. (4) *subscapularis*. (5) *latissimus dorsi*. (6) *biceps*. (7) *triceps*. (8) *teres major*. (9) *coracobrachialis*. (10) axillary space. (11) coracoid process. (12) long thoracic nerve (of Bell). (13) nerve to *latissimus dorsi*. (14) lateral pectoral nerve.
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If the tumour is fixed to the *pectoralis major* or *minor*, remove the affected muscle with the breast. If you dissect along the clavicle, *be careful not to damage the vessels deep to the muscle*. Remember that this is probably palliative surgery, so *don't attempt anything too heroic*.

If a solid mass forms in the scar after you have performed a mastectomy for carcinoma, excise it with a 2cm margin, and send it for histology: it is probably a recurrence.

If the patient is unable to pull the shoulder down post-op, you have damaged the nerve to *latissimus dorsi*. If there is also much pain as well, explore the wound to see if you have tied a ligature round the nerve: if so, carefully untie it.

If the shoulder becomes stiff, you have omitted exercises post-operatively. Start them now!

24.6 Gynaecomastia

If a male <20yrs has a firm tender discoid swelling deep to the nipple just larger than the areola, and concentric with it, this is physiological. It occurs as a normal variant in infants, in boys near puberty, and in young men. In infants it is nearly always bilateral, and is sometimes complicated by mastitis. In young men it may be uni- or bilateral. Reassure all these patients.

If one of or both an adult man's breasts enlarge, this is gynaecomastia. Look for disease of the liver and testes. Distinguish this from breast carcinoma.

Ask about alcohol consumption and marijuana use. Check for leprosy, and enquire about treatment with medicines such as cimetidine, ketoconazole, diazepam, spironolactone, efavirenz, diethylstilbestrol or others. Check also for adrenal or pituitary disease. Often, however, no cause can be found. It may be due to poor waste water management with accumulation of bisphenols (from plastic) which have an oestrogen effect.

N.B. Beware, however, the enlargement of breasts that comes about with simple obesity! However, true gynaecomastia cannot *be treated by liposuction as the enlarged tissue is* not *due to fat!*

DIFFERENTIAL DIAGNOSIS
Carcinoma of the male breast is harder and often irregular in shape with early skin tethering and fixity to the chest wall. Excise it, together with some of the skin and the muscle underneath it. Because there is so little fatty tissue, the tumour infiltrates the skin and deeper tissues at an earlier stage, and the prognosis is worse. Orchidectomy (27.26) usually produces a temporary remission.

SUBCUTANEOUS MASTECTOMY FOR GYNAECOMASTIA (GRADE 2.5)

INDICATION
Social embarrassment with significantly enlarged breasts.
N.B. It is difficult to make both sides look exactly the same post-operatively!

METHOD
Make an inferior circumferential incision in the periphery of the enlarged breast. Dissect off a skin flap containing the nipple making sure it is not too thin, and avoiding buttonholing the skin. Continue towards the nipple and dissect out all the breast tissue down to *pectoralis major*. *Don't remove all the fat*, otherwise there will be a depression under the scar. Make sure haemostasis is good; if you are not certain, insert a suction drain.

If the breast is very large, you may need to preserve the nipple with a tissue flap, using skin and subcutaneous tissue from the inferior part of the breast.

25 The thyroid

25.1 Introduction

The common surgical problem with the thyroid is a painless increase in its size, known as a goitre, or the appearance in it of a painless mass. A painful thyroid is due to either haemorrhage, not uncommon in colloid goitre, carcinoma, trauma, or infection: as in an abscess or thyroiditis (6.12).

(a) Diagnosis
Note the patient's age and gender (more commonly female), and where she lives. Simple and colloid goitres are common in females from 20-40yrs, and in anyone who lives in an iodine-deficient area (25.5). How long has it been present? Has there been a sudden increase in the size of the mass in the neck? Is it painful? Is there difficulty in breathing or swallowing?

Inspect the neck from in front, and feel it from in front and from behind. Give the patient a drink, and confirm that the thyroid swelling moves up on swallowing. Feel the size of its lobes and its isthmus; feel its surface and consistency, and listen for a bruit. If it is woody hard, it is likely to be a thyroiditis, and if it is fixed in the neck, an anaplastic carcinoma. Note the position of the trachea.

Look for retrosternal extension by asking the patient to raise her arms over the head and demonstrating superior vena cava obstruction (prominent neck veins). Look for enlarged neck nodes.

(b) Thyroid activity. You can diagnose moderate and severe thyrotoxicosis clinically. Minor degrees require measurement of the basal metabolism and/or hormone assays.

Suggesting hyperthyroidism: Loss of weight, tremor (especially of the outstretched arms and fingers), sweating, anxiety, hyperactivity, palpitations, tachycardia, cardiac irregularities (flutter, fibrillation), heart failure and exophthalmos, characterized by seeing the sclera below the inferior limbus of the cornea.

If exophthalmos is pronounced, there is sometimes even conjunctival oedema (chemosis), conjunctivitis and diplopia. The thyroid is usually but not always enlarged, and may or may not be nodular. You can often hear a bruit.

(c) Solitary thyroid nodule? First confirm that the nodule is in the thyroid, and then feel carefully for other nodules.

If there are other nodules, there probably is a nodular colloid goitre. If it really is a solitary nodule, it is quite likely to be a papillary carcinoma (which has a good prognosis with radical surgery) or a follicular carcinoma (which has equally good prognosis if found early, less so if found later).

(d) Special tests
(1) ULTRASOUND is very useful to detect a cyst, which you can then aspirate. If this has clear fluid, it is unlikely to be malignant.

(2) FINE NEEDLE ASPIRATION is only helpful if it can distinguish a well-differentiated carcinoma from normal thyroid tissue: this needs an expert.

(3) RADIOGRAPHY of the neck gives important information about compression and deviation of the trachea. Radiography of the chest will show if there is retrosternal extension or signs of cardiomegaly.

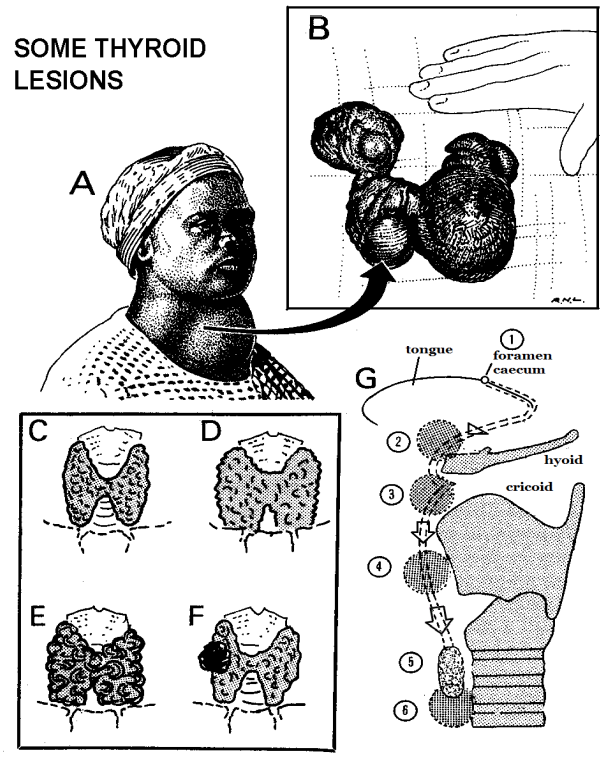


Fig. 25-1 SOME LESIONS OF THE THYROID.
A, patient with a non-toxic adenomatous mass in her thyroid gland. B, mass removed at thyroidectomy. C, smooth, soft, symmetrical goitre of puberty or pregnancy. D, large, smooth, firm, symmetrical swelling of a colloid goitre (25.5)**, thyrotoxicosis** (25.2)**, or Hashimoto's disease** (25.8)**. E, large, nodular, firm, asymmetrical goitre. F, solitary nodule of an adenoma, carcinoma, or cyst. G, congenital abnormalities of embryological thyroid 'migration'** (25.3)**: (1) *foramen caecum.* (2,3,4) positions for thyroglossal cysts. (5) pyramidal lobe (sometimes absent). (6) mediastinal (substernal) goitre.**
A,B, after Bowesman C. Surgery and Clinical Pathology in the Tropics, Livingstone, 1960 with kind permission.

25.2 Hyperthyroidism (Thyrotoxicosis)

There are two main types of hyperthyroidism:
(1) Idiopathic, often related to introduction of iodine to the salt in an endemic goitrous area,
(2) Auto-immune, where circulating antibodies cause exophthalmos. The first is much more common.

Features are weight loss, sweating, heat intolerance, agitation, and tachycardia.

There is a much rarer form of hyperthyroidism (de Quervain's thyroiditis) which may be viral and starts with fever, pain and tenderness in the neck and transient release of excess thyroid hormone into the circulation. Occasionally the thyroid may be over-stimulated by the use of amiodarone in treatment of cardiac dysrhythmias.

(a) Medical treatment is the first choice for almost all cases. Treat with propranolol 20-40mg tds to control the tachycardia and carbimazole 5-20mg tds. Propranolol gives a rapid response but is not useful for long-term treatment; you should use it, though, in preparation for surgery. You may have to adjust the dosages in terms of the response; carbimazole will take about 6wks to get a patient euthyroid.

To maintain medical treatment, you can then stop the propranolol, and lower the dosage of carbimazole to 5mg tds and continue for 12-18 months; unfortunately >50% of patients relapse after stopping treatment.

You can use propylthiouracil 200-400mg od instead of carbimazole, reducing the dose to 50-150mg od once you have rendered the patient euthyroid.

Both anti-thyroid drugs are contraindicated in pregnancy, and both can cause leucopenia or thrombocytopenia, so you should warn patients if they develop a sore throat or bleeding problems.

Remember, rarely, a choriocarcinoma (23.10) may present as thyrotoxicosis.

N.B. In de Quervain's thyroiditis, use anti-inflammatory drugs or steroids, *not anti-thyroid drugs*.

(b) Surgical treatment

INDICATIONS
(1) Thyrotoxic goitre,
(2) Poor supply of anti-thyroid drugs,
(3) Relapse of thyrotoxicosis >18 months of medical treatment.

PREPARATION
The patient must be euthyroid before surgery.
Propranolol orally is only effective for about 6h. If the presentation was with severe hyperthyroidism, a crisis may follow the omission of a single dose. Regular doses are especially important just before and immediately after surgery; continue them up to 10 days afterwards to avoid a rebound phenomenon.

Make sure you control the blood pressure well before the operation. Remember to make sure the patient receives both medications with a little water *on the day of operation* (1.8)!

CAUTION! It is dangerous to operate on thyrotoxic patients who have not had anti-thyroid drugs for 6wks pre-operatively. Even then, post-operative thyrotoxic crises (hyperpyrexia (>41°C), agitation, confusion, and seizure) may occur, and prove fatal.

Recurrence of hyperthyroidism after a bilateral subtotal thyroidectomy is very unusual. However, 30% of patients become hypothyroid within 10yrs and need levothyroxine 0·1-0·2mg od. This is an especial hazard if surgery is done on a small thyroid gland. You therefore need to follow up such patients.

25.3 Thyroglossal cyst

A thyroglossal cyst is a smooth, painless, subcutaneous lump which usually lies at or below the hyoid bone in the midline (25-1G). These cysts occur in both sexes equally, usually between 15-40yrs, and are formed from the epithelial pouch that gives rise to the thyroid gland. This runs from the junction between the anterior ⅔ and the posterior ⅓ of the tongue (the foramen caecum), to the pyramidal lobe of the thyroid, just above the isthmus (25-1). Cysts can arise anywhere along this track.

Excision is usually not difficult. Occasionally, however, an extension of the cyst goes up to and through the hyoid bone, which you may need to divide.

EXCISION OF THYROGLOSSAL CYST (GRADE 2.3)

Make a 6cm transverse incision in a skin crease over the swelling. Retract the skin flaps with a self-retaining retractor. Dissect around the cyst carefully, detaching it laterally from the infrahyoid (strap) muscles.

You can inject a little dye into the cyst to delineate any extension superiorly. If it does extend so (behind the hyoid bone), cut a small central segment of the hyoid away with the track using bone cutters. Then use Lahey swabs or a MacDonald's blunt dissector to detach the cyst posteriorly off the thyrohyoid membrane and *mylohyoid*. If the track extends further upwards, ask the anaesthetist to push down on the tongue to improve your view. Excise the cyst, track and hyoid segment *en bloc*.

No vital structures are in the way, and the divided hyoid does not need repair. If a remnant is left behind, the cyst may well recur.

25.4 Physiological goitre

A physiological goitre presents as a uniform, smooth, painless swelling of the thyroid gland, mainly in girls and women of 12-20yrs. It appears to be about equally common everywhere, and does not cause dyspnoea or dysphagia. It often resolves spontaneously as the period of maximal hormonal activity passes. *Don't operate on these goitres!*

25.5 Colloid goitre

Colloid goitres are worldwide, but are endemic in areas of iodine deficiency. They can be prevented by the administration of iodine to the entire community, which also prevents the other manifestations of endemic iodine deficiency (iodine embryopathy, etc).

Colloid goitres occur between 20-50yrs, and affect women more than men. Large ones obstruct breathing by narrowing or displacing the trachea, and they may occasionally obstruct swallowing. Sometimes, they extend into the thorax. They can be 'simple', in which case they are larger and firmer than a normal thyroid and have a regular surface. More often they are nodular. Although the patient may complain of a single nodule, she usually has more than one, with one lobe of the thyroid much larger than the other. There is no bruit over the nodule unless it is a toxic (hyperthyroid) nodule. Treatment, when it is indicated, is surgical. One of the dangers of a colloid goitre is that haemorrhage may cause it suddenly to increase in size.

If a colloid goitre is small, and is causing no obvious symptoms, surgery is not really necessary, and the indications for its removal are cosmetic. Discuss this with the patient in the light of the available surgical and anaesthetic skills and priorities.

If there is dyspnoea or dysphagia, or the gland is large, subtotal thyroidectomy or thyroid lobectomy is indicated, but is seldom urgent.

If there has been a sudden increase due to haemorrhage, and if dyspnoea is present, aspirate the haematoma, if possible under ultrasound guidance. You may have to aspirate at several sites. If this does not relieve the problem, you may have to try tracheal intubation, which will be difficult.

25.6 Thyroid tumours

(a) Papillary carcinomas are of low-grade malignancy, and present as a nodule with or without spread to the lymph glands of the neck. They may be multifocal or bilateral, and are often dependent on thyroid stimulating hormone (TSH) and so may be suppressed by levothyroxine therapy.

(b) Follicular carcinomas spread to bone early, so that the first sign may be a bony metastasis. The patient may have a lump or area of thyroid enlargement, or the thyroid may be clinically normal. Tumours are often greedy for iodine, so treatment with radio-iodine is very effective.

N.B. There may be mixed follicular and papillary features in the same specimen.

(c) Medullary carcinomas are rare and may have a familial incidence, and are transmitted as a Mendelian autosomal dominant. They have a characteristic histological appearance, a poor prognosis, and may be part of a system of multiple endocrine tumours (phaeochromocytoma and parathyroid, or neuro-fibromas).

(d) Anaplastic carcinomas are less rare and occur mostly in elderly women, and are insensitive to radiotherapy; radio-iodine is not taken up.

(e) Lymphomas may also occur in the thyroid (17.6), especially in elderly women.

CAUTION!
(1) Enucleation (*i.e.* remove the nodule only) is easy, but is *not satisfactory* because:
(a) It does not remove a carcinoma completely. This is particularly important if it is papillary.
(b) It gives the false impression of a cure.
(c) It makes a second operation more difficult.
(2) *Don't explore a solitary nodule* unless you can perform a thyroidectomy.

Follow up patients regularly, and measure the nodule. If it enlarges, try to persuade her to be seen again by an expert.

25.7 Thyroidectomy

Thyroid surgery is not easy; you need to have gentle fingers and enjoy careful anatomical dissection. You need to judge carefully whether you have adequate expertise to perform this sort of operation and whether your hospital can cope with the aftercare, because although it is very nice when all goes well, complications are serious and often unforgiving!

INDICATIONS
(1) Goitre, especially causing respiratory compromise.
(2) Hyperthyroidism, especially if associated with a sizeable goitre, well controlled.
(3) A thyroid nodule.

CONTRAINDICATIONS
(1) A physiological goitre (25.4).
(2) A small goitre where the indication for surgery is mainly cosmetic, especially in a young woman who may develop a recurrent goitre later in life.
(3) Thyrotoxicosis not controlled.
(4) Thyroiditis.

N.B. Operating on an anaplastic carcinoma of the thyroid or a repeat thyroid operation are difficult, as anatomical planes are obscured, and need an expert.

PREPARATION

It is essential that your patient is euthyroid before you start (25.2). Get neck and chest radiographs to determine the narrowing and deviation of the trachea. Perform an indirect laryngoscopy (29.13) to check whether both vocal cords are working: if you damage the recurrent laryngeal nerve on one side, and the other cord was paralyzed pre-operatively, you will be in trouble because paralyzed cords are closed cords (29-15)!

Cross-match 2 units of blood. Place the patient supine with a sandbag between the shoulders, the neck extended with the head held on a rubber ring, and the operating table raised head-up to an angle of 20º.

Drape the head putting two towels below it, and then fold the top one across the chin, thus leaving the neck exposed: in this way, the towels won't fall off, but still allow the anaesthetist access if he needs it. Make sure the suction is working properly.

ANAESTHESIA

It is perfectly possible to perform thyroidectomy under LA; this has the advantage that you can ask the patient to talk and check on the vocal cords as you go along. Learn this technique from an expert.

Otherwise, endotracheal intubation (especially with a long flexible tube) is necessary. If there is respiratory distress, this may be very difficult.

METHOD (GRADE 3.5)

A unilateral multinodular goitre needs only a unilateral thyroid lobectomy; a large bilateral or diffuse goitre will require a subtotal thyroidectomy. For hyperthyroidism, a subtotal thyroidectomy is necessary, aiming to leave behind enough gland not to render the patient hypothyroid afterwards. A confirmed malignant thyroid nodule should have a total thyroid lobectomy on that side; it is controversial whether more than this is required. Since the risks of surgery, and hypocalcaemia and hypothyroidism, are substantial with more radical surgery, this is unlikely to be appropriate. Since you are only likely to know about the histology of the gland after you have operated, the question is whether you need to excise more of the thyroid gland. The risks of doing this almost certainly outweigh the advantages. Suppress further tumour growth with levothyroxine or radio-iodine, if you can (25.6).

INCISION

Mark the position of the incision with a thread held taut against the neck; put this 4cm above the suprasternal notch, or higher if the goitre is very large. Infiltrate along this line with 1:500,000 adrenaline solution to reduce bleeding, and cut through *platysma* which is just under the skin.

Develop the upper skin flap by holding it with tissue forceps or skin hooks, and dissecting it off the subcutaneous layer with either a knife, scissors or the finger.

Keep anterior to the anterior jugular veins (25-2A). If you damage these or their tributaries, tie them. Continue your dissection till you reach the cricoid cartilage: this is important, because if you don't, you will not have enough room to mobilize the upper pole of the thyroid gland. It helps to re-apply the tissue forceps further up as you go along.

Then develop the lower skin flap in the same way. You may find it easier to change to the opposite side of the patient to do this. Continue the dissection down to the suprasternal notch, carefully controlling bleeding vessels as you go; get your assistant to retract the skin edges firmly downwards to let you see clearly.

Now hold the skin flaps open with two self-retaining Joll's retractors if you have them; otherwise use towel clips or simply suture the flaps down at the wound edges to hold the wound open.

Try to identify the midline between the strap muscles of each side; this may be significantly distorted in a unilateral goitre where the trachea is shifted. It does not matter too much if you divide some muscle fibres but the bleeding is reduced if you remain accurately between the strap muscles.

Cut gently down to the thyroid gland along this 'midline' and pull the strap muscles laterally with retractors of Babcock forceps (25-2B). It is important that you cut through all the fine layers including the pre-tracheal fascia which covers the thyroid gland itself, because if you are not in the right plane of dissection at this point, you will encounter much bleeding. Once you are down onto the gland, you can use a Lahey swab to develop this plane.

For very large goitres, where you simply cannot get far enough round laterally, you may have to divide the strap muscles between large straight artery forceps.

Stand on the opposite side of the lobe which you wish to remove.

When you are confident that you are in the right plane below the pre-tracheal fascia, place a swab over the thyroid gland so it does not slip from your hand, and gently insinuate your finger between gland and fascia, pulling that thyroid lobe medially (25-2C). This is easier with large goitres which have stretched the fascia. At this point the middle thyroid veins may get in the way: you can divide and tie them. As you retract the thyroid lobe medially, you can use the Lahey swab gently to push away tissues so that you can identify the crucial inferior thyroid artery. This may be quite small, and runs transversely to the gland as a branch of the thyrocervical trunk, *behind* the carotid sheath. Tease away surrounding fibres from the vessel so that you can pass a fine well-curved forceps behind the crucial inferior thyroid artery.

Try to ensure that you pick up the artery on its own because its relationship with the recurrent laryngeal nerve is *variable but intimate*.

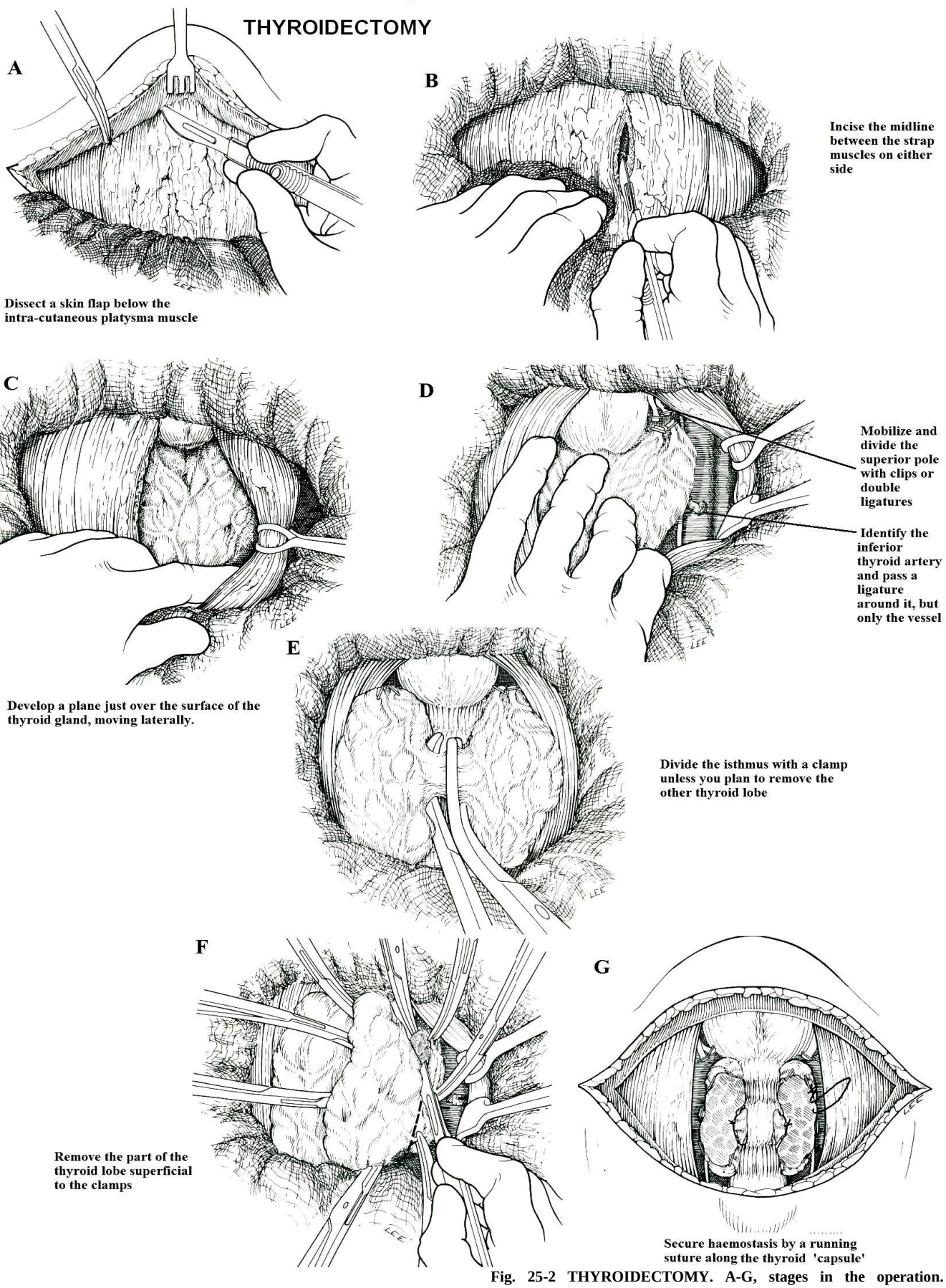

THYROIDECTOMY
A
Dissect a skin flap below the intra-cutaneous platysma muscle
B
Incise the midline between the strap muscles on either side
C
Develop a plane just over the surface of the thyroid gland, moving laterally.
D
Mobilize and divide the superior pole with clips or double ligatures
Identify the inferior thyroid artery and pass a ligature around it, but only the vessel
E
Divide the isthmus with a clamp unless you plan to remove the other thyroid lobe
F
Remove the part of the thyroid lobe superficial to the clamps
G
Secure haemostasis by a running suture along the thyroid 'capsule'

Fig. 25-2 THYROIDECTOMY. A-G, stages in the operation. ***N.B. The parathyroids lie posterior to the bulk of the thyroid gland.*** *After Rob C, Smith R. Atlas of General Surgery. Butterworth 1981 p.706-11 (Figs 5,8-9,11,13-15).*

Pass a 2/0 absorbable ligature mounted on an artery clip, and tie this around the artery; *don't divide it* because the vessel may recanalize and the blood supply of the parathyroid glands may still depend on this later.

You may see the recurrent laryngeal nerve, *but you should probably not go out of your way to look for it, in case, in so doing, you damage it inadvertently!*

Once you have ligated the inferior thyroid artery, the thyroid lobe will become a dusky bluish colour. Now turn your attention to the upper pole; sometimes it is easier to deal with this before the inferior thyroid artery but the vascularity of the gland will still then be undiminished.

Develop the pre-laryngeal space lateral to the thyroid cartilage so that you can pass a curved artery forceps around the branches of the superior thyroid artery and veins to the upper pole.

Ideally you should avoid the external laryngeal nerve which runs behind as division of this will affect the timbre of the voice (25-2D). Put 2 haemostats proximally and one distally, and divide between the latter.

Tie two 0 absorbable ligatures around the most proximal haemostat, release this, and then tie another ligature around the remaining haemostat; in this way you will avoid the ligature slipping and vessels disappearing deep into the neck causing a haematoma which will cause respiratory compromise.

Finally, you can mobilize the lower pole by ligating the inferior thyroid vessels. If the isthmus is not too thick, and you are only removing one lobe, you can insinuate a forceps between it and the trachea and clamp it across (25-2E).

Now, put fine haemostats all around the margins of the mobilized lobe especially where you see veins crossing over the surface, staying well *anterior* to the position of the recurrent laryngeal nerve and parathyroids (25-2F). Remove the excess bulk of the thyroid lobe distal to these fine haemostats with scissors or a knife, having haemostats ready to catch any bleeding points. Aim to leave a remnant 5x1cm (25-2G).

To control bleeding, take a running absorbable suture along the 'capsule' (pre-tracheal fascia) of the thyroid and secure it to the tracheal fascia.

If you are going to perform a bilateral thyroidectomy, you can now change sides and proceed as before on the contralateral side.

When you are satisfied the bleeding is controlled, ask the anaesthetist to make the table level to horizontal, or better, head down to 30º of Trendelenburg: some vessels may then start oozing.

Control these, and when all is dry, insert suction drains through the strap muscles into the thyroid bed, and secure them firmly with sutures on the lateral sides of the neck, *not in the middle where keloids are more likely to form*. If you have divided the strap muscles, plicate and overlap them to reduce the dead space.

Close the investing fascia with a continuous absorbable suture, the subcutaneous layer with interrupted absorbable sutures, and the skin with a subcuticular suture.

DIFFICULTIES WITH THYROIDECTOMY

If there is heavy bleeding, make sure the head is tilted up. Apply swabs soaked in adrenaline solution. Press on the bleeding point(s) for 5mins by the clock. Obtain suction and then carefully expose the bleeding point in order to catch it in a haemostat.
Don't plunge forceps blindly into the wound!
If the bleeding is from the surface of the thyroid gland, hold it firmly in a gauze and expose the inferior thyroid artery as above. Ligation of this will substantially reduce haemorrhage.

If there is a retrosternal extension, you can usually deliver this by gentle traction, with a finger behind the gland. Occasionally, you may need a sterilized spoon to deliver it. Very rarely is it necessary to split the sternum!

If during operation, the temperature, pulse, and blood pressure all rise alarmingly, this is a thyroid 'storm' where excessive thyroid hormone is released into the circulation through manipulation of the gland, especially when operating on a thyrotoxic patient. Speed up IV fluids, add propranolol 5mg IV, hydrocortisone 100mg IV, and carbimazole or propylthiouracil via a nasogastric tube. Stop operating till the situation has normalized. (If you don't have propranolol IV, you can place crushed tablets in the vagina, where they are rapidly absorbed.)

If there is continued oozing at the end of the operation, be careful you don't put in sutures that may compromise the recurrent laryngeal nerve. Use absorbable haemostatic gauze and insert drains. Keep the patient intubated and sedated post-operatively, and re-open the wound the following day when identifying the source of the bleeding will be much easier.

If the goitre is huge and the trachea has been pulled forwards, insert a prophylactic tracheostomy before you close the wound.

If the patient cannot breathe properly after the operation, suspect vocal cord palsy if there is stridor and cyanosis without swelling of the neck. You will need to pass an endotracheal tube rapidly, or else perform a tracheostomy (29.15); the quickest access to the trachea will be through the surgical wound: re-open the midline fascial closure.

If there is stridor and swelling of the neck after the operation, suspect haemorrhage beneath the deep fascia. Open the wound on the ward, and re-open the midline

fascial closure. Scoop out the blood clots: the patient's respiratory distress will be immediately relieved. Then take her back to theatre to try and identify the bleeding points. Secure these, put in new suction drains, and close as before.

If there is tetany after the operation, you may have removed or devascularized the parathyroids. These are four small yellowish-brown glands, responsible for calcium homeostasis, which are at the back of the thyroid (25-2). Treat with 10% calcium gluconate 10ml IV qds, followed if necessary by oral calcium and vitamin D supplements.

N.B. If you have inadvertently removed a parathyroid gland, you can re-implant it, after slicing it into 1mm sections, into the sternomastoid muscle.

If hypothyroidism develops later, characterized by fatigue, weight gain, cold intolerance, menstrual irregularity, gruff voice and bradycardia, treat with levothyroxine 0·1mg od initially. You may need to adjust the dose later, so follow up the patient.

25.8 Other thyroid problems

You may see the following three non-neoplastic diseases of the thyroid. Apart from lymphocytic thyroiditis, they are uncommon, and you may have to do a needle biopsy to distinguish them.

If a goitre is uniform and feels unusually firm and very well defined, but is not particularly tender, suspect autoimmune lymphocytic thyroiditis (Hashimoto's disease, not uncommon). This occurs between 20-70yrs and is usually found in females. Spontaneous resolution is usual but slow. Hypothyroidism often develops, and needs replacement therapy with levothyroxine 0·1-0·2mg od.

If the thyroid becomes woody hard, is fixed to the surrounding tissues, and is either normal-sized or a little enlarged, suspect Riedel's thyroiditis. Inflammation replaces the normal thyroid tissue and adjacent tissues in the neck, and can lead to hoarseness, stridor, and dysphagia. Distinguish this from malignant tumours by aspiration cytology.

If the thyroid becomes inflamed, hot and tender, often with respiratory embarrassment, suspect a thyroid abscess, or HIV-related thyroiditis (6.12) or glandular fever (mononucleosis).

26 Proctology

26.1 Introduction

Few people relish examination of the rectum, whether the patient or the doctor! As a result, many times you may learn of a diagnosis of 'piles' (haemorrhoids) made without any foundation whatsoever! The difficulty of proctology is described as 'the differentiation of the similar amidst the great diversity of the same'.

Indeed, the rectum and anus can be the source of much disability and discomfort. Where HIV disease is common, the anorectal area will feature frequently but pathology will differ whether or not homosexual practice occurs. Acquaint yourself with the particular prevalence of anorectal disease in your area: it may be different to what you are used to!

In the presence of severe leucopenia (absolute neutrophil count <100/μl), *there is a real danger of introducing a bacteraemia by performing an anorectal examination.* If it is essential, administer prophylactic ciprofloxacin and metronidazole.

You should have little difficulty diagnosing anorectal abscesses (6.17), fistulae (26.3), fissures (26.5), warts (26.6), cancer (26.7), prolapse (26.8), haemorrhoids (26.9), pilonidal sinuses (26.10), juvenile polyps, rectal stricture, ulceration or lymphogranuloma venereum (26.11), and imperforate anus (33.6) because all you need is an examining finger and a proctoscope!

But you do need to become familiar with anorectal conditions, and the only way to achieve this is always to examine the anus! Sometimes you will need histological support to confirm a diagnosis, but not always. You may need technological help, though, in finding out the cause of rectal bleeding (26.4).

Remember the common causes of anal pain:
(1) anal fissure or ulcer,
(2) perianal haematoma,
(3) perianal abscess,
(4) excoriated or eczematous perianal skin,
(5) worm infestation,
(6) thrombosed prolapsed haemorrhoids,
(7) coccydynia,
(8) anorectal carcinoma.

Because the anus is always a contaminated area, any surgical wounds near it are prone to become infected but the infection seldom spreads except in the presence of HIV disease or diabetes. The blood supply is normally good so wounds readily heal *if you let them granulate from below*; make sure your nurses understand this. *Only rarely attempt primary suture*, and instead make wide, shallow saucer-like wounds.

Don't let the subcutaneous tissues or the skin edges fall together and unite prematurely, before the depth of the wound has healed. A shallow open wound with trimmed edges heals better than one with much redundant skin and fat.

PHYSIOLOGY. The purpose of the anal musculature is continence. If it fails in this respect, it is a social disaster. Continence is mostly maintained by the external sphincters and the *levator ani*, especially its deep *puborectalis* part, which forms a sling at the anorectal line, in the angle between the anus and the rectum. The tone in the external sphincters is increased by reflex and voluntary contraction. The internal sphincter, which is under autonomic control, is responsible for maintaining anal resting pressure. In painful conditions, both the sphincters are in spasm. The lower part of the anal canal is sensitive enough to discriminate what is in the rectum: nothing, gas, liquid, or solid. Receptors in the smooth muscle of the upper rectum and the voluntary muscle of the pelvic floor alert when the rectum is dilated. Filling of the corpus cavernosa recti (the source of haemorrhoids) makes the anus gas-tight. The rectal mucosa is one cell layer thick, so is easily damaged (unlike the vagina which is 40 cells thick).

EQUIPMENT. A rectal tray containing a proctoscope, gloves, cotton wool, long applicators, short biopsy forceps and the light source. If you are going to pass a sigmoidoscope, you may need a suction tube, long biopsy forceps and a sucker. Have a waste bin near by, and preferably water and towels for washing and cleaning instruments.

PROCTOSCOPE, Gabriel, 64x25mm. This is the standard instrument for examining the rectum. The problem with it is that it needs a separate light source. A simple instrument that circumvents this problem is Dipankar Ray's proctoscope (available from Kolkata, India) which uses a laryngoscope handle and a cone speculum with a groove for the light. You will also find an ordinary Sims' speculum useful for examining the anal canal under GA.
SPECULUM, bivalve, Goligher pattern with detachable third blade. Use this for doing minor rectal operations, such as division of the internal sphincter.
SIGMOIDOSCOPE, Strauss, 330mm, Luer fitting, in case with bellows, cord and standard endoscope bulb complete with biopsy forceps, etc. Keep sigmoidoscopes and proctoscopes in a case so that their various parts don't get lost. This also needs a light source: a pen torch usually fits snugly in the side-access of the light source if this does not work.
N.B. Fibre-optic endoscopes are very much more expensive!
SPONGE HOLDER, for sigmoidoscope, 430mm.
FORCEPS, for biopsy through sigmoidoscope, Officer pattern. These are the most expensive part of the outfit. If necessary, you can use them to remove foreign bodies from the oesophagus, or even from the urethra.
SUCTION TUBE FOR SIGMOIDOSCOPE. You can make this from a piece of ordinary copper tube, 15cm longer than the sigmoidoscope, with a right-angle bend at one end.
BELLOWS, spare for Strauss sigmoidoscope, Luer fitting.
BULBS, endoscope, standard (34.1), small fitting. Endoscope bulbs are very easily blown.
N.B. A pen torch fits in the side tube quite well in case of bulb failure!
BATTERY BOX, for endoscopes, holding D type dry cells. This must be the same voltage as the standard endoscope bulbs, and have a lead which fits the endoscopes.
PROBE, medium-sized, malleable silver.
DIRECTOR, probe-pointed. This has a groove on it. Pass it through a fistula and then cut down on the groove.
PARKS ANAL RETRACTOR. This versatile retractor is almost essential if you want to do any anorectal operations (26-4).
LONE STAR RETRACTOR. This is a simple, but effective, self-retaining retractor virtually essential for good vision in more advanced anorectal surgery (26-17).
COLONOSCOPE, flexible. You may be lucky and have this as well as an upper GI endoscope (13.2). It is more difficult to manipulate, especially past the sigmoid colon, and particularly past the hepatic flexure.

THE ANORECTUM

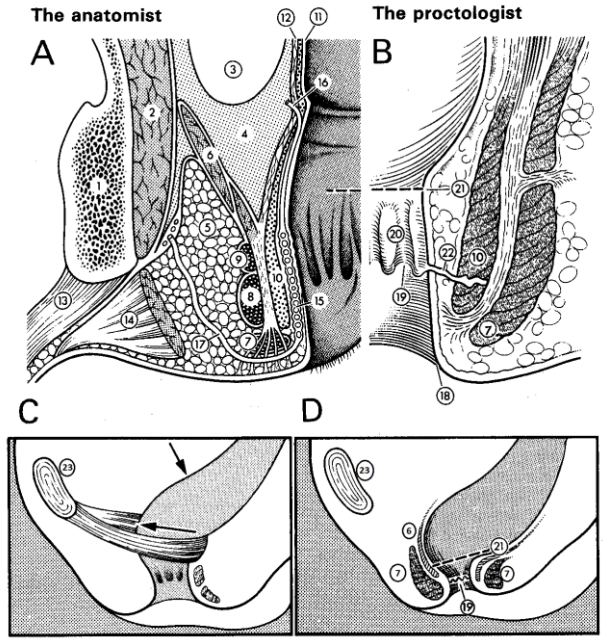


Fig. 26-1 THE ANORECTUM. A, anatomist's (cut coronal) view to show the bony pelvis (1) inside which is *obturator internus* (2). Below the pararectal fossa (3) lies the pelvirectal space (4). This is separated from the ischiorectal fossa (5) by the *levator ani* muscle (6). The external sphincter has three parts: a subcutaneous part (7), a superficial part (8), and a deep part (9). Inside the external sphincter lies the internal sphincter (10). This is continuous with the circular muscle of the bowel (11), outside which is the longitudinal muscle (12). Two other muscles are also shown, *semitendinosus* (13), and *gluteus maximus* (14). The rectal venous plexus (15) is drained by the superior rectal vein (16) and the inferior rectal vein (17).

B, a proctologist sees things more simply and has three reference points. First is the anal verge (18), where the anal and perianal skin join. Second is the dentate (or pectineate) line (19), where the anal columns (20) and sinuses end. Red, loosely attached rectal mucosa lies above this line, and pale, tightly stretched anal lining lies below it. Third is the anorectal line (21), which is the palpable upper border of the complex of anal sphincters. This is something you can easily feel with your examining finger, provided the patient has adequate muscle tone, and has not been anaesthetized or given a relaxant. It is about 2cm further in than the dentate line, and the rectum balloons out above it. Note that the external sphincter (7) comes down a bit lower than the internal (10). The anal canal is c.3-5cm long and extends from the anal verge to the dentate line. The anal glands (22) are an important site of infection, and the origin of fistulae and sinuses. They open into crypts just above the dentate line.

C, view of the rectum to show how the *puborectalis* muscle connected to the symphysis pubis (illustrated divided: 23) pulls the anorectal junction upwards and forwards when it contracts. You can easily feel it doing this when you do a rectal examination. During defecation, it allows the anorectal angle to straighten out. D, relation of the anorectal line to the external sphincter.

N.B. The classical description of the sites around the anus are with the patient in the lithotomy position, according to the clock face: the 12o'clock position being in the midline anteriorly, 3o'clock on the left laterally, 6o'clock in the midline posteriorly, and 9o'clock on the right laterally. This clockface nomenclature is confusing and we don't recommend it.

A, after Gray's Anatomy, Churchill Livingstone, 8.127. B, after MacLeod JH. A Method of Proctology, Harper and Row 1979 Fig. 1.1 with kind permission. C,D, kindly contributed by Brian Hancock.

THE DENTATE (PECTINEATE) AND ANORECTAL LINES ARE IMPORTANT LANDMARKS

PREPARATION. Put a drape over the patient and keep the instruments out of sight. Explain what you are going to do, and that it may be uncomfortable. Be gentle, *don't hurry*, and use warm instruments.

(a) Digital examination of the rectum

Lie the patient on his left side with the buttocks extending well over the edge of the bed (26-2A). Flex the hips fully, but keep the knees at 90° so that they are out of your way. It is convenient to have the right upper hip and knee a little more flexed than the left, and a pillow under the head and between the knees.

Draw the buttocks apart and look at the anal region for skin tags, excoriation, eczema, lumps and the openings of fistulae (26-2B). Feel any abnormalities, such as the tracks or openings of fistulae, or tumours (26-2C).

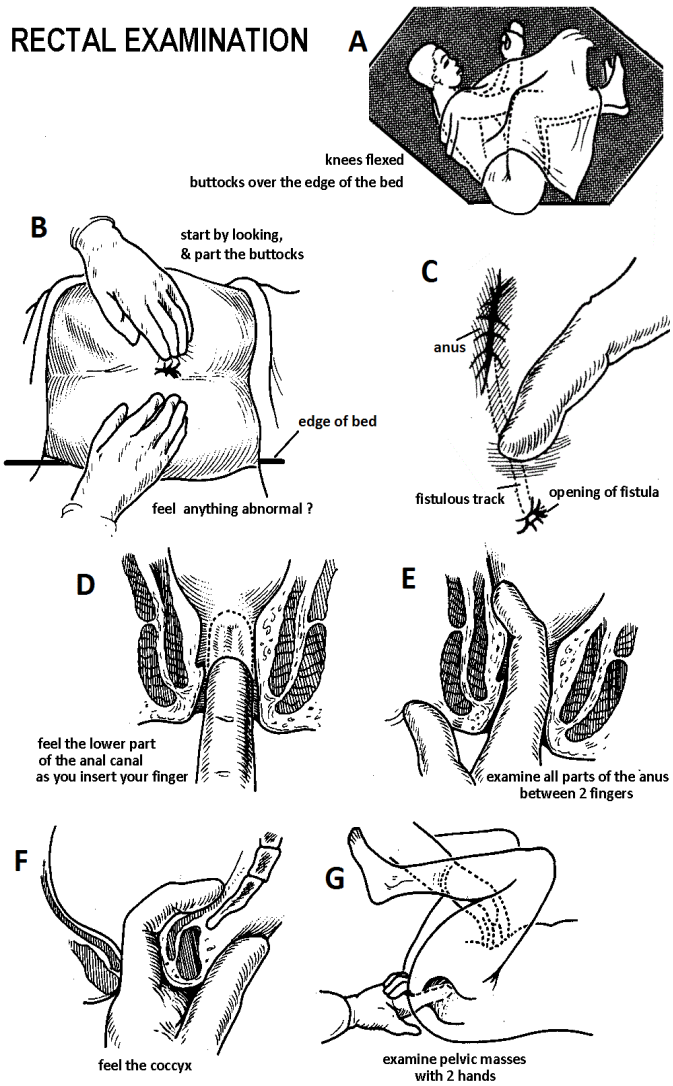


Fig. 26-2 EXAMINING THE RECTUM.
A, have the knees well flexed and the buttocks over the edge of the couch. B, start by looking. C, then feel: you may feel the track of a fistula. D, feel the anal canal as you insert your finger. E, feel all round the anus. F, feel the coccyx. G, if necessary, examine the abdomen between your two hands.
After MacLeod JH. A Method of Proctology, Harper and Row 1979 Fig.2.1,2.8,2.9 with kind permission.

Lubricate the end of your finger well. Insert it so that its larger broad dimension lies in the antero-posterior axis of the anal canal. When you touch the sphincter, it will contract. Wait, give it a few seconds to relax; if it does not, ask the patient to strain as if he were about to pass a stool, as this will also relax the sphincter. Then press firmly and gently in the axis of the anal canal. Keep pressing, until you can feel your finger suddenly slip easily into the anus (26-2D). Note the tone of the sphincter and the presence of stenosis or spasm, which may prevent you doing a rectal examination. In this case, you must administer an anaesthetic and do it: otherwise you might miss an inter-sphincteric abscess (6.17) or carcinoma (26.7).

As you put your finger into the anus, feel for lesions below and above the anorectal line. Then palpate the entire circumference of the anus between your 2 fingers (26-2E).
In a man feel each of the 2 lobes of the prostate separated by a median groove.
In a woman, look to see if she has a rectocoele, feel her cervix and uterus rectally, and feel for swellings in her rectovesical pouch. It may be helpful to feel a mass bimanually through the vagina with one finger of the right hand and the rectum with one of the left hand: *be sure to change gloves before you do this!*
Sweep your finger round and examine the coccyx between two fingers (26-2F).
Finally, if you suspect an intraperitoneal mass, a bimanual recto-abdominal examination will be useful (26-2G).

(b) Proctoscopy (GRADE 1.1). Examine the anus with your finger first. Lubricate the proctoscope and push it firmly with its introducer in place in the direction of the umbilicus. Remove the introducer. Examine the lining of the anal canal as you withdraw it slowly, looking for fissures, polyps, ulcers, or haemorrhoids as you do so.

(c) Sigmoidoscopy (GRADE 1.1)
Perform a sigmoidoscopy just after normal defecation, or after an enema. There is no need for a GA, unless it is too painful for the patient, *e.g.* for carcinoma.
N.B. Always do a digital examination first.

Ask the patient to breathe in and out while you gently insert the sigmoidoscope, lubricated and warmed with its introducer in place. You will feel the resistance of the anal sphincter suddenly diminish (26-3B) as it enters the rectal ampulla. Remove the introducer.

Watching where you are pushing the sigmoidoscope, turn it 90º posteriorly (26-3C), as you gently manipulate it past the mucosal valves of the rectum. As you advance the sigmoidoscope, gently pump in enough air by squeezing the bellows to distend the lumen in front of it. *Don't blow the sigmoid up with too much air*, or the patient will feel urgency and cramps.

The first 12-15cm, as far as the recto-sigmoid junction, is usually easy. However, it is easy to miss a lesion in the rectal ampulla. You will then see the smooth rectal mucosa giving way to the concentric *rugae* of the sigmoid colon. At this point the bowel passes over the sacral promontory, and may turn in any direction. Proceed anteriorly and to the left. You should be able to reach 25-30cm, but *don't force the passage of the instrument!* Be sure you can distend the bowel with air, and see where you are going before you push the sigmoidoscope further in.

If you are clumsy, you can perforate the bowel, so: (1) *Never push in a sigmoidoscope further, if you cannot see the lumen in front of it*. Follow the lumen at all times. (2) *Never force it*. If there is a pocket or a blind area in the way, withdraw it a little, and then advance it again.

If your view is obscured by faeces, remove them with cotton wool on a swab holder, or if this fails, withdraw the sigmoidoscope, clean it and start again. If the stool is very loose or there is copious bleeding or mucus, make sure you have a good suction available.

Concentrate on getting the sigmoidoscope as far up as you can; note the presence of lesions by their position (in cm) from the anus, and review them as you withdraw the sigmoidoscope. Rotate the sigmoidoscope, as you withdraw it, so that you inspect every part of the mucosa. Be careful to examine the posterior wall of the rectal ampulla. This lies at 90º to the anal canal, and you can easily miss it.

N.B. You can use the sigmoidoscope to deflate a sigmoid volvulus (12.4).

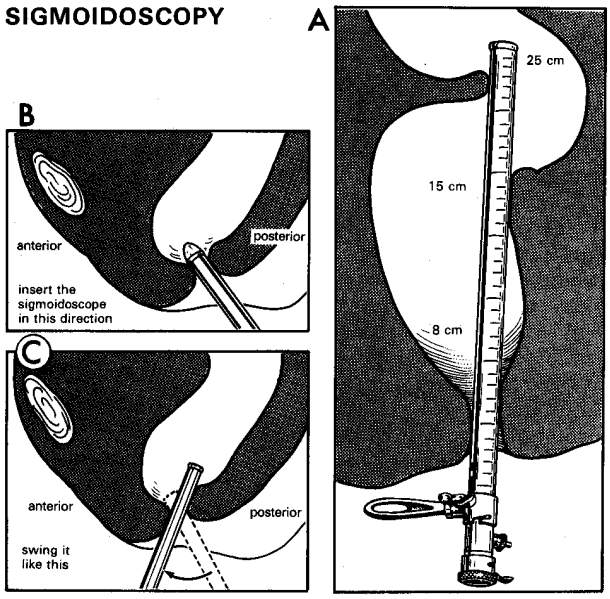


Fig. 26-3 PASSING A SIGMOIDOSCOPE.
A, enables you to inspect the last 25cm of a patient's colon. With your finger you can only feel the most distal 8cm. B, introduce the sigmoidoscope, pointing it towards the umbilicus, and when it is through the anal sphincter (C) swing it backwards, and to the left.
After MacLeod JH, A Method of Proctology. Harper and Row 1979 Figs 2-12-14 with kind permission.

If you think you may have perforated the rectum or colon (when you see loops of bowel at the end of your sigmoidoscope) or there are signs of peritonitis after a sigmoidoscopy, start an IV infusion of saline, commence gentamicin and metronidazole, and get an erect chest radiograph to look for free sub-diaphragmatic air.

If there was a clean bowel with good bowel preparation before the sigmoidoscopy, particularly if the perforation has occurred in the distal retroperitoneal section of the rectum (<12-15cm from the anal verge) and symptoms and signs settle within 12h, continue this conservative treatment for a further 48h. Otherwise, don't hesitate to perform a laparotomy and try to close the perforation with interrupted sutures; you may add a defunctioning stoma if there is significant soiling.

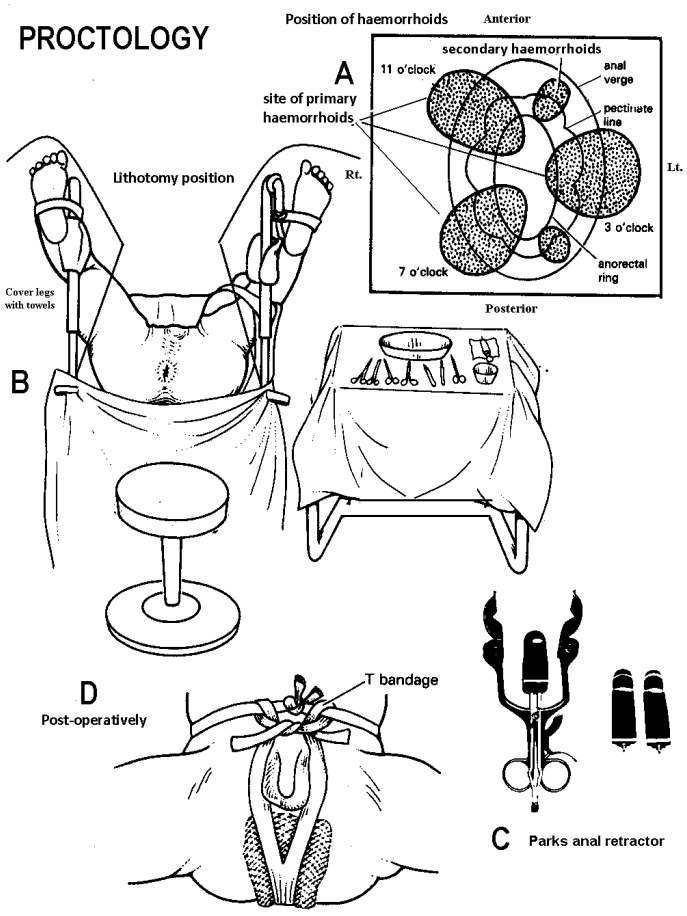


Fig. 26-4 PROCTOLOGICAL SURGERY.
A, diagram for recording abnormalities around the anus. This has 3 lines, an inner one for the anorectal line, a middle wavy one for the dentate line, and an outer one for the anal margin (26-1)**. Record your findings in relation to these 3 lines. Note the sites of the 3 primary haemorrhoids and the common sites of 2 accessory ones are shown with the classical 'o'clock' positions, corresponding to left and right, anterior and posterior. B, arrangements for operating on a patient's anus. You must also have a light on a stand which will direct its beam horizontally into the wound. C, the Parks anal retractor. D, a T-bandage tied up after the operation. *Use this only for 24h.***
After Goligher JC. Surgery of the anus, rectum and colon. Ballière Tindall 4th ed, 1980. Figs 64,65 with kind permission.

(d) Pre-operative care for anal operations
Perform a proctoscopy or sigmoidoscopy before all anal operations to exclude coexisting rectal lesions. For this to be possible, the bowel must be empty, so use an enema or a glycerine suppository pre-operatively.

(e) Post-operative care after anal operations
(1) BATHING is more effective than irrigation. Encourage soaking in a warm bath; you can add some antiseptic to the water if you are not sure about the cleanliness of the tub!

(2) DRESSINGS are much less important. Soiled dressings will perpetuate sepsis, so encourage frequent bathing or douching.

(3) BOWEL ROUTINE. Treat with a laxative od from the day of the operation for a maximum of 2wks. Encourage mobilization from the first day. *Don't use an enema* as introduction of the funnel will be painful and may disrupt the wound. If there is no stool passed by the 3rd day, gently insert a glycerine suppository.

26.2 Anorectal pathology in HIV disease

Almost 25% of HIV+ve patients will have some kind of anorectal lesion. These are particularly numerous in homosexuals, especially in those who practise anal sexual intercourse, but they adopt a rather different pattern. Their severity relates to the CD4 level.

The healing of anorectal lesions in HIV+ve patients is particularly poor, especially if their CD4 levels are <200/μl and they are therefore particularly difficult to treat; moreover the aetiology of many of the HIV-related anal conditions is not easily diagnosed. These conditions will probably not heal properly, and almost certainly recur.

It is exceptionally risky to perform incisional anorectal surgery on HIV+ve patients, unless they are receiving antiretroviral therapy (5.8), and so you should use different, more conservative methods:
For fistulae (26.3), use a seton. *Don't lay open fistulae*.
For fissures (26.5), *avoid a sphincterotomy*.
For haemorrhoids (26.9), use sclerosant injections with oily phenol. *Avoid a haemorrhoidectomy*.
For anal skin tags, and **perianal haematoma**, adopt a conservative approach.
For frank daily uncontrollable faecal incontinence, consider a defunctioning colostomy (11.6).

Some anorectal lesions are typical, almost pathognomonic, of HIV disease, where they are often more extensive and complex, whilst others are found in HIV-ve patients.

(a) Idiopathic anorectal ulcer (26.5). This starts as a mucosal laceration within the anal canal, and gives rise to symptoms identical to the classical anal fissure. It is virtually pathognomonic of HIV disease. However, no anal skin lesion is visible on gentle parting of the buttocks, because the lesion is internal, usually just proximal to the dentate line. Furthermore there is rarely anal sphincter spasm, and often diarrhoea rather than constipation. Pain is persistent, usually associated with some intermittent bleeding *per rectum*, particularly after defecation.

The mucosal defect then deepens and becomes palpable as an ulcer with smooth benign-feeling edges. As this ulcer deepens further, it may penetrate into the vagina or appear as a large fistula externally. Often pus collects in the ulcer crater and discharges through the anus. No specific agent is often implicated in this lesion, although in some cases *cytomegalovirus* and *herpes simplex* virus are found. The ulcer edge is smooth and round, unlike the syphilitic ulcer which is irregular.

Treatment with glyceryl trinitrate is unsuccessful, because there is no anal spasm. *Sphincterotomy, or worse, anal stretch, is a disaster, resulting in faecal incontinence.* If there is pus, use nalidixic acid, or ciprofloxacin with metronidazole.

N.B. A shallow triangular ulcer posteriorly placed, without spasm or bleeding, may be due to syphilis (26.5). *Check also for gonococcal infection especially if there has been ano-receptive sexual intercourse* (26.11).

(b) Superficial breakdown of perianal skin with excoriation often occurs with chronic diarrhoea. There is often also excoriation in the intergluteal cleft, which may be due to excessive sweating. Vesicular excoriation is due to *herpes simplex*. Otherwise the causative agents are usually *candida* and/or *trichuris* (whipworm). It is vital to examine patients carefully to exclude fistulae and abscesses.

Use nystatin ointment bd, or miconazole 2% cream bd for 2wks or itraconazole 200mg bd for 1wk and mebendazole 2mg/kg bd for 3 days. Zinc oxide or manganese sulphate paste is better than mercurochrome. You must try to control diarrhoea and sweating. These patients are usually grossly cachectic, so encourage high-protein, high-calorie diets.

N.B. If cortisone preparations have been used, the perianal skin becomes paper-thin, and atrophied and readily damaged.

(c) Sinuses and fistulae (26.3) in HIV+ve patients are often complex and multiple. They are frequently high or intersphincteric (intermediate). Many fistulae arise from sepsis, but some as a result of extension of the idiopathic anal ulcer described above. In these cases, the fistula is wide and may readily admit the examining finger. Fistulation can occur to the outside skin, but also to the vagina or bladder.

This is found in both adults and small children, where the history of whether the fistula is congenital or acquired is diagnostic.

Because these fistulae are complex, multiple and often high or intersphincteric, they cannot be simply laid open. In fact, even for low superficial fistulae, the laying open may result in non-healing perianal wounds, especially if CD4 counts are <200/μl. Treatment by passing one or more setons (26.3) is not only simple, but very effective. You need to be patient, however: these wounds may take 8-12wks or even more to heal.

(d) Proctitis (26.11). Just as in colitis, the rectum may be affected by a severe inflammatory process: *cytomegalovirus, herpes simplex, chlamydia* or enteropathic *E. coli* may be the cause. You will have to treat blind with broad-spectrum antibiotics, as it is unlikely you will have the resources to make a specific diagnosis. *Avoid steroids and sulfasalazine*, unless you can confirm ulcerative or Crohn's colitis.

(e) Anal and perianal warts (26.6) are often very extensive and may coexist on the urethra and external genitalia, and even elsewhere on the body; their excision or diathermy ablation surprisingly results in rapid wound healing, presumably due to an epithelial growth factor in the causative *papilloma* virus, despite other types of anorectal surgery having poor healing in HIV+ve patients. Contact tracing in the developing world is a pipe-dream, and therefore recurrence by re-infection is frequent. Moreover, if you don't remove all condylomata, including penoscrotal ones, and they can extend far up in the anal canal, they quickly re-establish themselves. They may become infected and ulcerated, and if chronic, develop into squamous carcinoma.

They may occur in children through cross-infection by sleeping in the same bed, and don't necessarily imply sexual abuse, although this should always be kept in mind and investigated.

If the warts are extremely voluminous (then grandiosely known as Buschke-Loewenstein tumours), you may remove them in staged procedures. Always use lidocaine with adrenaline infiltration, because they can bleed heavily. There is a risk of anal stenosis if you remove crops of warts right up to the anal verge, or even inside the anal canal. In this case, get the patient to use a plastic anal dilator bd for 6wks. Make sure he takes baths post-operatively; a shower is not really adequate.

(f) Squamous carcinoma (26.7) may be the result of neoplastic change seen in anal intra-epithelial neoplasia (AIN), or be the result of chronic infestation by condylomata, especially if florid, or arise *de novo*. There appears to be much greater risk of developing malignancy if ano-receptive sexual intercourse is carried out.

You must establish a histological diagnosis; it is rarely possible to perform a local excision without causing

stenosis, or resulting in inadequate tumour excision. This means that an abdominoperineal resection will be necessary (12.11), which is a formidable undertaking in a patient with HIV disease.

Lymphoma and Kaposi sarcoma may also be found at the anus, but you will only make this diagnosis by biopsy. Very rarely, a malignant melanoma is found at the anus: it looks like a thrombosed haemorrhoid.

(g) Rectal prolapse (26.8) is often the result of a very lax anal sphincter. Although this may occur in patients with chronic diarrhoea or neuropathy, it is much more common in homosexuals who practise invasive rectal procedures for pleasure.

Local perineal operations don't correct the essential problem of laxity; you can amputate redundant prolapsing full-thickness rectum but you will need to be careful that you restore bowel continuity.

Doing a laparotomy to lift up the rectosigmoid is an alternative: you should avoid using any foreign implant material for fixation, and *do this only if the patient is getting ARV treatment*.

26.3 Anorectal sinuses and fistulae

Anorectal abscesses (6.17), sinuses and fistulae are usually part of the same disease process. An abscess is the acute phase, and a sinus or fistula the chronic results. Both sinuses and fistulae are tracks lined by granulation tissue, which open on to the skin near the anus. The difference between them is that a sinus has no internal opening, whereas a fistula opens into the anal canal, or occasionally into the rectum. Usually, there is only one internal opening, but there may be several external ones. These can be either insignificant little holes, or prominent little nodules of granulation tissue, which heal over temporarily. With HIV disease, they may be complex, multiple, and chronic.

Likewise, if tubercular in origin (up to 15% in India), they may be complex, multiple and chronic, and often, but not always, associated with HIV disease. The cause may be *mycobacterium tuberculosis* or *mycobacterium ulcerans*.

Occasionally, the cause may be Crohn's disease where the appearance is very similar to tuberculosis.

Other infections may also give rise to fistulae: actinomycosis, gonococcus, *chlamydia*, schistosomiasis, mycetoma and amoebiasis.

Another inflammatory process which may give rise to fistulae is so-called hidradenitis suppurativa (Verneuil's disease) or pyoderma fistulans sinifica (fox-den's disease: 34.9). These result in quite marked skin thickening and multiple skin bridges, but are essentially superficial skin problems, and are related to smoking.

Typically, a fistula starts with a papule, abscess, nodule, or ulcer, which either bursts and fails to heal, or is not drained properly, after which there is a chronic painless discharge which soils the clothes. The fistula is only painful when it becomes temporarily blocked, when pus builds up inside it.

Fistulae can take any of the paths shown (26-5); they can be subcutaneous (common), low anal, high anal, or intermuscular (rare).

Rather wider fistulae exist as extensions of the idiopathic anal ulcer of HIV disease (26.2): these are usually superficial and have a significant absence of granulation tissue.

A fistula seldom heals spontaneously, and almost always needs surgery. The options are passing a seton, laying open the fistula track (fistulotomy), or fashioning a defunctioning colostomy. The aim of surgery is to obtain a permanent cure while preserving sphincter function; you therefore must know the relationship of the fistula track to the sphincters, and cause minimal damage to them.

Fistulae which have external openings anterior to the anus enter directly into it by the shortest path. Posterior fistulae usually curve round, so that they enter the anus posteriorly in the midline (Goodsall's rule: 26-6I). In doing so, they follow a horseshoe path, and are often bilateral, one side communicating with the other. There are exceptions, and very superficial fistulae behind the line may occasionally track directly into the anus. The track of a horseshoe fistula hugs the *puborectalis* part (26-1D) of the *levator ani* muscle, as it forms a sling round the sides and back of the anorectal junction, external to the external sphincter. Fortunately, the internal opening of such a horseshoe fistula is usually at the dentate line, although the fistula itself may go much deeper.

PERIANAL ABSCESSES, SINUSES AND FISTULAE ARE NOT HELPED BY ANTIBIOTICS!

(a) Examination. Prepare for light GA or ketamine. (Spinal anaesthesia or using relaxants is unhelpful because you will not then readily feel the anorectal ring.) Before you start, warn that you are going to examine under anaesthesia to try to find where the fistula runs. If you use ketamine, be sure to put the legs up on lithotomy poles first before you start anaesthesia, as muscle rigidity may prevent you doing so afterwards!

Introduce the Parks anal retractor well lubricated with jelly.

If the opening is <5cm from the anus, the fistula is perianal; if it is >5cm away, it is probably high. Multiple openings suggest a horseshoe fistula. Record the position of all external openings carefully on a copy of the diagram (26-4A).

Feel for the thickened track which runs from the external opening(s) towards the anus. If a fistula is superficial, you can usually feel its firm, fibrous track quite easily. As you press it, pus may exude from the external opening.

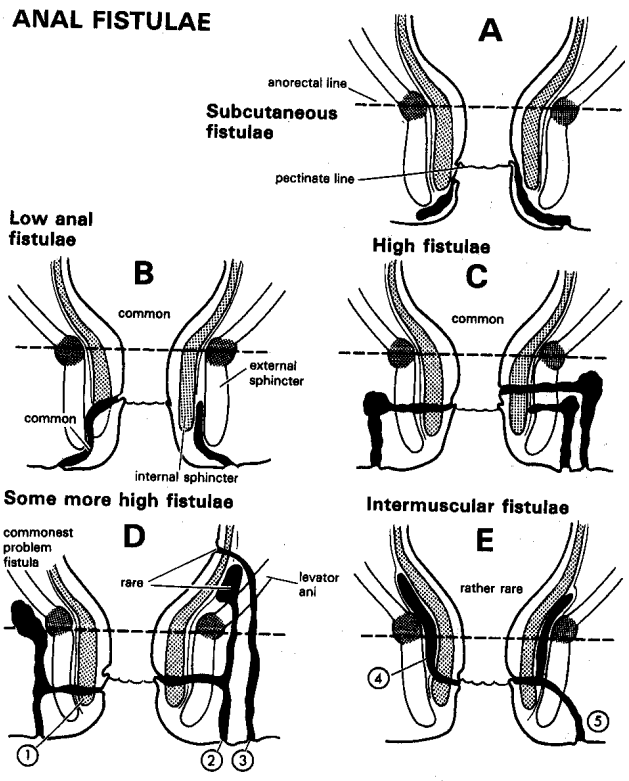


Fig. 26-5 PATTERNS OF FISTULAE.
A, 2 subcutaneous fistulae, 1 opening at the dentate line, and 1 just below it. B, two low anal fistulae. C, several high ones. D, some more high fistulae. Fistula (1) is the commonest high fistula; it goes high towards the *levator ani*, but does not penetrate it. The high extension is often missed, but it must be explored and laid open. Fistulae (2) and (3) penetrate the *levator ani*. E, high intermuscular fistulae (rare) may exist alone (4), or be an extension of a low anal fistula (5).
After Goligher JC. Surgery of the anus, rectum and colon. Ballière Tindall 4th ed 1980 Figs 121-6 with kind permission.

Put a finger into the anus and try to feel the internal opening: you may be able to feel an induration at its internal end. Feel the entire circumference of the rectum, as far as your finger can reach. Determine particularly where the fistula might be in relation to the anorectal ring and the dentate line. Try to feel the track between your two fingers. Does it appear to come to an end low down, or high up in the anus? If you feel induration at the level of the *puborectalis* or above (rare), there is a complex high fistula.

Examine the anal canal with a proctoscope. You may be able to see the internal opening of the fistula, usually posteriorly in the midline on the dentate line.

Insert the proctoscope as far as it will go, withdraw the introducer, and then gradually withdraw the instrument itself. As soon as its end becomes obstructed and closed by the anorectal ring, stop. If you can still see the opening of the fistula, it is safely below the critical level of the anorectal ring.

(b) Probing. *Don't do this until you have finished your initial inspection*. Decide where a track is probably going to go before you start probing. Pass the probe as far as possible towards the anal canal, and feel for its end in the anus. It may pass through into the lumen, or it may stop before getting there. If the fistula is superficial, it will pass horizontally; if it is deep, the probe will pass almost vertically, parallel to the anus.

CAUTION!
(1) If the probe passes vertically, and not towards the mid anal canal (even though there is an opening there), it is probably a high complex fistula or a deep sinus.
(2) Only pass a probe into the rectum through a fistulous track: *don't force it through normal tissues*.

In 50% of cases, you will find the opening easily; in the other 50%, it will be present but tiny. A probe may show it, but if it does not, inject methylene blue (or similar dye) into the external opening, and look for this flowing into the anus: finding the internal opening is the key to all fistula operations! You can add hydrogen peroxide to the dye: its bubbling froth will help show the opening more easily.

(c) Passing a seton (GRADE 2.2) is one of the oldest operations in history, first described by the Indian master surgeon Sushruta c.6000BC. Tie a thread to the probe and withdraw it through the fistula track, release the probe and tie the thread loosely. By slow, progressive inflammatory response, allowing simultaneous drainage, fibrous healing from deep to superficial parts occurs. This method does not divide sphincters and so preserves their function, and so can be used for low or high fistulae. Furthermore, as there is no wound, it is ideal in HIV+ve patients. It is also delightfully simple! The disadvantages are varying degrees of discomfort, and prolonged treatment needing usually 8-12wks or more.

You can use any non-absorbable thread such as *ethibond*, though silk will stimulate more of an inflammatory reaction; corrosive 'soaps' applied to thread such as the latex of *Euphorbia neriifola* or solution of ash of *Achyranthes aspera* increase the efficacy but may result in excessive inflammation and surrounding cellulitis. A nylon suture is sharp and painful, and not well tolerated. For best results, replace the thread weekly by tying a new one to the old one and pulling it through the track, and tying it loosely. Remove it when the distal hole is almost completely closed.

If the fistula is complex or indurated, take a biopsy for histology.

(d) Laying open (deroofing) the fistula (fistulotomy) (GRADE 2.2)
You should now know where the fistula runs. Only open a simple low fistula superficial to the dentate line if there is no HIV disease. *Don't open high or complex fistulae*; you may render a patient permanently incontinent. *If there is untreated HIV disease, the wounds may never heal.*

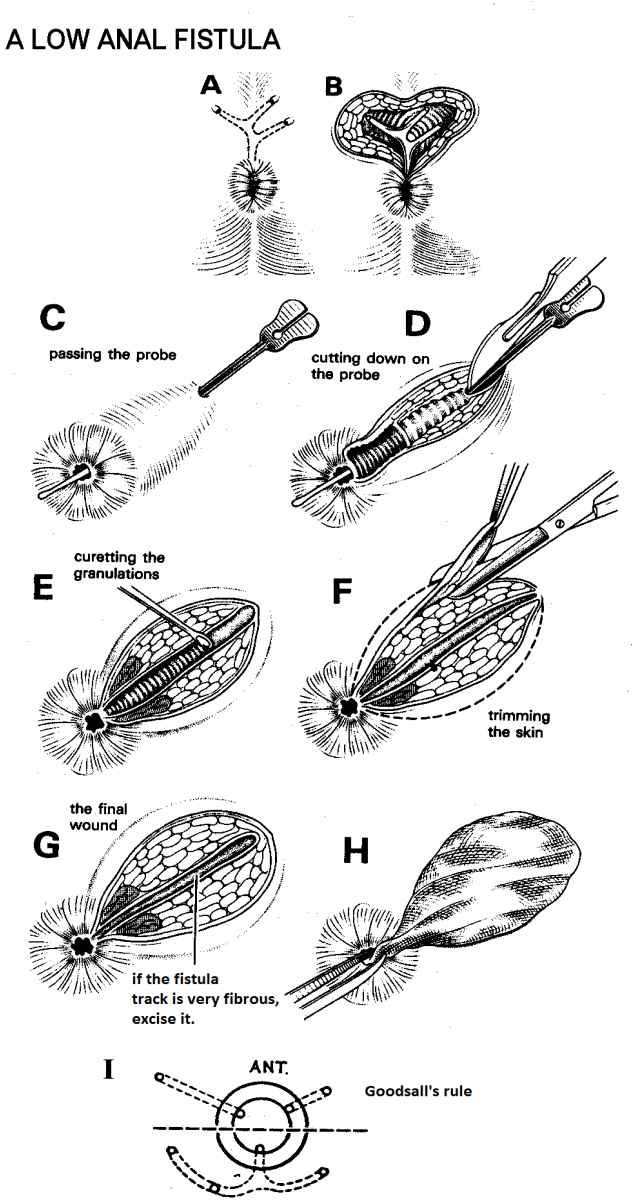


Fig. 26-6 LOW ANAL FISTULA can have several tracks, A,B, or only one. C, pass a probe-pointed director along the track from the external to the internal opening, and out through the anus. D, cut down on the director. E, scrape away the granulation tissue with a sharp spoon. F, trim the edges of the wound. G, final pear-shaped guttered wound. If there is much fibrous tissue round the track, excise it. H, pack the wound with gauze. I, Goodsall's rule: in 60% of cases, fistulae anterior to the anus usually pass directly into it; fistulae posterior to it curve round it to enter in the midline.
After Goligher JC, Surgery of the anus, rectum and colon. Ballière Tindall 4th ed 1980 Figs 139-40 with kind permission.

Pass a probe or director through the track, from the external opening towards the anal canal, either completely through to its lumen, or as far as it will go. It may enter the anal canal, or it may stop before doing so.

Confirm that the probe enters the anus superficial to the dentate line, cut down through the skin on all structures superficial to it, and lay the track open.

If you are using a director (26-6C,D), cut down onto its groove through the skin. Look at the purplish track of the opened fistula. If there is no such track, you have probably opened up a false passage. Look carefully for any side openings, and feel among the fatty tissue for nodules of induration, which might be branches of the fistula. As a general rule, all fistulous tracks communicate with one another. Using a sharp spoon, curette the tracks, so as to leave only healthy tissue, and trim away any overhanging skin.

Alternatively, make a narrow pear-shaped incision to include both the internal and external openings. Excise both of them, and the track of tissue that still clings to the probe.

Control bleeding with diathermy, or tie off bleeding vessels with 2/0 absorbable suture. Bevel the skin edges by making an inclined cut, so as to leave a conical or pear-shaped concave raw area. Be sure that there will be no pockets or overhanging edges when muscle tone returns.

Always send tissue for histology if possible to exclude tuberculosis or other pathology.

(e) Post-operative care. Make sure you keep the wounds clean: dressings may simply retain sepsis, or worse, stool and urine which will secondarily cause soiling. A douche is essential and bidets are very useful; *there is no advantage in using special solutions*: soap and warm water are all that are required. *Don't soak the perineum for a long time:* this will cause maceration. You may have to insist, though, on this tds or qds. For complex fistulae, it is wise to re-examine the fistulae after 6wks under GA to see if they are healing well and no new fistulae have formed. Use a laxative if constipation ensues.

(f) Difficulties with anal fistulae
If the probe enters the anus deep to the dentate line, pass a seton. (*Don't cut deep to the dentate line, or you will cut too much sphincter.*)

If the probe does not enter the anal canal, there is a sinus. Lay it open in the same way, but without opening into the anus.

If you find any other sinuses or fistulae, pass setons. *Don't try to lay open complex fistulae* (26-8).

If a fistula passes forwards from the anus, it may be a URETHRAL FISTULA (27.11), or originate in Bartholin's glands (23.5).

If a fistula is posterior, *don't confuse it with a PILONIDAL SINUS* (26.10).

If the external opening is far from the anus, look out for a long curved fistula, or a high one. Its thickened track will usually show you its course and destination. Probe it; you will probably need dye and hydrogen peroxide to show its internal opening.

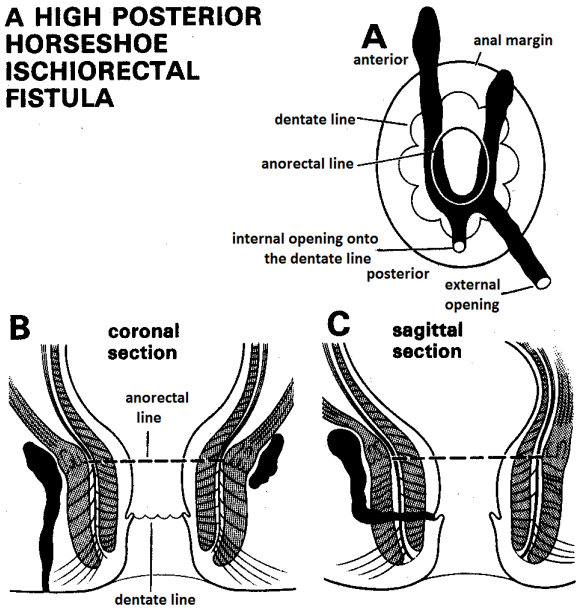


Fig. 26-7 HIGH POSTERIOR HORSESHOE FISTULA (I).
A, fistula shown on a standard diagram of the anus. B, coronal section. C, sagittal section. See also 26-8.
After Goligher JC Surgery of the anus, rectum and colon. Ballière Tindall 4th ed 1980 Fig 128 with kind permission.

If there is hydradenitis suppurativa, the infected sweat glands will need deroofing (34.9).

If haemorrhoids are also present, inject them with oily phenol and defer treatment of the fistula for at least 6wks.

If there is a recurrent discharge from the track, the wound has healed over externally, without healing from below. It will not heal with antibiotics. Consider the possibility of tuberculosis. Pass a seton.

If there is gross faecal incontinence, especially with HIV disease, counsel appropriately and fashion a defunctioning sigmoid loop colostomy (11.6). The wounds may heal in time, but *don't close the colostomy* unless you can start antiretroviral therapy and the CD4 count improves.

If there are multiple fistulae, consider HIV disease with or without tuberculosis, lymphogranuloma venereum (26.11), colloid anal carcinoma, or Crohn's disease. Check the HIV status and take a biopsy before starting treatment, though using setons will not cause harm and may effect a cure.

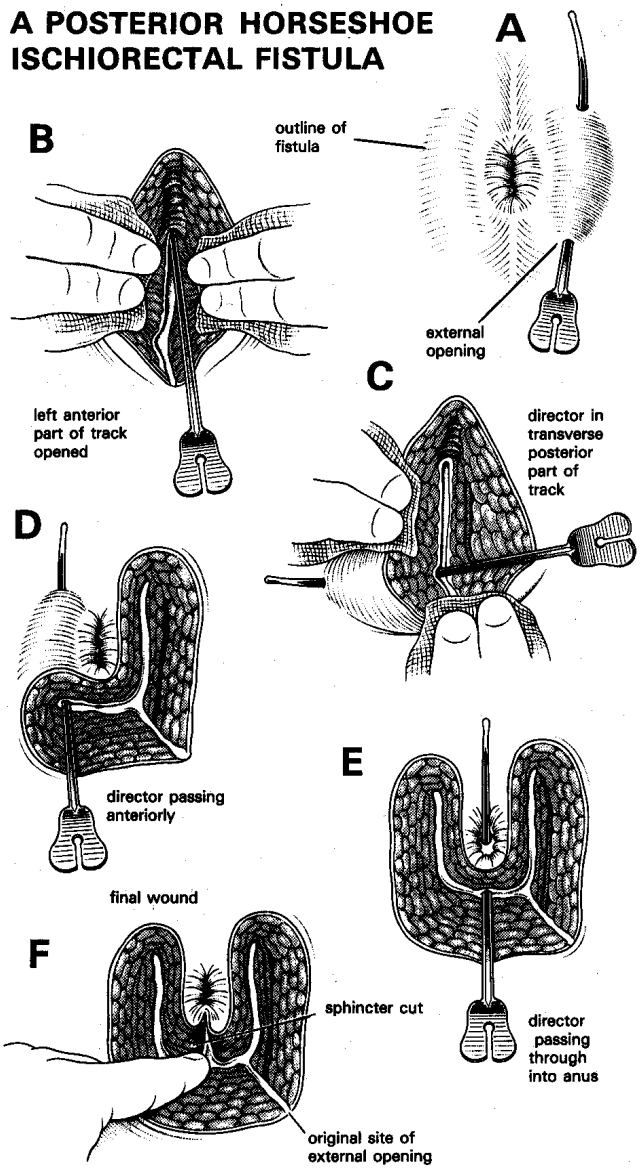


Fig. 26-8 HIGH POSTERIOR HORSESHOE FISTULA (II).
This is the same fistula (26-7) laid open for demonstration purposes. A, pass the director forwards. B, cut down on the track on the left side. C, pass the director along the posterior part of the track towards the right. D, expose the track on the right side. E, pass the director forwards through the posterior communication into the patient's anus. F, final horseshoe-shaped wound, with part of the sphincter divided. *N.B.* Passing a seton is simpler and causes less morbidity.
After Goligher JC, Surgery of the anus, rectum and colon. Ballière Tindall 4th ed 1980. Fig 147 with kind permission.

26.4 Rectal bleeding (Haematochezia)

Bleeding from the rectum may be sudden and very alarming, but is usually not very copious. It often stops and starts again, with the result that a patient may not seek attention until he is profoundly anaemic. You should try to distinguish fresh rectal bleeding from blood mixed with the faeces. The latter may actually be bloody diarrhoea (dysentery) or just traces of blood that come out with defecation, sometimes associated with change in bowel habit. Fresh rectal bleeding may be spots on toilet paper or moderate volumes.

Obviously, patients who defecate in pit latrines are unable to describe much about the blood they pass, and therefore you have the obligation to find out as best you can. There are clues, and you must try to distinguish lower intestinal from upper gastrointestinal bleeding: the latter is usually dark purplish and sticky with a sweet pungent odour (melaena) or, if the bleeding is more rapid, comes out as dark red burgundy-coloured blood. This bleeding is not necessarily from the stomach or duodenum (13.4) although that is the most common site, and may come from the small bowel and rarely from the large bowel. It is often severe, is usually more serious than it looks, and frequently threatens the life.

There are clues:
Fresh blood separate from stool:
(small amount with pain): anal fissure
(moderate amount, intermittently, without pain): haemorrhoids
(at monthly intervals): endometriosis

Fresh blood mixed with stool:
(loose motions): proctitis, colitis, dysentery (including typhoid, 14.3), necrotizing enteritis (14.4), amoebiasis (14.5), or schistosomiasis,
(no change in bowel habit): polyp
(change in bowel habit): carcinoma, diverticular disease
(with mucus): intussusception, juvenile polyp, chlamydia, gonorrhoea, rectal prolapse

The major mistakes are:
(1) To misjudge the severity of the bleeding.
(2) To fail to use your finger, a proctoscope and a sigmoidoscope, to label the cause as 'haemorrhoids' without a proper examination, to fail to investigate fully, and so to miss the diagnosis.
(3) To confuse iron-black stools (from ingestion of iron supplements) with blood.
(4) To miss the more treatable diseases, such as amoebiasis, as the following case shows.

POUL (53yrs) had passed several bloody stools since the morning, but had no other gastrointestinal symptoms. He was neither anaemic nor hypotensive, but during the next few days he continued to bleed, and the haematocrit fell to 23%. Sigmoidoscopy showed friable, oedematous, reddish-yellow areas in the rectum, but no obvious ulcers. A smear from the rectal mucosa showed amoebae. Metronidazole cured him dramatically.
LESSONS (1) Amoebiasis is readily treatable, if you diagnose it. (2) A severe bleed in the absence of previous symptoms of amoebiasis is unusual.

(a) Examination
Assess the degree of hypovolaemia and anaemia. Does sitting up in bed cause light-headedness, or exercise produce breathlessness? Do a general abdominal examination.

Examine the rectum with your finger *and* a proctoscope, and *don't forget to look at the stool*.

CAUTION! Never forget to perform a proctoscopy and/or sigmoidoscopy in an adult presenting with rectal bleeding.

If attempted rectal examination is exquisitely tender, stop and do it under anaesthesia.

If you palpate a polyp, try to pull it down through the anus, tie the stalk, and cut it off. *If you cut the stalk without tying it first, it may bleed massively*.

If you feel a craggy mass or stricture, examine under anaesthesia and take a biopsy (26.7).

If you see a prolapsed lesion, distinguish between haemorrhoids (26.9), rectal prolapse (26.8) and intussusception (12.7). Try to reduce it.
(The intussusception needs a laparotomy.)

ON PROCTOSCOPY,
If you find haemorrhoids (26.9), inject oily phenol.

If you see inflamed mucosa, take a biopsy, a smear, and examine the stools. Enquire about the use of herbal enemas. *Only use steroids and sulfasalazine if you can confirm inflammatory ulcerative or Crohn's colitis.*

If you see a polyp, try to hold it with a biopsy forceps and pull it down so you can tie its stalk. If you can't do this, try to diathermy it taking care that you don't touch the metal sides of the proctoscope. If this is not possible, twist it by 360º and hold it twisted for 2mins so that it will thrombose. *Don't pull it off: it may bleed massively.*

ON SIGMOIDOSCOPY,
If you see blood coming from further proximal in the intestine, investigate for a peptic ulcer (13.4), and if that is unhelpful, by barium enema or colonoscopy. If you can't do this, you may have to try to find out where it is coming from at laparotomy.

If bleeding continues from the rectum, and you are not sure why, you will have to decide:
(1) if you are going to operate,
(2) when, and
(3) what you are going to do when you get inside.

Try to distinguish between upper gastrointestinal and colorectal bleeding. In most areas, the commonest cause of massive bleeding is a peptic ulcer; but in some areas it is bleeding from the terminal ileum, or ascending colon, due to typhoid or amoebiasis. Perform a sigmoidoscopy to check if the bleeding is coming from higher up. At laparotomy, you may be able to tell quite easily if the bleeding is from the small or large bowel, but it may be more difficult to determine if it is from the right or left colon: make a small longitudinal opening, preferably with diathermy, in the caecum (the Peep test). If you find blood in the lumen, the source is likely to be the right colon. Then perform a right hemicolectomy; otherwise perform a left hemicolectomy.

(b) Indications for laparotomy for rectal bleeding
(1) Loss of >1500ml of blood with unknown cause. If he is *in extremis*, surgery may be life-saving.
(2) The presence of a mass.
N.B. Most colonic bleeding stops on its own, so don't operate too early.

PREPARATION. Resuscitate with IV fluids if shocked, and transfuse blood to get an Hb level of at least 80g/L, and have more blood in reserve for operation.

LAPAROTOMY. Make a long midline incision. Look at the stomach and duodenum and feel for irregularities and signs of ulceration. Then examine the entire bowel from the duodeno-jejunal flexure down to the rectum. Note the colour of the contents of the bowel, which is purplish if it has blood inside. What is the highest site in the bowel to show bleeding? Look for abnormal vessels going to the bleeding area, and feel for induration or an ulcer. If necessary, open the bowel (11.3) or stomach (13.5) to find the level of the bleeding. Using an endoscope (13.2) through the opened bowel is very helpful.

If you are confident you have found the lesion, perform a localized resection (11.3), but if there is severe bleeding from the right colon, you don't find a lesion, and there is no bleeding more proximally, perform a 'blind' right hemicolectomy (12.11). This will not be easy, so don't do it lightly. Afterwards, open the specimen to see where the blood is coming from.

If the bowel is severely inflamed, resection may be more hazardous than a conservative approach (12.3,4).

26.5 Anal fissure

An anal fissure causes suffering out of all proportion to its size. There are essentially two types, the classical and the HIV-related. The latter is internal and not seen by gentle parting of the buttocks, and not usually associated with muscle spasm. Both types present as a defect in the mucosal lining in the lower part of the anal canal, which makes defecation, and the half-hour following it, acutely painful. Even the thought of a bowel movement may fill the patient with such dread that he suppresses the urge, so that the hard constipated stools that he eventually passes make the fissure worse, and may occasionally make it bleed. With HIV disease, however, he often has a loose stool.

(a) The classical fissure usually occurs posteriorly in the midline, between the anal verge and the dentate line, directly over the distal end of the internal sphincter. A small oedematous skin tag commonly forms on the anal verge, just posterior to the fissure. This is the 'sentinel skin tag'.

Later, the fissure may become indurated and infected, and may lead to a low perianal abscess (6.17). This may discharge through the fissure, and externally, to produce a low anal fistula. The internal sphincter lies directly under the fissure, and after several months of exposure this becomes fibrosed and spastic.

(b) The HIV-related fissure arises often posterolaterally proximal to the dentate line, and develops into a smooth shallow ulcer which deepens and may collect pus. This may then fistulate with a wide track (26.3) externally or into the vagina. The cause of this problem is rarely clear, and its treatment is unsatisfactory.

(c) Examination
An acute fissure is very painful, so don't try to do a rectal examination if you thereby hurt the patient even more. You may not be able to pass a proctoscope until you have gently introduced a submucosal injection of LA or administered a GA. You can, however, usually see a classical fissure by parting the buttocks gently; there is often a sentinel skin tag. The HIV-related fissure feels like an irregularity *inside* the anal canal; you may see pus discharging from the anus. Distinguish this from pus discharging from an adjacent fistula.

If there is more induration, a larger ulcer, and perhaps enlarged inguinal nodes, think of a carcinoma or sexually transmitted infection: a **primary chancre** (the 1st sign of syphilis) has indurated margins, a symmetrical lesion on the opposite margin of the anal canal, and *no pain*. **Secondary syphilis** presents with a moist, pruritic anus, with flat, slightly raised lesions, which are usually symmetrical on both sides.

(d) Treatment depends on how long the fissure has been present.

If it is acute (<10 days), only the epithelium is involved and it will heal, if you keep the stools soft for 2wks with a laxative. When it has healed, warn that it may return, if constipation recurs. So advise a high-fibre diet. To reduce severe acute pain, introduce a condom (or the closed finger of a rubber glove) filled with water and frozen, into the anal canal. You can also help with LA ointment (5% lidocaine): this should be smeared over the sphincter inside the anus, *not outside it*. Injection of submucosal LA, though effective, is rarely tolerated by most patients.

If the fissure is chronic (>30 days), fibrosed, of classic type, and has a sentinel skin tag, and especially if you can see the exposed fibres of the internal sphincter under it, it will probably not respond to non-operative treatment, though it may improve with glyceryl trinitrate 0.2% or diltiazem 2% cream locally. If symptoms persist for months and there is anal spasm, and the HIV test is negative, consider a lateral sphincterotomy, or excision of the fissure and skin tag. This second is a delicate procedure best left to an expert, though.

If the fissure is of the HIV-related variety, treat with laxatives, if there is constipation (unusual), and nalidixic acid or ciprofloxacin with metronidazole, if there is a purulent discharge from within the anal canal.
Advise against using jelly with the spermicide nonoxylon-9.

LATERAL ANAL SPHINCTEROTOMY (GRADE 1.3)
Don't do this operation in an untreated HIV+ve patient; it may well not heal, and is unlikely to help!

Use laxatives before the operation. Use the lithotomy position and administer a GA. Examine the fissure to exclude an intersphincteric abscess. Insert either a bivalved operating proctoscope or Lockhart-Mummery anal retractors, and rotate these to show the left lateral wall of the anal canal. Palpate the edge of the internal sphincter. Make a 1cm radial incision into the mucosa over the sphincter and, with scissors, separate the epithelial lining of the anal canal from the internal sphincter and the internal from external sphincters. Open the scissors with blades either side of the *internal* sphincter and divide it. *Don't make a large cut*, which might render the patient incontinent: a partial division of the internal sphincter usually suffices. *If you cannot define the anatomy, don't proceed!*

LATERAL ANAL SPHINCTEROTOMY

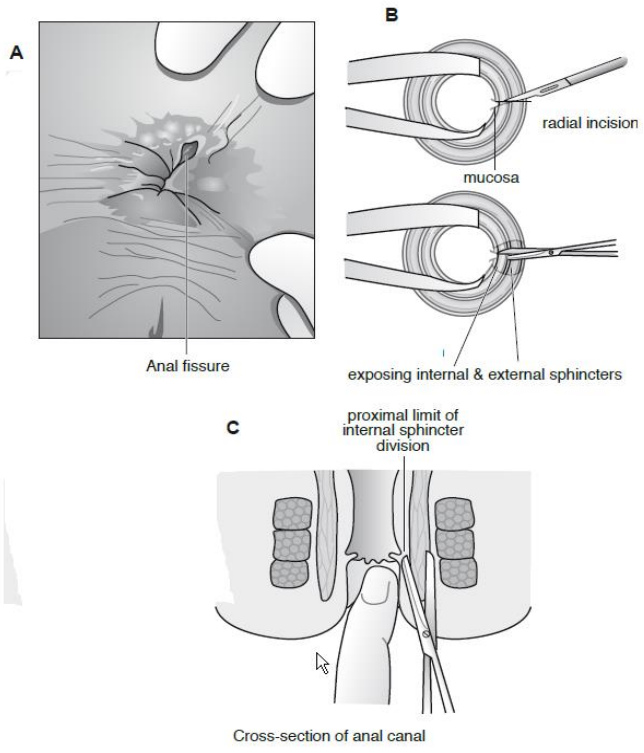


Fig. 26-9 LATERAL ANAL SPHINCTEROTOMY.
A, appearance of chronic anal fissure. B, radial incision exposing internal and external sphincters, and scissors inserted between internal and external sphincters, with one blade below and another above the internal sphincter, before division of the *internal* sphincter. C, position of incision of internal sphincter with the index finger at the dentate line.
After Morris PJ, Malt RA. Oxford Textbook of Surgery, OUP 1994 p.1141.

N.B. NEVER PERFORM AN ANAL STRETCH! It is a crude, uncontrolled way of doing a sphincterotomy and may well result in permanent incontinence, or even anal gangrene, especially in HIV+ve patients.

26.6 Perianal warts

These are most commonly discrete multiple cauliflower-like lesions in the perianal area, known as *condylomata acuminata*. They are caused by a papilloma virus, transmitted sexually or through close physical contact (*e.g.* sleeping in the same bed), and may extend inwards as far as the dentate line, and become infected and ulcerate. There is a very strong but not absolute association with HIV disease, where if untreated they develop into squamous carcinoma.

Less commonly, they are large verrucous lesions with a pale brown centre, composed of many smooth warts, known as *condylomata lata*. They are caused by syphilis (*treponema pallidum*) and respond to penicillin, doxycycline and azithromycin. There is frequent association with HIV disease.

EXCISION OF PERIANAL WARTS (GRADE 1.3)
Make sure you are dealing with *condylomata acuminata.*

Infiltrate the perianal skin with dilute lidocaine with adrenaline. Carefully remove the growths with cutting diathermy or scissors. Treat the raw areas that are left with hypochlorite diluted 1:100 for 1wk, then with saline dressings, like any other perianal granulating lesion. If the warts are extremely voluminous, you should remove them in staged procedures in order to prevent the development of anal stenosis, which occurs if you excise warts at or inside the anal verge.

Treat with laxatives and adequate analgesia post-operatively, and insist on a daily douche and every time after defecation. Try to trace sexual contacts, and examine them for genital or perineal warts.

26.7 Anorectal carcinoma

A malignant ulcer at the anus is usually a squamous carcinoma if arising *de novo* or as a result of chronic infestation with *condylomata acuminata* (26.6); it may be an adenocarcinoma if extending inferiorly from a low rectal carcinoma. Rarely, you may find a malignant melanoma at the anus.

In HIV+ve patients a tumour related to the papilloma 16 virus, called anal intra-epithelial neoplasm (AIN), may develop. This looks like patches of reddened skin resembling Bowen's disease (a pre-cancerous scaly red ulceration), and progresses to squamous carcinoma.

If you see suspicious areas like this early, and can excise them locally, you may be able to prevent the degeneration to invasive carcinoma.
Anal Kaposi sarcoma and non-Hodgkin's lymphoma also occur in HIV+ve patients.
These lesions may be extensive and fungating on presentation, when you will be unable to achieve excision with adequate clearance. Special techniques in well-equipped centres may still effectively deal with such tumours; otherwise the alternative is an abdomino-perineal resection, which many such patients will not be able to tolerate. Consider carefully if a defunctioning sigmoid colostomy (11.6) will benefit; biopsy is essential to make a diagnosis.
Occasionally if the lesion is small and near the anal margin, you can infiltrate it with LA solution containing adrenaline, and excise the tumour widely. You will need anal retractors and an assistant to achieve this.

Differentiate between an anal carcinoma and LGV, chancroid, schistosomiasis, amoebiasis and donovanosis, all of which can produce a destructive ulcer.

N.B. Rectal carcinoma arises above the dentate line (12.11, 26-1) where the rectal mucosa starts.

26.8 Rectal prolapse (Procidentia)

Occasionally, the rectum prolapses out of the anus. It may prolapse incompletely, so that only a pink fold of mucosa shows, or it may prolapse completely, so that the whole thickness of the rectal wall is turned inside out (procidentia), and may ulcerate. At the same time the anal sphincter may stretch and become patulous, so that incontinence results. At first the rectum prolapses only with defecation, later it does so on minimal coughing and straining; finally it is outside all the time.

Although the rectum can prolapse at any age, it commonly does so in children of 3-5yrs (usually incompletely), and occasionally does so in the aged (usually completely). Prolapse is more common in malnourished children, perhaps because of poor tone and weakness of the anal sphincter mechanism, and is also associated with diarrhoea as well as straining when seriously constipated. If a child's diarrhoea and malnutrition are treated, the prolapse is usually cured also. A chronic cough, especially with whooping cough and cystic fibrosis, whipworm (*trichuris*) infestation and coeliac disease (reaction to gluten) predispose to prolapse. Prolapse often occurs in babies with spina bifida (33.10) and ectopia vesicae (33.15). Prolapse occurs also in those who practise anal intercourse.

A child's rectal prolapse is usually noticed by the mother who says that something red appears at the anus after defecation. When she brings him to you, there is usually nothing to see. If there is, you can usually replace the rectum manually, but it is likely to prolapse again. If it remains prolapsed too long, it ulcerates.

The prolapse will however correct itself with age and improved nutrition. You should make sure the child sits properly during defecation, rather than squatting, and you can also strap the buttocks (26-10). If this does not prevent prolapse recurring, apply gallows skin traction (pulling the buttocks up off the bed) and watch the prolapse reduce spontaneously.

An adult's rectal prolapse is much more difficult to treat. Symptoms are the result of the prolapse itself. However, it may be due to chronic large bowel obstruction from malignancy or *schistosomiasis*, so ask about a history of constipation.

Examination
If the prolapse is intermittent, the history may be of 'something coming down', but there will be nothing to see. In an adult, pass a proctoscope and ask the patient to strain down. The anal mucosa will prolapse into the hollow of the proctoscope, and extend beyond the anus as you withdraw it. If the prolapse is complete, the whole thickness of the rectum slides out all round, sometimes for several centimetres. At rectal examination, the anal sphincter feels weak.

(a) **To find out if the prolapse is partial or complete**, put your finger into the rectum, and feel the protruding ring of mucosa between your finger and thumb. If all you can feel is 2 layers of mucosa, it is incomplete; if you can feel more tissue than merely mucosa, it is complete.

(b) **In a child**, distinguish a prolapse from a rectal polyp, or an *ileorectal intussusception*. Examine immediately after defecation. Feel the outer aspect of the swelling, up to the anal orifice. In an ileorectal intussusception, you can pass your finger *between* the *intussuscipiens* and the anal wall: you can't do this with an anal prolapse. A rectal polyp is mobile and dangles from the anorectal wall, and you can flick it with your finger: make sure it cannot be squeezed like faeces.

N.B. Strictly speaking, a rectal prolapse is a recto-rectal intussusception.

If a child has diarrhoea, treat that. If the nutrition is poor, treat that first. These are the common causes of prolapse, and treating them usually provides a cure and avoids an operation. Regular small doses of a mild sedative help; put the child on a potty-chair, not sitting on a pot on the floor.

MANUAL REPLACEMENT AND STRAPPING
Using a glove and lubricant jelly, replace the prolapse manually. You may have to squeeze it for 15mins to do so. If it is very oedematous, apply gauze with icing sugar, which will soak up the oedema fluid and allow you to reduce the prolapse later.

Strap the buttocks securely together with the large gauze pad up against the anus. If this method is to work, the strapping must be adequate, painless, and easily applied. Apply a large square to each buttock.

Join these with a 2½-5cm transverse strip, so as to close the buttocks, and leave this strip on during defecation. Afterwards, remove it, clean the buttocks, and replace it with a fresh strip (26-10). Ask the parents to repeat this after each bowel movement, and give them some petroleum jelly gauze, plain gauze, and strapping with which to do it. After a time, the rectum will stay up where it belongs. Strapping is often all that is necessary. If, after 3-4 reductions, the prolapse soon recurs after defecation, put up gallows traction.

CAUTION! Too much trauma trying to reduce a prolapse causes bleeding; in this case, proceed to gallows traction.

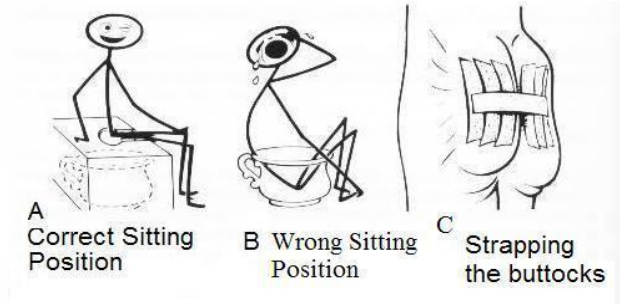


Fig. 26-10 CORRECT SITTING POSITION FOR DEFECATION AND CORRECT METHOD OF STRAPPING FOR RECTAL PROLAPSE. A, correct sitting position. B, avoid the squatting position. C, only the transverse strip requires replacement after defecation.
After Jones PG, Woodward AA. Clinical Paediatric Surgery Blackwell 3rd ed 1986 p.324.

GALLOWS TRACTION usually allows the prolapse to reduce: use this for a maximum of 2wks.

SCLEROSING PHENOL INJECTIONS
Put 0·5ml of 5% phenol in almond oil into the submucosa at three equally spaced points, 2cm above the dentate line. This will cause some fibrosis; use this method only if strapping and gallows traction fail in those cases with loose stools and flabby tone.

THIERSCH'S OPERATION FOR RECTAL PROLAPSE (GRADE 2.1)
This method is really only applicable to children with severe anal hypotonia or other neurological problems: *it is absolutely contraindicated in cases of constipation!*

Use the lithotomy position and use ketamine; replace the prolapsed rectum (26-11A). Put your finger in the anus and feel the sphincter. It may be so loose that you can hardly feel it.

Make short incisions anteriorly and posteriorly in the midline 2cm from the anus (26-11B). Then, put a large curved round-bodied needle with #1 absorbable suture into the skin anteriorly in the midline 1cm from the anus. Pass it subcutaneously round the anus 1cm from it and out again posteriorly in the midline (26-11C). Pull the suture material through. Put the needle back into the posterior hole from which it has just come. This time pass it round the other side of the anus and out at the anterior incision (26-11D). Ask your assistant to put the little finger into the child's anus (26-11E).

Tie the suture round the finger. Secure it with several knots, cut the ends 1cm long and bury them. Close the 2 skin wounds.

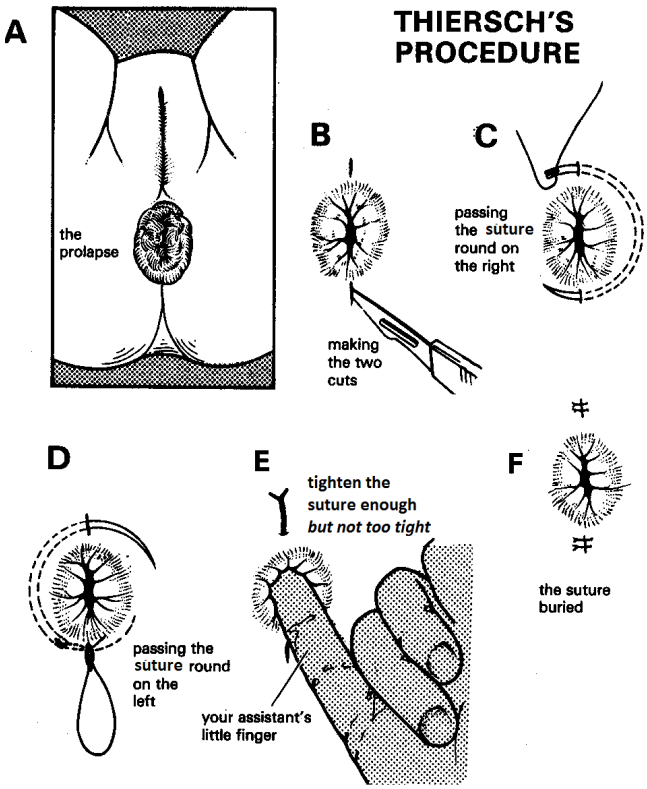


Fig. 26-11 THIERSCH'S PROCEDURE.
A, child's prolapsed anus. B, make two cuts in the skin 2cm from the anus. C, pass the suture material from anterior to posterior on the right-hand side. D, now pass it round on the other side. E, tighten the suture material with your assistant's little finger in the child's anus. F, finally bury the sutures.
After Goligher JC. Surgery of the anus, rectum and colon. Ballière Tindall 4th ed 1980 Fig 187 with kind permission.

CAUTION!
(1) You must be able to get the tip of your little finger into a child's anus. Getting the tension of the suture material right is difficult. If it is too tight, it will interfere with defecation, and cause faecal impaction, or the wire may cut out. If it is too loose, it will not cure the prolapse.
(2) *Don't forget to make sure that he can pass stools normally before discharge*.
The major complications are breakage of the suture, and difficulty in passing even a soft stool, if the suture is too tight.

(c) **In an adult**, you may find that the prolapse is reduced when you start your examination, but appears with straining. The anal orifice may be widely open, and the sphincters abnormally lax. Assess their tone with your finger, because this is an important determinant of treatment and prognosis. You may feel very little contraction. If it is very lax, you may be able to put 3 or 4 fingers into the anus without discomfort. This is especially so amongst those who practise anal intercourse.

If there is an incomplete prolapse and the tone of the sphincter is fairly normal, you can treat it in much the same way as large third-degree haemorrhoids (26.9). Reduce the prolapse and inject 2ml 5% oily phenol at three equally spaced points under the redundant mucosa.

If there is a complete prolapse, try to reduce it manually with adequate lubrication. If this proves difficult because the prolapsed rectum is very oedematous, inject 10ml solution of 3,000U of hyaluronidase submucosally, and squeeze gently after 2-3mins.

If the prolapse recurs frequently, you can either excise the prolapsing bowel leaving no more slack to allow further prolapse (perineal rectosigmoidectomy), or pull up the rectum from inside the abdomen and fix it (abdominal rectopexy). The Thiersch procedure does not work well in adults, being either too tight causing constipation (when often the suture breaks on straining), or too loose resulting in recurrent prolapse.

PERINEAL RECTOSIGMOIDECTOMY

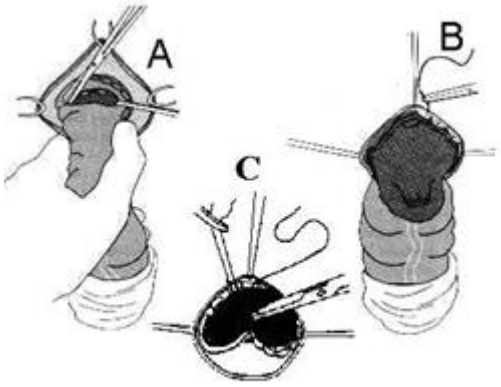


Fig. 26-12 PERINEAL RECTOSIGMOIDECTOMY.
A, after putting stay sutures and dividing the outer tube of rectum, pull down the inner tube to bring the sigmoid to the pelvic floor. B, divide the redundant bowel superiorly and suture outer and inner rectal tubes. C, after dividing the redundant bowel inferiorly, complete the suturing by fixing the anal remnant to the sigmoid, leaving no slack for further prolapse to occur.
After Morris PJ, Malt RA. Oxford Textbook of Surgery, OUP 1994 p.1106 Figs 6-7.

PERINEAL RECTOSIGMOIDECTOMY
(ALTERMEIER OPERATION) (GRADE 3.2)
Administer bowel preparation and use the lithotomy position with the legs elevated. Use LA or spinal anaesthesia, rather than GA, as these patients are often old.

Don't reduce the prolapse, but rather pull it fully out; put 4 stay sutures anteriorly, posteriorly, left and right through the outer rectal wall 1·5cm above the dentate line and divide the two layers of prolapsed colorectal tube circumferentially (26-12A).

Then hold the inner colonic tube with Allis forceps and pull it down till no more protrudes; it is important that you take up all the slack in order to prevent further prolapse. Close any gap or laxity in the puborectal sling (the *levator ani*) posteriorly, if necessary by overlapping the muscle layers.

Place 4 stay sutures on the inner tube in the same way as before, just proximal to where you intend to cut off the redundant bowel, and then divide it anteriorly, preferably with diathermy. Clip the anterior stay sutures together, and likewise the others: this aligns the 2 rectal tubes nicely.

Then suture the remaining parts of both rectal tubes anteriorly with continuous long-acting absorbable suture (26-12B). You can then safely divide the remaining posterior part of the inner tube and complete the suturing (26-12C). Finally, pull on the stay sutures to check your anastomosis, and when you are satisfied, cut them and allow the bowel to retract inside the anal canal.

CAUTION! Don't let go of the inner colonic tube; if you do and cannot retrieve it, perform a laparotomy to find the retracted portion of bowel in order to bring it down again.

ABDOMINAL RECTOPEXY (GRADE 3.3)
Expose the pelvis through a lower midline incision, and pack away the bowel. Mobilize the rectum down to the pelvic floor, anteriorly and laterally by incising the peritoneum, but *not* dissecting posteriorly. *Don't divide the lateral ligaments* (the sacro-uterine ligaments in a woman, 21-18), but use them to keep the bowel up out of the pelvis when you pull up the rectum. Using non-absorbable #1 multifilament sutures, pull the rectum firmly upwards towards the sacral promontory, and fix it there. Then suture the taut 'lateral ligaments' to the presacral fascia.

CAUTION!
(1) *Don't penetrate the wall of the rectum*.
(2) Be sure to put all the sutures in first and then tie them later.
(3) Make sure the rectum is pulled up well out of the hollow of the sacrum.

26.9 Haemorrhoids (Piles)

The arterio-venous haemorrhoidal plexuses of the anal canal may become swollen and start to protrude. They form in the left lateral, and right antero- and postero-lateral (*i.e.* the 3, 7 and 11o'clock) positions, and although they usually cause no symptoms, they can bleed and cause severe anaemia; the bleeding is painless, fresh and not mixed with stool, coming especially after defecation. They can also cause an irritating mucous discharge.

They can prolapse, and spontaneously reduce, being known as 2º haemorrhoids, or prolapse permanently as 3º haemorrhoids. These may then thrombose and become very painful.

Untreated, however, 1º (non-prolapsing) haemorrhoids usually eventually shrink. They may be the site of porta-systemic blood shunting in portal hypertension: check for this before you decide to operate!

N.B. Haemorrhoids are NOT painful unless prolapsed. Pain is usually due to an anal fissure or ulcer (26.5). *Don't treat normal anal structures when there are minimal symptoms.*

(a) Examination

You cannot see haemorrhoids unless they are prolapsing, except through a proctoscope. Prolapsing haemorrhoids form large projecting bluish swellings protruding from the anus, only their outer parts covered with skin, and their inner parts with purple anal mucosa, separated by a groove, at the three main positions. There may be accessory haemorrhoids in between.

You may see distended veins at the anorectal junction in portal hypertension: these don't look exactly like haemorrhoids.

If you see a single tender bluish swelling c.1cm diameter at the anal verge, totally covered by skin, this is a perianal haematoma, *not a haemorrhoid*. If it is <24h old, you can incise and drain it under LA. Otherwise leave it to organize and resolve, providing the patient laxatives and analgesia.

Never incise prolapsed haemorrhoids: the bleeding is catastrophic! Don't confuse skin tags with haemorrhoids, which have an internal mucosal lining.

(b) Proctoscopy (26.1) is the only satisfactory way to diagnose 1º and 2º haemorrhoids. They bulge into a proctoscope like grapes, as you withdraw it and ask him to strain. Withdraw it just to the anus, and then ask him to continue straining.

(c) Sigmoidoscopy (26.1) must be a routine, if there is a history of bleeding, to exclude serious pathology, especially a carcinoma, particularly after age 40yrs and you cannot see any haemorrhoids!

(d) Sclerosing phenol injections

Put 2ml 5% phenol in almond or peanut oil under the mucosa just proximal to the haemorrhoids at each site (26-13): you will need a proctoscope and a light to do this, and it helps to have an assistant. The oil is viscous and so you need a long wide-bore needle: attach this to a syringe small enough to fit inside the proctoscope. The procedure should be virtually pain-free; if you are causing pain, you may have injected deep into the prostate; withdraw and re-insert the needle.

You can use this method safely with HIV+ve patients, where other methods (such as open haemorrhoidectomy or tying with rubber bands which anyway needs special equipment) are not advisable. With large haemorrhoids, the injections may need to be repeated after 6wks, and again at 12wks. If this fails, you should think about haemorrhoidectomy.

Don't use sclerosants on prolapsed haemorrhoids; you should wait till they are reduced.

(e) Prolapsed 3º haemorrhoids

Don't rush into recommending haemorrhoidectomy for these: the surgery may be difficult and bloody. Try to reduce them after applying gauze with fine sugar and petroleum jelly to absorb oedema fluid, and using sedatives and laxatives, for 4-5 days. This will, however, not work if the haemorrhoids are already thrombosed.

SCLEROSANT INJECTION OF HAEMORRHOIDS

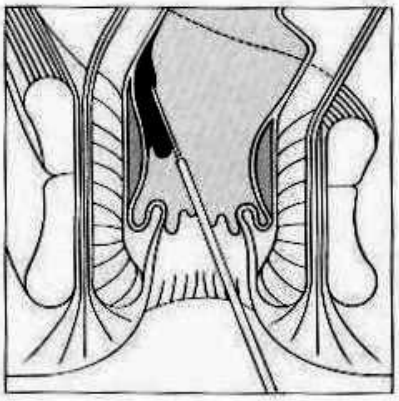


Fig. 26-13 SITE OF INJECTION OF SCLEROSANT FOR HAEMORRHOIDS. Site of injection under the mucosa.
After Geile D, Scheidter KH. Proctological Compendium. PMS Munich 1981.

NEVER PERFORM AN ANAL STRETCH

(e) Haemorrhoidectomy (GRADE 2.3)

INDICATIONS

(1) Irreducible 3rd-degree haemorrhoids.
(2) Chronically thrombosed 3rd-degree haemorrhoids.

CONTRAINDICATIONS

(1) HIV+ve patients.
(2) Septic haemorrhoids.
(3) Acutely thrombosed haemorrhoids.
(4) Portal hypertension.

RELATIVE CONTRAINDICATION

Severe constipation.

PREPARATION

Treat with bowel preparation (an enema is usually too uncomfortable) and metronidazole pre-operatively. Check the HIV status, and cancel the operation if the test comes back +ve.

You can use LA (with sedation) if you are gentle: infiltrate c.50ml 1% lidocaine with 1:200,000 adrenaline in the perianal skin, ischiorectal fossae (26-1), retrorectal space and haemorrhoidal pedicles.

Use the lithotomy position, with the buttocks well beyond the end of the table. A sandbag under the sacrum helps exposure. Clean the anal region, and do a careful digital examination to make sure that there is really no other pathology. Perform a sigmoidoscopy if you have not already done so.

If you don't have diathermy, infiltrate the subcutaneous tissues round the anus with 1:100,000 adrenaline in saline or lidocaine (26-14A).

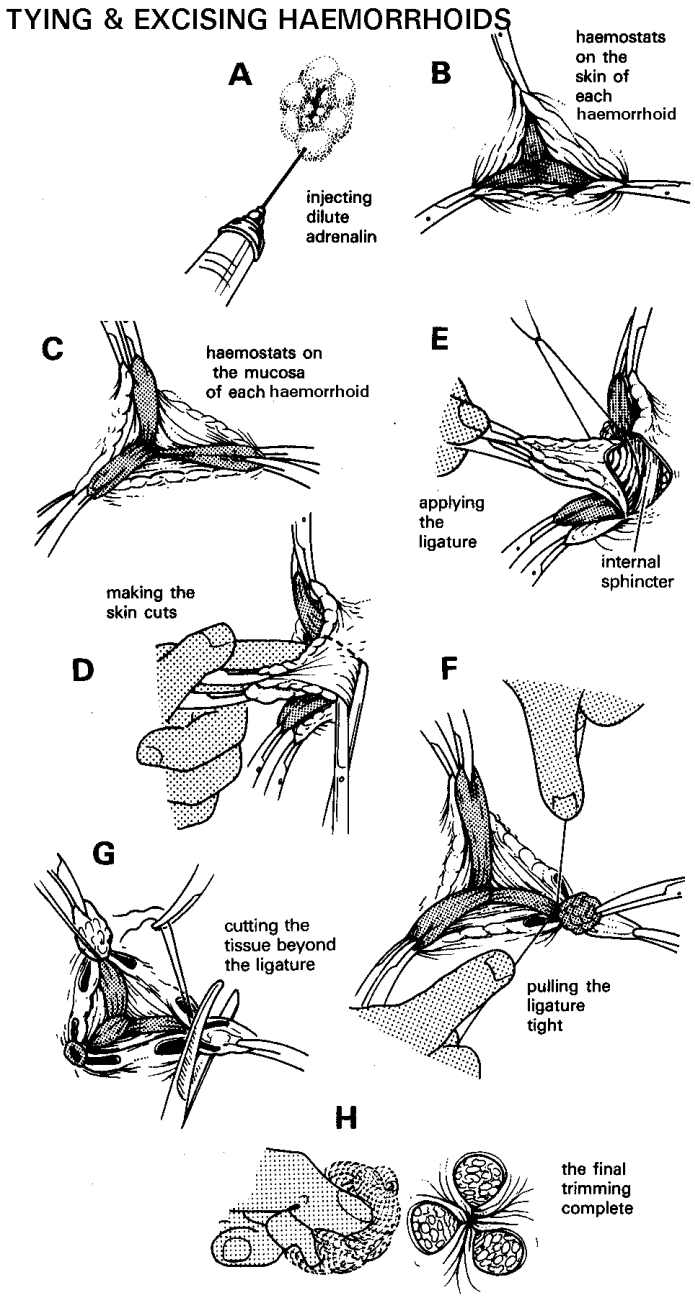


Fig. 26-14 TYING AND EXCISING HAEMORRHOIDS.
A, inject adrenaline in saline or lidocaine to control bleeding. B, apply forceps to the skin of each primary haemorrhoid. C, apply a 2nd pair of forceps to each haemorrhoid where it is covered by mucosa. D, make the skin cuts for the left lateral haemorrhoid. E, snip the mucocutaneous junction at the neck of each haemorrhoid and tie it. F, pull strongly as you tie a haemorrhoid, release the forceps as you do so, and allow the ligature to sink into the tissues. G, after you have tied all 3 haemorrhoids, excise the left lateral and then the right postero- and antero-lateral haemorrhoids, taking care to leave adequate stumps and skin bridges. H, final skin wounds trimmed.
After Goligher JC, Surgery of the anus, rectum and colon. Ballière Tindall 4th ed 1980 Figs 80-7 with kind permission.

METHOD
Push some dry gauze into the rectum, and slowly pull it out. The haemorrhoids will prolapse with it. Grasp the skin at the mucocutaneous junction of each haemorrhoid with haemostats, and pull them outwards (26-14B). Take the purple mucosa-covered part of each haemorrhoid in other larger haemostats, and draw them downwards and outwards.

This will bring all 3 haemorrhoids well out of the anus, so that you see the pink rectal mucosa at their upper ends (26-14C). Pull on all 6 haemostats until you see the rectal mucosa, not only at the upper end of each haemorrhoid, but also between them, and secure the haemostats with towel clips to give you a clear field. Draw the haemorrhoids out as far as they will go, which will allow you to tie them at their upper poles, rather than around their middles.

With cutting diathermy, make a V-shaped cut in the anal and perianal skin opposite the left lateral haemorrhoid (26-14D). The ends of the V should reach the mucocutaneous junction, but not extend into the mucosa beyond it. The apex of the V should lie 2.5-3cm from the junction. You will see the lower edge of the internal sphincter laid bare. This is a firm, whitish ring which should be clearly visible and which you should avoid damaging. If you hold the haemorrhoid aside (26-14E), you will see it quite clearly.

Transfix the pedicle of each haemorrhoid using #0 or #1 long-acting absorbable suture (26-14F), cut off the haemorrhoid 1cm distal to the transfixion suture (26-14G) and transfer the haemostats holding the haemorrhoid to hold the ends of the ligature, retracting them laterally once more. Leave the ligature ends long. Cut them short after 24h.

CAUTION! A slipped ligature can cause fearsome bleeding, so always *transfix* the haemorrhoid!

Treat the other haemorrhoids in the same way, *leaving 1cm skin and mucosal bridges between each pedicle*, but *don't be tempted to remove any accessory haemorrhoids.*

Push some dry gauze into the anus, and examine for haemostasis; you may need a lubricated speculum to look at the ligatures. Infiltrate 2ml bupivacaine into each pedicle for post-operative pain relief. The end result should look like a clover leaf (26-14H). Apply petroleum jelly gauze to each of the 3 raw areas on the anus, and cover this with cotton wool. Hold it in place with a T-bandage.

Remove the dressings in the bath the next day. Use showers on subsequent days. Treat with laxatives and adequate non-constipating analgesia, paracetamol or NSAIDs. If no stool is passed by the 3rd day, use a glycerine suppository. *Don't discharge the patient before he passes stool* because otherwise faecal impaction may result!

(f) Difficulties with haemorrhoidectomy
If there are accessory haemorrhoids, only excise the main ones, so that that you only make 3 skin wounds.

Leave the accessory haemorrhoids: they will shrink of their own accord. *Don't try to excise separate skin tags outside the main skin wounds.*

If there is an associated anal fissure, treat it (26.5) and leave the haemorrhoids alone.

If there is post-operative pain, treat with pethidine. If severe pain follows defecation, a hot bath will soothe the discomfort.

If there is difficulty passing urine, try using pethidine and encourage passing urine in a warm bath. If this fails, catheterize the bladder, and remove the catheter after 48h. Alternatively, perform a suprapubic aspiration.

If there is bleeding within 12h (reactionary haemorrhage), usually due to a slipped ligature, adrenaline wearing off, or a rise in BP: pull down on the ligatures, which you left long deliberately for this eventuality, and try to secure the bleeding vessel with artery forceps in the ward. If this fails, return to theatre to perform a formal proctoscopy to find and ligate the bleeding vessel under GA.

N.B. There may be torrential bleeding from portal hypertension if the haemorrhoids are the sites of porta-systemic shunts: in this case, use vitamin K, fresh frozen plasma and tranexamic acid if you can get these.

If there is bleeding between 7 and 10 days (secondary haemorrhage), this may occur into the rectum, appearing as clotted blood with the next stool. It is due to infection eroding into a blood vessel.

Bleeding may stop spontaneously; if it does not, try pushing a lubricated, adrenaline-soaked pack into the anus and lower rectum. If this is inadequate or impractical, insert a large Foley catheter, inflate it, tie a 500g weight to it, and exert traction on the bleeding site. If bleeding persists after 24h, prepare theatre as above.

If the stools become impacted, this is probably the result of being allowed home without adequate or with constipating analgesia. You will need to extract faeces manually under anaesthesia. If the wounds are still raw, start metronidazole and use sufficient laxatives.

If a stricture develops, you probably did not leave adequate bridges of tissue between the excised haemorrhoids. Provide the patient with an anal dilator and show him how to pass this daily for 3 months.

If there is mucous prolapse post-operatively, *don't rush in to excise further tissue.* Healing will in most cases cause the anal mucosa to retract spontaneously.

26.10 Pilonidal infections

Long straight hair sometimes works its way into the skin; this occurs especially in the natal cleft just posterior to the anus but also at the umbilicus to form an abscess, sinus or fistula. This occurs in people with copious long straight hair, most commonly young males.

There may be one or more openings, sometimes with hairs coming out of them, exactly in the midline 5cm posterior to the anus. Often, there is another sinus, 2-5cm superiorly, and slightly to one or other side of the midline, with an indurated track joining it to the first one.

Don't mistake a pilonidal sinus for a subcutaneous or perianal fistula (26.3). If you are in doubt, remember:
(1) In a pilonidal sinus, there will be no induration between the lowest sinus and the anus.
(2) There will be no fistulous opening inside the anus.
(3) When you probe the lowest sinus, the probe will pass towards the sacrum, not the anus.

Aim to excise the sinus without any surrounding tissue, make sure that the wound heals properly, and prevent hairs growing into it as it heals.

If there is no infection, excise the affected area; if infected, aim initially only for simple drainage. The most important part of the post-operative care, after either method, is to make sure that new hair does not grow into the granulating wound.

(a) Acute infection. Incise and drain the abscess through a short incision, taking particular care to remove all hair and granulation tissue with a curette. Insert a drain. Treat any sinus developing later.

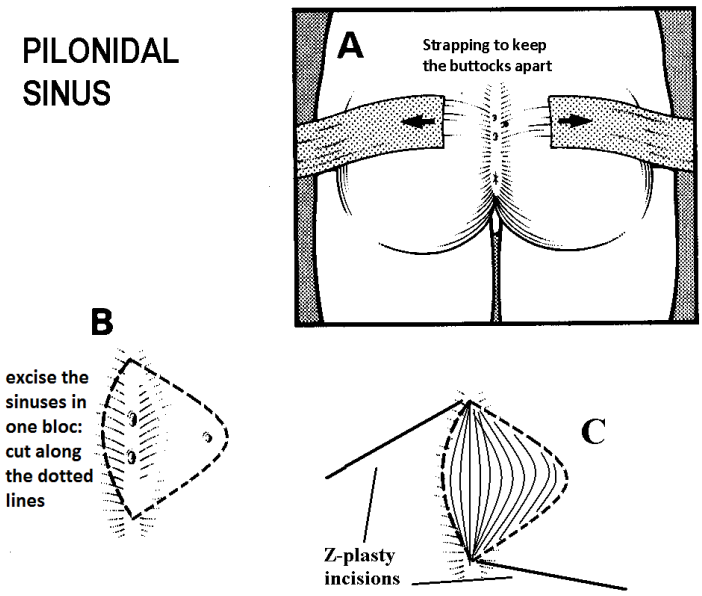


Fig. 26-15 EXCISION OF A PILONIDAL SINUS.
A, strap buttocks apart to show openings of the sinuses. B, excise all the sinuses *en bloc.* C, close the wound with a Z-plasty without tension (34.2).
After Rob C, Smith R. Operative Surgery: Abdomen II, Butterworth 1981 with kind permission.

(b) Excision of pilonidal sinuses (GRADE 2.4)

INDICATIONS
At least 2 episodes of infection, and a persistent discharge. Be sure to operate at a time when the symptoms are quiescent and infection absent.

PREPARATION
Advise the use of depilatory creams, or pull out offending hairs individually. Shave the area near the buttocks. Use gentamicin and metronidazole perioperatively. Draw lines on the lines of contact of the buttock edges when they are pushed together.

Use the left lateral position, with the buttocks over the edge of the table. (If you use the prone position, you will need intubation and GA.) Put a piece of gauze soaked in an antiseptic, such as chlorhexidine, over the anus, and towel up carefully. Ask your assistant to stand at the other side of the table, and to retract the right buttock (or use strapping, 26-15A). Injection of methylene blue dye makes the tracks much more visible, although this might not be necessary.

ANAESTHESIA
If the area is limited, you can operate under LA; otherwise with the patient on his side, with the hips flexed, there is no need for intubation; you can use ketamine or GA. *Don't use spinal or epidural anaesthesia* as there is a potentially septic lesion too close to the injection site.

METHOD
Probe all the openings to find in which direction the sinus tracks run. If this is difficult to elucidate, gently inject methylene blue dye into the tracks to mark their pathways: *beware not to inject too brusquely*, otherwise the whole area will be coloured blue!

If there are individual sinuses, remove a core of tissue 5mm around each pit, so that the midline defect remains <7mm wide. Clean the track you have made, if possible with a very small brush (as made for electric razors) or a small curette. Treat all side openings in the same way. When you are sure that there are no more pockets that might contain hairs, close the wounds primarily and apply a gauze dressing.
N.B. Don't try to pack the cavity.

If you cannot excise all the sinus tracks individually because there are lateral extensions, lay open the main sinus track and cut round the subsidiary sinus openings. Excise the whole affected area, down to the sacral periosteum (26-15B). *Don't leave any hairy sinuses behind*, because recurrence is then inevitable.

Avoid a midline closure. An effective way to do this is to perform the Bascom II cleft lift procedure; separate the skin from the side of the wound nearest the midline from its underlying fat, and advance this across the midline as an advancement flap.

If there is a wide area involved, which a simple advancement flap will not close (especially if your excision goes beyond the lines you draw on the buttock edges), perform a Z-plasty (26-15C, 34.2) to alleviate tension which would give rise to ischaemia and a high risk of re-infection and wound failure.

If you can, mobilize the gluteal fascia off the sacral edges, and re-suture it over the sacral bone, so that skin closure above is neat and totally without tension. *There is no indication for deep tension sutures!*

Close the wound only if there is really no infection, otherwise leave it open for several days, use daily showers and perform a delayed closure when there is no longer any sepsis.

For more complex extensive sinuses, you can achieve tension-free closure using the Limberg flap: make a rhomboid incision, with an extension arm as a transposition flap (26-16).

Post-operatively, regular sitz baths, a douche or shower are important. Keep the back and buttocks shaved free of hairs while the wound heals, or the sinus will recur. Eventually, the scar will become strong enough to withstand them.

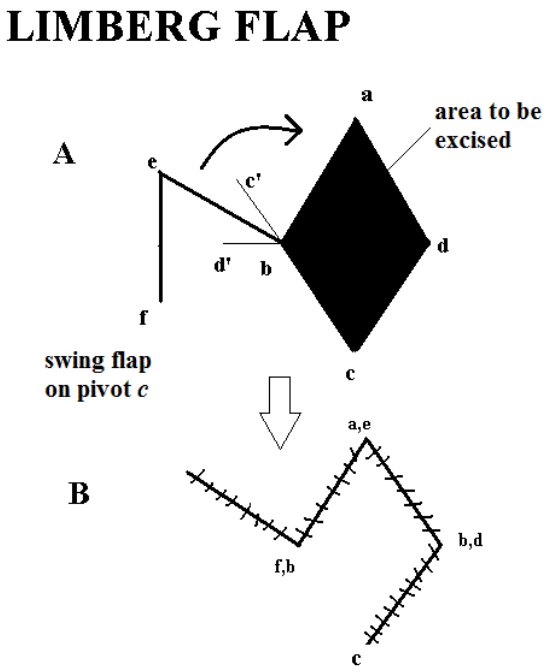


Fig. 26-16 LIMBERG FLAP.
A, draw a rhomboid with equal side around the area you want to excise, make a line *be* bisecting the angle *c'bd'* where *bc'* and *bd'* are extensions of *cb* and *db* respectively, and drop a perpendicular, *ef*, the same length, parallel to *ac*. Deepen the incisions along *be* and *ef* down to the gluteal fascia. B, swing the rhomboid flap (*cbef*) round into the excised area (*cdab*) without tension.
After Akca T, Colak T, Ustunsoy B et al, Randomized clinical trial comparing primary closure with the Limberg flap in the treatment of primary sacrococcygeal pilonidal disease. Br J Surg 2005;92:1081-4.

(c) Difficulties with pilonidal sinuses

If the wound bleeds post-operatively, apply some gauze, and apply pressure.

If the wound shows signs of infection post-operatively, reopen the wound, use daily sitz baths and perform a delayed closure once the wound is free of infection.

If there is excessive granulation tissue, curette it. Remove loose hairs.

If the skin forms a bridge across the lesion, with a dead space underneath, the sinus will recur. This is the commonest cause of recurrence, and is the result of poor operative technique or poor post-operative care; so try to get it right next time. Debride the wound and leave it open.

26.11 Other anorectal problems

(a) Rectal ulceration

An ulcer of the rectum may be benign or malignant; the distinction may not be obvious and so biopsy is important. All result in constipation, tenesmus (the feeling of something left behind after evacuation of a stool), mucus discharge and rectal bleeding. Causes are:
(1) **Persistent digital self-evacuation of faeces** (common in some communities) may produce a solitary linear ulcer 8-10cm from the anus.

(2) **An amoebic granuloma** (14.5) is a soft proliferative ulcer associated with amoebic trophozoites in the stools, and responds to treatment with metronidazole.

(3) **Gonorrhoea** produces an ulcer with thick yellowish purulent discharge, most commonly in active homosexuals. Treatment is with doxycycline or ciprofloxacin.

(4) **Primary syphilis** produces multiple eccentric irregularly located ulcers (chancres), which may be painful but often give no symptoms. The VDRL test is +ve. Treatment is with intramuscular penicillin.

(5) **Carcinoma of the rectum** (12.11) usually has a distinctive craggy hard feel, with an ulcer having rolled everted edges and a central crater.

(6) **Tuberculosis** may likewise produce a hard rectal ulcer.

(7) **Lymphogranuloma venereum** (caused by *chlamydia*) can produce an ulcer, especially in HIV patients, as well as a stricture. Treatment is with doxycyline for 3wks or azithromycin.

(8) **Radiation** >45 Gray from treatment of uterocervical, ovarian or prostatic cancer can lead to ulceration particularly resistant to treatment.

(9) **Herbal enemas**, in some communities, are used not just to ease bowel motions, but as aphrodisiacs or abortifacients. Potassium chromate as an ingredient can cause serious mucosal burning, and may be carcinogenic. Resultant ulceration or scarring may be extensive.

(10) **Colorectal leiomyopathy** is a strange condition affecting children and young adults possibly also related to enema use, where the bowel muscle wall becomes replaced by fibrous tissue. The rectum and the colon progressively distend enormously and fail to evacuate stools properly, but usually the patient's abdomen remains soft and he eats well. You might confuse this with Hirschsprung's disease (33.7).

Don't be tempted to fashion a defunctioning colostomy! This too becomes grossly distended! Treat these patients conservatively with bowel wash-outs.

LONE STAR ANORECTAL RETRACTOR

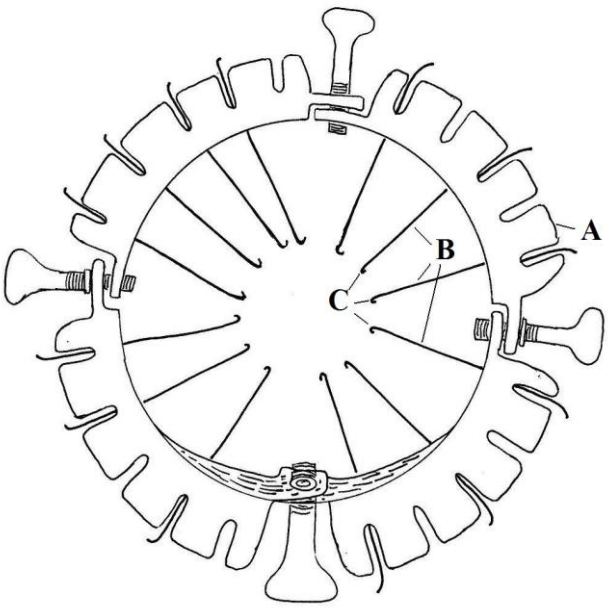


Fig. 26-17 LONE STAR RETRACTOR.
This very versatile device can be home-made. It consists of an outer ring (A), which can be jointed. You can fix skin hooks (B) to retract the anal margin, and attach them to elastic bands inside plastic tubing (C) onto the outer ring.

Managing a rectal ulcer
Make a careful examination under anaesthesia in theatre with good illumination. Assess the extent of the ulcer, its consistency, its fixity, and its spread to prostate, vagina, cervix or bladder. Look for adjacent fistulae, suggestive of tuberculosis. Take a biopsy; if you cannot get a histology report, smear cells onto a microscope slide and look for AAFB and irregular cancer cells. *Don't forget to check for amoebic trophozoites in the stool.*

A defunctioning colostomy (11.6) is only indicated when a rectal tumour is causing bowel obstruction, or if there is intolerable intractable faecal incontinence. If amoebiasis is common, try metronidazole whilst waiting for stool tests and biopsy results. The only medication that may help a radiation ulcer is sucralfate instilled rectally.

(b) Foreign bodies in the rectum

Any number and size of objects may be found in the rectum, or higher up in the sigmoid colon. It may be a stick or device to remedy constipation, or any sort of supposedly therapeutic tool, even a humming vibrator for sexual stimulation! Insist on good bowel preparation, and then do a sigmoidoscopy to try to coax the foreign object down. Ask the patient to perform a Valsalva manoeuvre.

You may have to use your ingenuity to grasp it; pushing on the abdomen from above may help. Extracting a round smooth object may produce an insurmountable vacuum; in this case, pass a catheter beyond the foreign body and introduce some air.

You can fill an open glass jar with plaster around a long forceps. For a smooth ball, use two long spoons and apply traction as with obstetric forceps! For a soft object, you can try skewering it with a myomectomy screw. *Beware, if the object or the tool you use is sharp*, that you don't lacerate the colorectum on removal; if you do so, and the damage is retroperitoneal, keep the patient nil orally and treat with metronidazole. If the damage is intra-peritoneal, perform a laparotomy to find the perforation and close it primarily in 2 layers (11.5).

Beware the 'body stuffer' who carries opioids in plastic containers within the rectum: these may burst, releasing a toxic dosage of drug which is rapidly absorbed, if you are too aggressive with laxatives or instrumental extraction.

(c) Rectal stricture

This may partly obstruct the rectum and cause alternating constipation and diarrhoea, with faecal incontinence, and ultimately cause total obstruction.

It may be due to:
(1) Lymphogranuloma venereum.
(2) Fibrosis following a corrosive traditional enema (usually producing a long stricture).
(3) Schistosomiasis.
(4) Amoebiasis (14.5).
(5) Haemorrhoidectomy without adequate skin bridges (26.9).
(6) Excision or diathermy of very extensive anal warts (26.6).
(7) Tuberculosis.
(8) Carcinoma (12.11).

A stricture due to lymphogranuloma venereum is usually a localized shelf-like lesion of hard fibrous tissue about 1cm deep, 5cm in from the anus, and lined by thin adherent anal skin. Sometimes there is a recto-vaginal fistula below the stricture. There may be multiple colonic strictures also.

If you remove the stricture entirely, *the result may be incontinence*.

The options are:
(1) carefully dilate it with Hegar's dilators under GA. Try not to tear it, or you will cause further inflammation and fibrosis.
(2) using the lithotomy position, and preferably using diathermy, make four V-shaped incisions (the apex pointing inwards) in the 12, 3, 6 and 9o'clock positions to remove four triangular pieces of fibrous tissue. (*N.B.* This way you don't completely excise the stricture.)

If obstruction is severe, fashion a defunctioning sigmoid colostomy (11.6) whilst you prepare for an abdominoperineal resection and a permanent end colostomy. If you have an anastomosis gun, it may be possible to place the anvil proximal to the stricture and thereby resect it endo-anally whilst at the same time improving the luminal diameter.

(d) Proctitis

Inflammation of the rectum may be localized or spread proximally into the colon. You can really only make a diagnosis by proctoscopy and biopsy. Look to see if the inflammation is patchy or continuous, and assess its extent and severity. The mucosa initially appears red, and then becomes heaped up, sometimes with polypoid protuberances; if severe, there is contact bleeding, and it may then be too painful to proceed without an anaesthetic.

Symptoms are pain with loose blood-stained stools. You should exclude dysentery (*shigella* may cause quite a florid proctitis). There are indeed many causes, and so a biopsy is essential, as well as stool examination.

Proctitis may be due to:
(1) *Chlamydia trachomartis.*
(2) Gonococcus.
(3) Amoebiasis.
(4) *Schistosoma mansoni*.
(5) Tuberculosis.
(6) Herpes simplex virus and fungal infections, especially in HIV disease.
(7) Ulcerative colitis.
(8) Crohn's disease.
(9) Radiation (best treated by sucralfate).
Suspect (1) or (2) where there has been anal sexual penetration.

(e) Pruritis ani

The commonest cause of perianal itching is infestation with threadworms (pinworms, *enterobius vermicularis*), whose adult females deposit ova on the perianal skin. You can detect these by putting transparent sticking tape (sticky side up) round a tongue depressor and placing this against the anus, but bathing in the morning will wash off ova laid in the night! Scratching transmits ova to the fingers, and hence to food and to the mouth.

All members of a household require treatment, which is fortunately very simple: mebendazole 5mg/kg stat, or albendazole 7mg/kg stat, either repeated after 2wks, or piperazine 30mg/kg, stirred in water or milk, od for 2 consecutive days.

Look for other parasitic skin infections, especially scabies and body lice, as well as dermatoses such as eczema, seborrhoeic intertrigo, and lichen planus.

Examine the perineum for excessive moisture, skin excoriation, faecal soiling, and purulent discharge. Ask about the use of ointments, perfumed powders, sprays, creams and occasionally foods which can cause an allergic dermatitis. Candidiasis may be present, especially in HIV+ve patients, or those treated with a multitude of broad-spectrum antibiotics.

Check for jaundice, diabetes and any signs of cancer.

Occasionally, you may see a raised, erythematous, straight or snaking subcutaneous ridge (cutaneous myiasis) due to deposition of fly or nematode larvae through the hair follicles when in contact with contaminated soil. The larva migrates after an incubation period of 2-10 days, especially at night, at speeds of up to 2cm/day. Albendazole 7mg/kg for 3days is curative; the larvae can be extracted with a sharp needle followed by douching the wound with 15% chloroform in vegetable oil.

You should, however, do a proctoscopy and look for haemorrhoids (26.9) or a polypoid lesion. Sometimes you won't find anything, and may suspect a psychological problem. First exclude diabetes, diarrhoeal and other dermatological diseases, and allergies to soaps. Use miconazole 2% with a low-dose hydrocortisone (1%) as a cream, with an antihistamine (chlorphenamine or promethazine); this often abolishes a vicious cycle of itching, scratching, and irritation.

(f) Imperforate anus

Rectal agenesis can occur with or without fistulation into the vagina or bladder (33.6); always remember to examine for an anal opening on the neonate.

(g) Faecal incontinence

Involuntary leakage from the anus is common in children, after traumatic childbirth with a 3rd-degree perineal tear, the debilitated, homosexuals, paraplegics and the elderly. It happens often after a cerebrovascular accident; it is important to know whether the incontinence is because the rectum is loaded with faeces and only loose stool comes past, or whether the anal sphincter mechanism is not working. *So always do a rectal examination!* Check if there is a fistula (26.3).

If you find hard obstructed faeces, do a manual evacuation under ketamine. Then use daily laxatives, and make sure he mobilizes (if possible) and eats a high-fibre diet.

If there is a 3rd-degree post-partum perineal tear (21.16), effect a repair taking care to look for the retracted sphincter muscle.

If you find a loose anal sphincter, the patient may benefit from a postanal repair. This is surgery for the expert. Check first for HIV.

If there is no sphincter tone, you can train the rectum to evacuate by stimulating the medial thighs. You need to start with a bowel wash-out and then establish a regular bowel routine. This needs patient persistence, and a regular dietary regime and laxative use.

27 Urology

27.1 Equipment for urology

Disease of the urinary tract can be very distressing, so much so that some sufferer in the Middle Ages is said to have prayed, “O Lord, take me home but *not* through my bladder.” Urological cases are often smelly and septic. Because it is usually considered sensible to start making rounds at the 'clean' end, you and your staff are likely to arrive tired at the urological patients, and can easily neglect them.

You should be able to treat acute, chronic, and ‘acute on chronic’ retention (27.6,19), and urethral strictures, whether they are passable (27.9) or not (27.10). You may be able to remove the prostate (27.20), and take stones from the bladder in adults (27.16) and children (27.17) and from the urethra (27.18) and ureter (27.15), but not so easily from the kidneys. You may alleviate carcinoma of the penis (27.33) and prostate (27.22), but not so readily of the bladder (27.5). You probably cannot do much about the congenital anomalies of the urinary tract, except to remember that the absence of a kidney is most important.

The most useful urological investigations are urinalysis, microscopy and culture, and a blood urea, followed, when necessary, by ultrasound, an intravenous urogram and cystoscopy.

The commonest urological procedure is to pass a catheter to let the urine flow out of the bladder. Catheters are graduated according to the Charrière gauge, which is their circumference in mm. If there are 2 numbers, for example, Ch18/22, the smaller one refers to the circumference of the tip, and the larger one to the circumference of the shaft. Think of catheters in three sizes: Ch8 or 10 for simple drainage, Ch14 or 16 Foley self-retaining catheters for the relief of retention, and large Ch20 or 24 catheters for post-operative drainage or evacuating blood clots. *Don’t use the wrong sizes for the wrong indications!* There are many different types of catheter, but in practice the Foley and the Jacques (or Nelaton) types are usually the most readily available. The latter is similar to the Foley, but has no balloon to inflate to keep the catheter in place.

27.2 Catheters and how to pass them

Before you pass a catheter, think for a moment about what function you want it to serve. If you are going to drain urine from a healthy patient who cannot pass urine after a hernia operation, use a soft catheter. If it has only to let out urine, its lumen can be narrow. If you expect bleeding, and want to irrigate a bleeding bladder, so as to dilute the blood in it and prevent it clotting, use a catheter with an additional irrigating channel.

If you need to suck out clots, choose a large catheter made of stiff material which will not collapse. If it is only to monitor urine output, and the patient is able to pass urine, make sure that the amounts passed are correctly recorded, and then ask yourself if the catheter is really necessary.

An indwelling catheter has usually to stay in place for ≥10 days, so:
(1) *Avoid red rubber*, and use latex, plastic, or ideally silicone, because these will be less irritant.
(2) *Be sure that it does not fit so tightly* that it blocks the mouths of the paraurethral glands: there must be plenty of room beside it for their secretions to ooze out.
(3) It must be soft, because a stiff tight-fitting catheter can press on the mucosa of the urethra at the external sphincter or the penoscrotal angle, and cause a pressure sore, and finally a stricture. So use the narrowest, softest catheter which will serve your purpose, and remove it as soon as you can. *Don’t use a catheter for incontinence in the male:* use a Paul’s tubing (a condom catheter, 27-3).
Finally, remember that passing a catheter should be a sterile procedure; *you can so easily infect a patient and cause avoidable misery.*

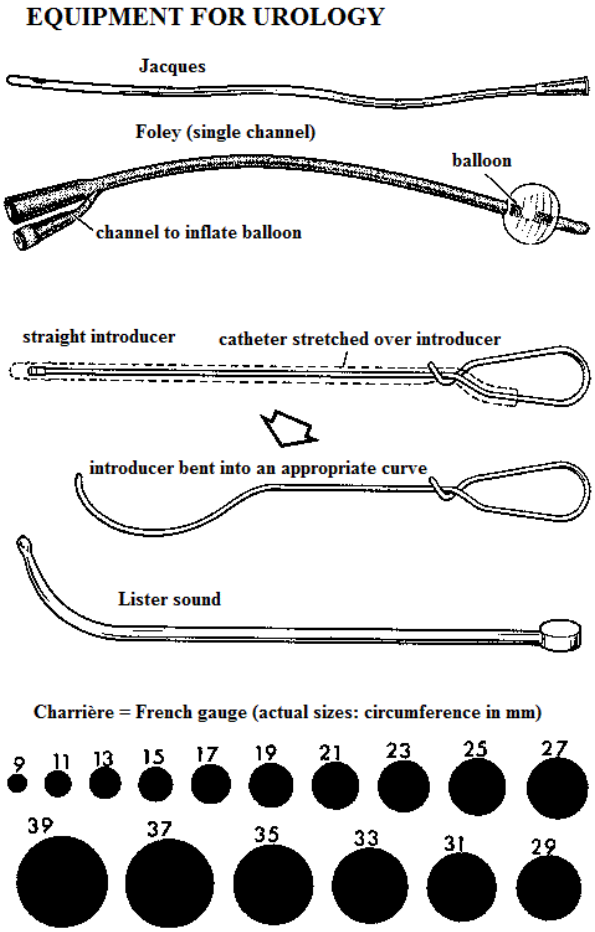

EQUIPMENT FOR UROLOGY
Jacques
Foley (single channel)
balloon
channel to inflate balloon
straight introducer
catheter stretched over introducer
introducer bent into an appropriate curve
Lister sound
Charrière = French gauge (actual sizes: circumference in mm)
9 11 13 15 17 19 21 23 25 27
39 37 35 33 31 29

Fig. 27-1 EQUIPMENT FOR UROLOGY. The round black circles show the actual size of each catheter. (Diameter *in mm* = Ch gauge ÷ π)

USE THE NARROWEST, SOFTEST CATHETER; AND REMOVE IT AS SOON AS YOU CAN

CATHETERS, Foley, latex rubber, self-retaining, double channel, 30ml balloon, (a) Ch14, (b) Ch16, (c) Ch18, (d) Ch20, (e) Ch22. These have a balloon at the end which you can blow up through the second channel, and so keep the catheter in the bladder.
CATHETERS, Foley, silicone rubber, self-retaining, double-channel, 30ml balloon, (a) Ch14, (b) Ch22. Keep these expensive silicone Foley catheters for long-term drainage and stricture management.
CATHETER, plastic, Foley balloon, three-channel for continuous irrigation, 75ml balloon, straight tip, Ch22. This catheter holds itself in place and allows you to irrigate the bladder. One channel is for the balloon, one is for irrigating fluid going into the bladder, and one for fluid and urine coming out. Use this for pervesical prostatectomy (27.20).
CATHETER INTRODUCERS. Keep one of these straight for introducing suprapubic catheters, and bend the other one into a smooth curve for introducing urethral catheters. If you use the same introducer for both purposes and keep on bending and unbending it, it will soon become so kinked that you cannot withdraw it. *An introducer can be a dangerous instrument in the urethra, so use it with great care, and only when absolutely necessary.*
SPIGOTS, plastic, for catheters (a) small, (b) large. If a patient has a spigot in the indwelling catheter, he can walk about with it in, and remove it to drain the bladder. If you don't have plastic spigots, make wooden ones.
BOUGIES, neoplex, Porges, filiform, (a) smaller sizes olive-tipped, (b) larger sizes plain, sizes Ch1-20, (c) follower to fit. Use these for dilating strictures, for which they have many advantages over metal bougies except that they are much less durable. You can control their passage through the anterior urethra more easily, they follow the curve of the posterior urethra, and you will less easily cause false passages, even with small sizes. Cheatle's forceps can crush plastic bougies when they are hot, so tie them into bundles, and handle them only with tapes. Lift them out of the sterilizer and put them into cold boiled water. They will then be the right consistency for use.
SOUNDS, metal, large, blunt, straight, plain-end, Powell's, sizes Ch12-20. Historically, a 'sound' refers to a metal instrument used to locate stones in the bladder by the sound of the instrument hitting the stone. The terms 'sound' and 'bougie' are now used synonymously. If you are going to use a metal sound, Powell's straight sounds are the safest – a large one is much too large to be led down a false passage.
SOUNDS, metal, Otis-Clutton urethral, curved with olive ends, Ch9/14, Ch11/15, Ch12/17, Ch14/18, Ch15/20, Ch17/21, and Ch18/23. Use these for dilating a strictured urethra. The set is deliberately incomplete, because small sounds are dangerous, and can too easily cause a false passage.
SYRINGE, bladder wash-out 50ml, plastic, boilable.
RETRACTOR, Thomson-Walker, with 2 pairs of detachable abdominal wall blades, and a long blade for posterior wall. Use this for a suprapubic prostatectomy. It is designed to reach into the floor of the bladder.
URINE BAGS, simple pattern, with drainage outlet at the bottom, plastic, reusable. Although these bags are intended to be disposable, you can boil them and reuse them many times. Put 20ml of a disinfectant, *e.g.* cetrimide, into the bag to help prevent infection, especially in longstanding cases. If you don't have any urine bags, you can wash blood-giving bags and blood-giving sets, cut them, and adapt them.
CLAMP, penile with rachet. If a patient is incontinent, he may find a penile clamp useful. You can also use one to retain local anaesthetic before you pass a catheter.
BIOPSY NEEDLE, Travenol 'Tru-cut' (TRAV) code 2N2704. This is for the operative treatment of priapism (27.32). Several punctures with a large ordinary needle or a sharp trocar may be effective.

(a) Passing a catheter on a male adult (GRADE 1.1)

EQUIPMENT. 2% lidocaine gel, preferably with 1% chlorhexidine; an antiseptic suitable for the scrotum; the right catheter; a penile clamp to retain the anaesthetic; receivers, a sterile bottle in which to send urine for culture; a syringe to blow up the Foley balloon; a sterile connecting tube; a bag to receive the urine.

METHOD
If the patient is in severe pain, administer pethidine. This may help urine pass spontaneously. Explain what you are going to do. Make sure you have help. Arrange the patient sitting or lying comfortably in a good light with the legs apart, hands on the chest (*not behind the head*) and a waterproof sheet under the bottom. Expose the urethral meatus, and clean the glans and surrounding tissues thoroughly with antiseptic solution.

If you cannot expose the urethral meatus, make sure you pull back the foreskin completely, and you remove any sebaceous smegma properly. If the foreskin is very oedematous and you cannot retract it, use a McGill or sponge-holding forceps (4-4) to prise the foreskin apart, and then gently pull the glans forward, whilst at the same time pulling back on the foreskin. You can also reduce the oedema in the foreskin rapidly by injecting hyaluronidase.

Remember you may find the urethral opening in an abnormal position (proximally and ventrally in hypospadias (33.9), or dorsally in epispadias).

When the penis is buried in a large scrotal swelling (hydrocoele or inguinoscrotal hernia), it can be very difficult to grasp: use a proctoscope or vaginal speculum to push against the scrotum around the penis, thus allowing the urethral meatus to emerge.

Using a syringe without a needle, put 10ml of lidocaine gel into the urethra, and get the patient to keep it there for 4mins; otherwise use a penile clamp, or light pressure from your own finger and thumb.
CAUTION!
Half the trouble in passing a catheter comes from not allowing time for LA to act.

Scrub and put on sterile gloves. Drape the patient with sterile towels. Clean under the foreskin thoroughly. Start with a Ch14 or 16 Foley catheter (smaller latex ones may be too supple). Hold the penis straight upwards to straighten out the urethra (27-2A,B). Take the catheter in your other gloved hand. *Don't touch either your skin or the patient's skin*. Push the catheter gently into the meatus, and down the urethra, while keeping the penis upright.

If it sticks at the junction of the penis and scrotum, there may be a stricture, because this is the common site.

If it sticks at the external sphincter (27-2C), wait, be gentle, and allow it to relax. If it remains taut, you will never pass the catheter. Force is dangerous. Be slow, gentle, and crafty. Suggest breathing in and out, and pretending to pass urine. If you can catch the sphincter 'off-guard', the catheter will slip in.

If it sticks in the posterior urethra, the prostate may be enlarged. Put your non-dominant finger in the rectum, and press on it. You may find that the catheter will now pass onwards.
CAUTION! Never use force.

If it still does not pass:
(1) It may be too large (try a smaller one).
(2) The sphincter may not be relaxed (27-2C) because the patient is frightened (try sedation).
(3) The urethra may not be properly anaesthetized (try introducing 5-10ml more lidocaine, with lubricant jelly).
(4) The catheter may have caught in mucosal pockets in the urethra: you can easily make these into a false passage, if you are not careful.
(5) There may be a large prostate, which distorts the urethra, and prevents the catheter following it into the bladder. Try passing a small Ch12 catheter folded back on itself (27-2E); if this fails, perform a suprapubic catheterization (27-9).

When the Y-connection of the catheter reaches the urethral meatus *and* you see urine coming out, *then* you can inflate the balloon, *not before! Don't fill it to its maximum capacity*: 10ml is adequate to stop the catheter falling out. *Remember to pull back the foreskin over the glans penis to prevent paraphimosis!*

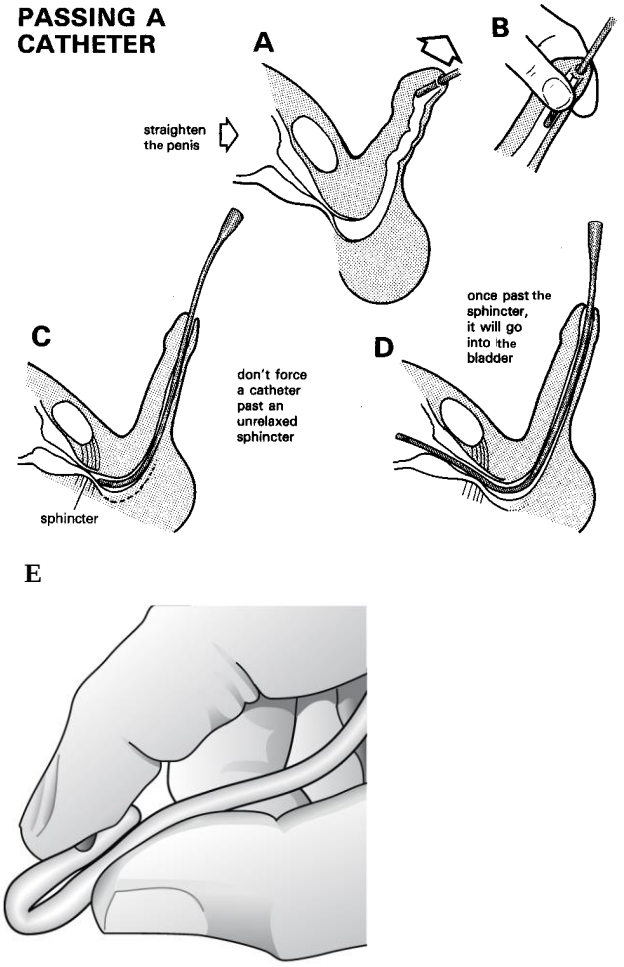


Fig. 27-2 PASSING A CATHETER.
A-B, straighten out the urethra to remove its kinks. C, if the mucosa over the external sphincter is not well anaesthetized, it may go into spasm: *never force a catheter past an unrelaxed sphincter*. D, when it is past the relaxed sphincter, it will find its way into the bladder, provided it is flexible and well-lubricated. E, passing a smaller Ch12 catheter folded back on itself: being more rigid, it may well pass the prostate and once in the bladder open itself out spontaneously.
A-D, after Blandy J. Operative Urology, Blackwell 1978 Fig 2.54-6, with kind permission. E, after Buddha S. Complication of urethral catheterisation. Lancet 2005;365,909.

If the Y-connection does not reach the meatus or urine does not come out, *don't inflate the balloon*: it may still be in the urethra rather than the bladder, and inflating it may cause urethral damage and ultimately stricture formation. There is no harm in strapping or suturing the catheter in place, if you see no urine but are fairly sure the catheter is in the bladder (*e.g.* in renal failure), and waiting. If you can, check with ultrasound to see if the bladder is really full. If you are sure the catheter is in the bladder, it might be blocked with debris or blood: try flushing it gently with a little sterile water. *If after an hour, there is still no urine, and the bladder remains full, the catheter cannot be in the correct position.* Use ultrasound to check where it is (38.2i).

CAUTION! Ask your nurses to empty the urine bag *before* it is full and at least every 24h, aseptically and without getting urine organisms on their skin. *Don't let a full bag pull on the inflated balloon:* it may cause pressure necrosis of the posterior urethra. Be sure your nurses chart the urine output meticulously.

(b) Passing a catheter on a female adult (GRADE 1.1)

EQUIPMENT. An antiseptic suitable for the vulva; the right selection of catheters; receivers, a sterile bottle in which to send urine for culture; a syringe to blow up the Foley balloon; a sterile connecting tube; a bag to receive the urine. *Don't use a metal catheter!*

METHOD. If the patient is in severe pain, administer pethidine. This may help urine pass spontaneously. Explain what you are going to do. Make sure you have help. Arrange the patient sitting or lying comfortably in a good light with the legs apart, hands on the chest (*not behind the head*) and a waterproof sheet under the buttocks.

CAUTION! Half the trouble in passing a catheter comes from not properly visualizing the urethral orifice, which is situated below the clitoris above the vagina. It may be ectopically placed more posteriorly, especially in elderly women. If you have difficulty locating the orifice, ask the patient to cough, whereupon some drops of urine may come out spontaneously. Make sure you have good lighting, and the thighs are well abducted. Drape the patient with sterile towels. Clean the vulva thoroughly. Start with a Ch14 or 16 Foley catheter (smaller latex ones may be too supple). Take the catheter in your other gloved hand. *Don't touch either your skin or the patient's skin*. Push the catheter gently into the urethra, until it will go no further. During delivery, in a female, you may have difficulty pushing the catheter inside: insert 2 fingers of your left hand into the vagina between the foetal head and the symphysis pubis. Using one finger on either side of the urethra, gently guide the catheter into the bladder. Remember the female urethra is <4cm long.
In some cases, *e.g.* VVF repair, it is best to let a catheter drain freely (21.18).

If the urethra is stenosed, this may be due to mutilating surgery or damage from childbirth associated with a vesico-vaginal fistula (21.18). Simple dilation is usually possible (27.9).

If you cannot expose the urethra, especially in an elderly woman with atrophy of the vagina, mount a Ch16 catheter on an introducer, and gently pass this along the anterior wall of the vagina till it drops effortlessly into the bladder. You may need to palpate the urethral orifice with a finger and guide the introducer accordingly.

(c) Difficulties with catheters

If the catheter leaks, it probably has encrustations partially blocking its channel; *don't try to insert a larger size to 'fill' the urethra!* Try irrigating the catheter with sterile water, and if this fails, change the catheter.

If the catheter blocks, especially with clot after prostatectomy, this is usually because of inadequate irrigation. Try to dislodge the clot by instilling heparinized water with a bladder syringe, and sucking out the bloody urine and clots. If this does not work and water can be instilled but not withdrawn, thus making the patient more and more uncomfortable, deflate the catheter balloon and push the catheter in, wriggling it about; this might cause the clot in the eye of the catheter to dislodge. Otherwise, replace the catheter with a larger size. This might be difficult, and require GA; make sure the patient gets a proper bladder wash-out in this case.

If the catheter balloon will not deflate, cut the catheter across, and leave it for 6h to empty. It will often deflate by itself. If it does not, inject 10ml mineral oil up the balloon channel, and wait 10mins. Repeat this if necessary and check if the balloon has deflated. Wash out the bladder with 200ml sterile water, to remove the oil and any balloon fragments which may have been left behind. You can also use ether, chloroform or acetone, but these irritate the bladder. Alternatively, palpate the balloon *per rectum*, and direct a needle guided by your finger to burst it: this is potentially hazardous, so wear thick gloves and administer gentamicin! If you have ultrasound, it is easy to guide a needle into the balloon suprapubically to rupture it. If not, you can pass a well-lubricated catheter introducer through the urine drainage channel and thereby push the end of the catheter up against the anterior abdominal wall: when the balloon is palpable or visible, rupture it with a needle.

N.B. This will only work in thin patients.

If you cannot remove an indwelling catheter, even though you have deflated the balloon, you have probably left it in much too long, so that crusts have anchored it to the mucosa. *If you pull it out firmly, you will damage the mucosa and may rupture the urethra*. You may have to open the bladder to remove the catheter; if the reason the patient has a catheter in the first place is prostatic enlargement, use this opportunity to perform a prostatectomy (27.20).

N.B. A latex catheter becomes encrusted in 3-4wks, and a silicone one in 3-4 months. Be safe, and change a latex one every 4wks and a silicone one every 12wks.

If you have inflated the balloon in the urethra, deflate it and remove the catheter; *don't attempt re-catheterization via the urethra*.

If the patient develops urinary retention, insert a suprapubic catheter (27.7). If the balloon will not deflate, palpate it through the penoscrotal skin, and rupture it with a needle.

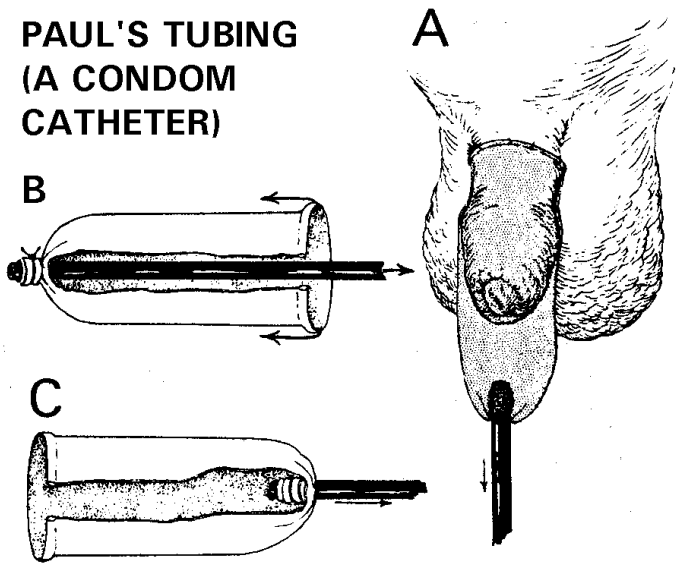


Fig. 27-3 PAUL'S TUBING (CONDOM CATHETER).
If a patient is incontinent, you will find this very useful. You may not have a ready-made catheter, but can fashion one easily like this. *Kindly contributed by Jack Lange.*

27.3 Cystoscopy

(a) Cystoscopy (GRADE 1.5)
Looking at the bladder with a cystoscope is:
(1) Often the best way to know what is going on inside.
(2) Usually more useful than an ultrasound.
(3) Particularly useful in areas where *Schistosoma haematobium* is endemic, because it is the most practical way of diagnosing the cancer of the bladder that commonly complicates this disease, and which also causes haematuria (27.4).
For this you only need the simplest instrument, without provision for catheterizing the ureters.

Cystoscopy is an acquired skill, even with equipment using a fibre-optic light source rather than a solid rod lens system. The problems are:
(1) to get the instrument in (it is best to do this under direct vision, so as to avoid causing damage),
(2) to have a good enough instrument to give you a diagnostic view, and
(3) to know what the normal looks like.

Flexible fibre-optic instruments are not necessarily easier to use.

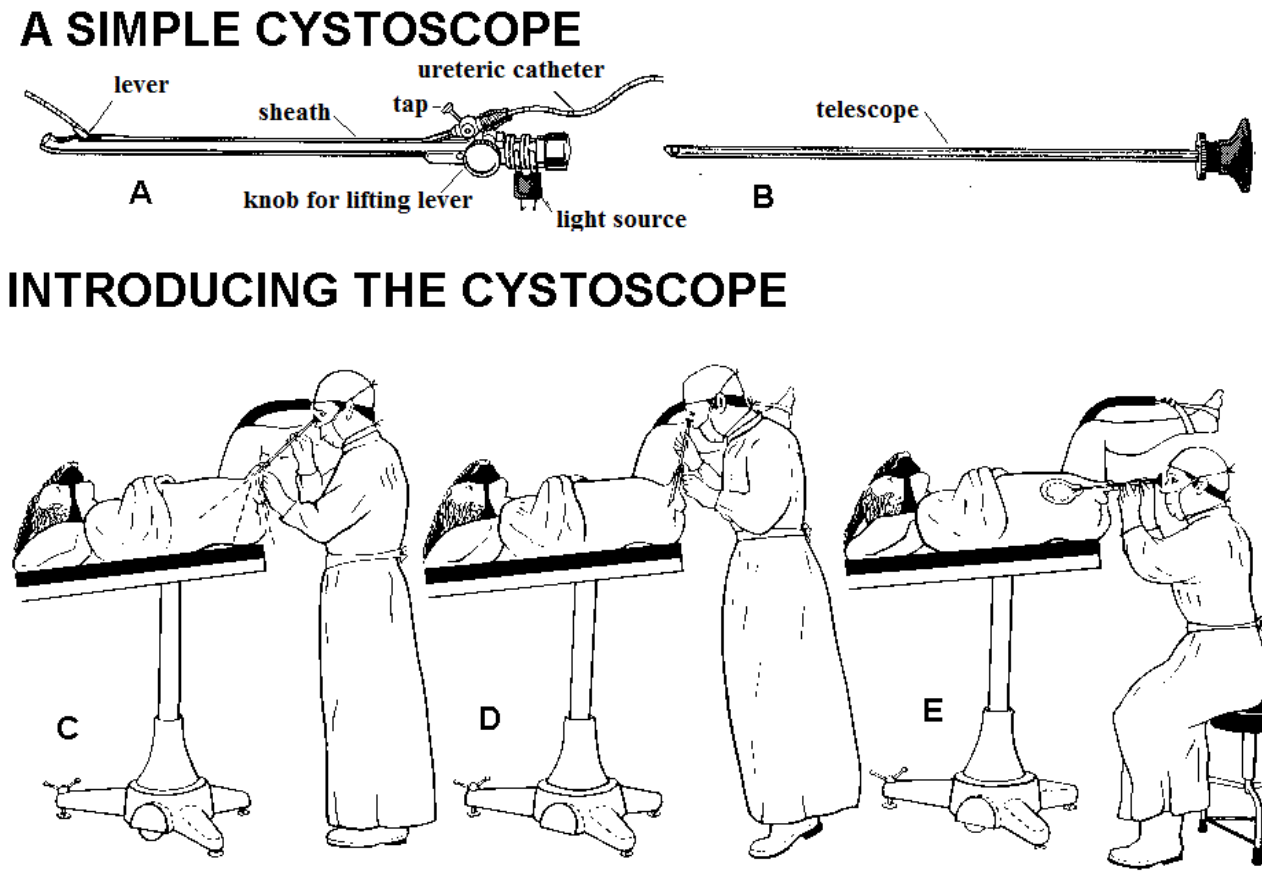


Fig. 27-4 SIMPLE CYSTOSCOPY.
A, sheath. B, telescope. (The Albarran lever is for manipulating the ureteric catheter.) C, introduce the cystoscope under direct vision of the anterior urethra, D, the bulbar urethra, and E, the prostatic urethra. ***(N.B. The right leg has been removed for clarity!)***

CYSTOSCOPE. There are more sophisticated operating and simple viewing cystoscopes. All need an irrigation channel.
The urethroscope has 0º viewing angle (to look straight ahead), and a viewing cystoscope 30-70º (to look around).
Methods of irrigation differ. Some cystoscopes have a tap with two positions, some have two taps, and in others you have to remove the telescope and fix a tap ('the faucet') in the hole where the telescope was.
SYRINGE, bladder, Barrington's metal. Use this to wash out the bladder during cystoscopy.
CAN, douche, metal, 3L, with rubber tubing.

INDICATIONS
(1) Urinary retention, or difficult micturition, particularly as a prelude to prostatectomy. If the patient has an enlarged prostate, cystoscopy may precipitate acute retention, so do it as the first stage of a prostatectomy (27.20).
(2) Haematuria in a patient >30yrs in areas where *Schistosoma haematobium* is highly endemic (27.5).
(3) To diagnose *schistosomiasis* when it is strongly suspected clinically, but you cannot find ova in the urine.
(4) Recurrent urinary infection.
(5) Investigation of bladder stones.

CONTRAINDICATIONS
Acute cystitis, until you have controlled the infection.

TESTING THE CYSTOSCOPE
Do this before you sterilize it. Look down the telescope; the image should be clear. If it is misty, there is dirt on the lens; clean with spirit-soaked swabs. If it is still misty, water has probably entered the telescope, so return it to the makers or an agent for repair. If a crescentic part of the visual field is cut off, the telescope has been bent. If this only happens after you have inserted the sheath, it is the sheath which is bent.

STERILIZATION
Even though you may be able to put your cystoscope (if it is an old-fashioned sort) in an autoclave, it will last longer if you use an antiseptic solution. Keep it in its box until you want to use it.
Remove the compression ring and valve, and immerse it in glutaraldehyde solution, 1% chlorhexidine, $^{1}/_{1000}$ mercury oxycyanide, or $^{1}/_{80}$ phenol for 10mins; immersion will kill all bacteria capable of infecting the bladder. Rinse it thoroughly in sterile water, and place it on a sterile towel. After use, rinse it in water, and dry it with swabs soaked in spirit.

ANAESTHESIA
You may be able to examine a woman as an outpatient without any anaesthesia if you are gentle and use a lubricated instrument, unless she has a painful stricture of her external meatus, or a very irritable bladder.
GA will be easier in the male and if you are inexperienced, or he has carcinoma of the bladder (27.5) or tuberculosis (16.7).

If you use LA, clean the glans penis with cetrimide, and use the nozzle of a tube of 2% lidocaine jelly to inject 5g down the urethra. Apply a penile clamp proximal to the glans. After 4mins inject a further 5g, and reapply the clamp. Massage the penile urethra, so as to squeeze the jelly into the posterior urethra. Wait a further 10mins before performing a cystoscopy.

PREPARATION
Use the semi-lithotomy position: flex the hips to only 75º and abduct them 30-45º, so as to leave the buttocks further up the table than the poles. *Don't use the full lithotomy position*. To provide fluid for irrigation, you can use autoclaved water in a receptacle maximally 60cm above the patient.

INTRODUCING THE CYSTOSCOPE
Introduce the cystoscope into its sheath, and lubricate the outside with petroleum or lidocaine jelly. It is best to start with a 0º urethroscope or 30º cystoscope to visualize the urethra.

In a female, you will have no difficulty, unless her meatus is stenosed. If so, dilate it with sounds.

Clean the glans penis of a man thoroughly under the foreskin and hold the penis vertically with your left hand. Introduce the cystoscope gently into the urethra (27-4C) viewing it directly, and stretch the penis along it, as it descends under its own weight. Look at the bulbar urethra (27-4D), and then the prostatic urethra (27-4E). When the cystoscope tip lies against the triangular ligament, swing the eyepiece down between the thighs with a circular motion, so that the beak of the cystoscope faces downwards, and the telescope will slip directly into the bladder.

If the beak sticks in the external urethra, depress the eyepiece further and it will probably slip in: *never try to push it in by force*. Guide it under direct vision. If it still will not pass, put the index finger of your free hand in the rectum or on the perineum and guide it in that way: this is seldom necessary. *Remember then, though, to change gloves!* If the beak is in the bladder, the cystoscope will rotate freely.

WASHING OUT THE BLADDER
Remove the telescope from its sheath and collect the urine which comes out. If it is hazy, send it for microscopy and culture. Crystal-clear urine will probably be sterile.

Fill a bladder syringe with water, and expel any air by holding its nozzle upwards, and depressing the plunger. Then squirt some of the water on to your own hand, to make sure that it is not too hot. Wash out the bladder by injecting 50ml at a time, until the washings are clear. Alternatively, use irrigation.

INSPECTION. Distend the bladder with 250ml of water. (A normal bladder holds 250-400ml.) You can also use air: this often gives a better view.

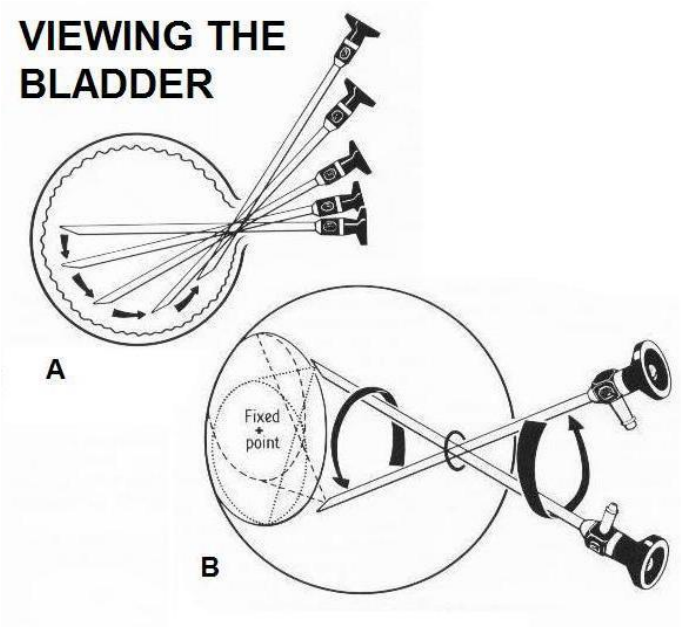


Fig. 27-5 VIEWING THE BLADDER.
A, swing the cystoscope in an arc to examine the bladder mucosa. B, rotate the cystoscope to examine the fundus using the air bubble at the top as a reference point.
After Clark P. Operations in Urology Churchill Livingstone 1985 p.19 Figs1.29,30.

N.B. Advanced carcinoma, severe *schistosomiasis* or tuberculosis may make a bladder very small, in which case you may cause considerable bleeding if you try to overfill it with >50ml.

Insert the telescope and look around (27-5). Examine the bladder systematically, starting with the fenestra (window), looking downwards towards the base of the bladder. Note the size of the median lobe of the prostate as you enter the bladder (it looks like a 'termite hill', 27-7). Observe the inter-ureteric bar (27-7A). This is a ridge of tissue between the two ureteric orifices (27-19H). It is a useful landmark, but it is sometimes not very conspicuous. Another landmark is the small air bubble which is always present in the dome (top) of the bladder. Return to the inter-ureteric bar, and look all round the side walls and roof of the bladder.

Turn the cystoscope through 360º, so as to examine a circular strip of bladder wall. Then push it further into the fundus, withdraw it 2cm and look around 360º again. Find the ureteric orifices by finding the inter-ureteric bar, and tracing it laterally.

When you see an orifice, the cystoscope must be in either the 5o'clock or the 7o'clock position. Now you can thread the ureteric catheter (if you have one) into the orifice, by adjusting the Albarran lever (27-4A). Depress the eyepiece to look at the anterior wall of the bladder. This may be impossible to see in a man, unless he is fully relaxed under GA.

The mucosa of a normal bladder is a yellow sandy colour, and has fine branching vessels under it. If the fluid in the bladder is bloody, the mucosa may look pink: *don't confuse this with cystitis*. A normal trigone, the area between the urethral and ureteric orifices (27-7C), is pink and vascular.

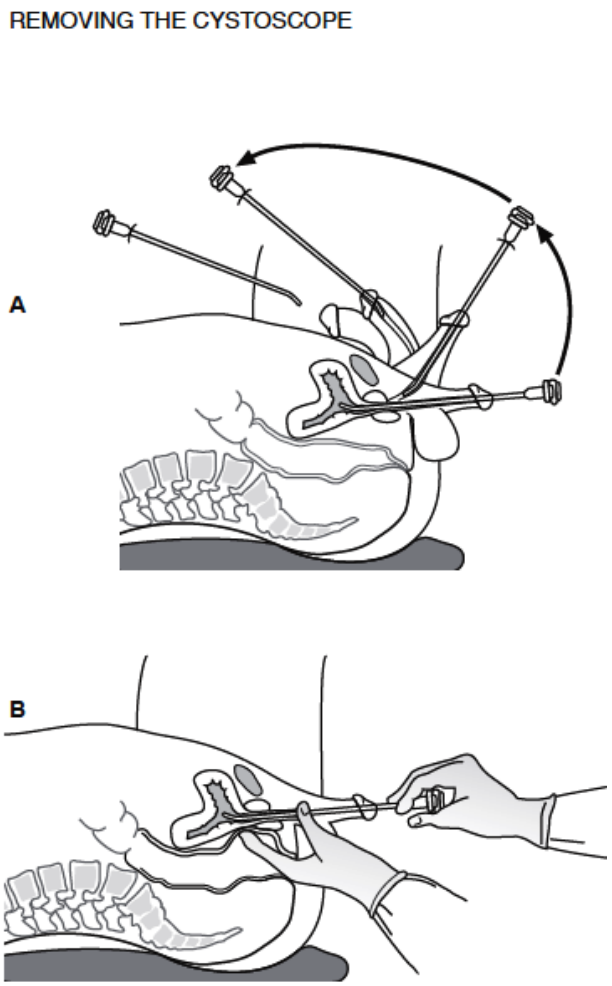


Fig. 27-6 REMOVING THE CYSTOSCOPE.
A, remove the telescope from the urethra over the pubis. B, examine the prostate rectally against the cystoscope.
After Clark P. Operations in Urology Churchill Livingstone 1985 p.21 Figs1.33,34.

Finally, partly withdraw the cystoscope (27-6) and examine the trigone and the internal urinary meatus. You should be able to see everything except the base when it is obscured by a very large median lobe. When this is very large, you may not see the ureteric orifices either. Palpate the prostate *per rectum* with your finger pressing against the cystoscope.

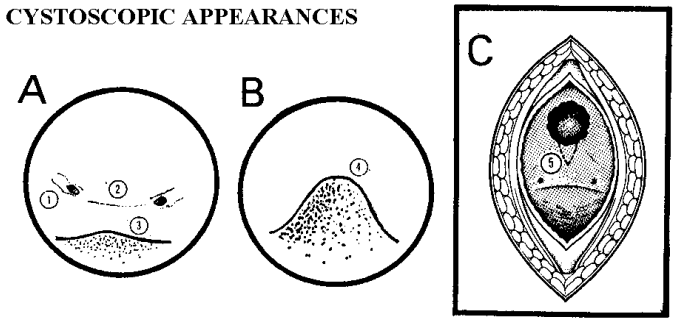


Fig. 27-7 CYSTOSCOPIC APPEARANCES.
A, the base of the bladder, with the ureteric openings (1), inter-ureteric bar (2) and middle lobe as a hardly visible swelling (3). B, in benign prostatic hypertrophy the middle lobe (4) projects into the bladder to obscure the inter-ureteric bar and the ureteric openings. C, position of the wedge (5) excision in bladder neck stenosis (27.21).

If you see nothing to begin with:
(1) The beak of the cystoscope may still be in the urethra.
(2) The light may have gone out.
(3) You may have inserted the telescope incorrectly. The small pin in the eyepiece should fit into the expanded end of the valve collar. Try twisting the compression ring a little.
(4) There may be blood or clot on the objective.
(5) If you see nothing but a 'red out', you may have failed to run fluid into the bladder, or your instrument is right up against the bladder wall.
(6) If the bladder holds <50ml, *e.g.* because of advanced carcinoma, severe *schistosomiasis*, or tuberculosis: beware there may be bleeding if you fill the bladder too full!

In **bladder outflow obstruction**, look for:
(1) Enlargement of the lateral lobes of the prostate (27.19), which will make the prostatic urethra appear as a cleft before you see into the bladder. The median lobe will project from the posterior aspect of his bladder like 'a termite hill', and may make it difficult to see the ureteric orifices (27-7B).
(2) Thickened criss-cross pattern of the bladder musculature (trabeculation), showing that its outflow is obstructed. In between you may see saccules, which enlarge to form diverticula. These are usually above and lateral to the ureteric orifices, with radiating folds around their openings. You may be able to get the beak of your cystoscope inside one. Diverticula rarely matter; once any outflow obstruction has been relieved, diverticulectomy is seldom necessary.

N.B. Dyskinesia (bladder neck dysfunction, 27.21) causes retention of urine but cannot be diagnosed cystoscopically. But, suspect **bladder neck stenosis** if:
(1) The bladder is obviously obstructed, as shown by muscle hypertrophy, residual urine, and perhaps diverticula.
(2) The prostate shows no bulging, there is no urethral stricture and no CNS disease.
(3) The bladder neck is tight and looks like a bar (27-19K) over which you have to pass the cystoscope.

27.4 Haematuria

Blood in the urine can be the result of almost any pathology at any level, but is much more likely to be coming from the bladder than from the upper urinary tract. Bleeding can be the result of injury, bacterial infection, parasitic infestation, stones, or neoplasia.

If *schistosomiasis* is endemic in your district, frank haematuria in someone >30yrs has a 25% chance of being due to a carcinoma of the bladder, so be sure to check this by cystoscopy. Frank (visible, macroscopic) haematuria <30yrs is much more likely to be due to urinary infection or *schistosoma* than carcinoma.

The other important cause is prostatic hypertrophy and HIV-related cystitis. Other, rarer causes are renal tuberculosis, renal carcinoma, radiotherapy, ketamine abuse and vascular abnormalities.

Because cystoscopy is so necessary in the diagnosis of malignancy in areas where *S. haematobium* is endemic (27.5), it should be the first investigation after Hb, urea, urine microscopy and culture and ultrasound; *an IV urogram is not usually helpful*.

Bladder stones seldom cause macroscopic haematuria. Ureteric stones usually present with renal colic and microscopic haematuria.

Haematuria in a male cannot be simply attributed to a urinary tract infection: there is *always another* underlying cause.

Haematuria in a woman may arise from the urine being contaminated by a menstrual flow.

Confirm that there really is blood in the patient's urine by examining it microscopically.

(a) The 2-glass test. Ask the patient to pass the urine into 2 containers, and watch him do it:
(1) A constant ooze from the urethra, indicating a lesion distal to the external sphincter.
(2) Initial or terminal haematuria, indicating a local lesion of the bladder or prostate. Terminal haematuria is typical of *schistosomiasis*.
(3) Total haematuria which is equal in both glasses, and may contain worm-like clots, indicates bleeding from the upper urinary tract or bladder; it is common in *schistosomiasis* and carcinoma of the bladder.

(b) Cystoscopy is usually best done after bleeding stops. The exceptions are:
(1) A bladder full of clot which needs immediate evacuation.
(2) Recurrent haematuria when you cannot find a cause, and when you would like to know from which kidney blood is coming: this is rare.

(c) Ultrasound (38.2i) will show if there is a foreign body or tumour in the bladder.

(d) Radiographs. A plain radiograph (in oblique orientation) may show calcifications in the kidney or line of the ureter. You can confirm these with an intravenous urethrogram (38.1).

CAUTION! Occasionally you may see *red urine from eating excessive amounts of beetroot, chewing huge quantities of betel leaf, or in porphyria.* Even more rarely, a patient may have deliberately dripped blood into a urine sample!

27.5 Bladder carcinoma

Histologically, there are two main kinds of carcinoma in the bladder:
(a) Squamous carcinoma, related to exposure to *Schistosoma haematobium.* In areas where this is endemic (especially along the Nile), it is often the most common cancer found.
(b) Transitional carcinoma, which has a 75% chance of being papillary, and of such low-grade malignancy that it can be controlled with diathermy. Anaplastic degeneration indicates end-stage disease.

In non-schistosomal areas, most bladder tumours are transitional, papillary, and of low-grade malignancy, and associated with simultaneous tumours of the ureter or renal pelvis. There is a high risk of bladder cancer where the water has >350µg/L of arsenic (*e.g.* in Bangladesh, India and parts of Central and South America).

In areas where *S. haematobium* is endemic, only about 5% are like this, 10% are anaplastic, and 85% are squamous. Of these, most are either sessile or ulcer-cancers, both of which grow rapidly, and penetrate early into the muscle of the bladder or into the paravesical tissues. They may also have obstructed the ureters or the urethra by the time they present.

The patient is usually 35-60yrs and has a 2:1 chance of being male, complaining of:
(1) haematuria, which is initially painless,
(2) passage of white sludge, or small pieces of white material (necrotic tumour),
(3) increased frequency of micturition, as the result of irritability, infection, and a small bladder,
(4) a suprapubic mass,
(5) retention of urine (5%), as a result of the tumour obstructing the urethra.

In areas where *S. haematobium* is not endemic, you should cystoscope all patients with haematuria. This is impractical in endemic areas, because so many patients pass bloody urine.

Macroscopic haematuria, due to *S. haematobium* alone, becomes less common as age advances, because of the fibrosis round the ova, so that by the time someone is 30yrs, there is a 25% chance that, if he sees blood in the urine, it is caused by a bladder tumour, rather than merely by the worms laying their eggs. So, if you are in an endemic area, cystoscope everyone >30yrs who complains of haematuria (27.4). Few cases of carcinoma of the bladder occur in anyone <30yrs.

There is little that you can do for aggressive *schistosoma*-associated carcinoma of the bladder, but do try to confirm the diagnosis. Patients need to know if they have a serious condition or not. *Don't give repeated treatment for presumed* schistosomiasis.

Stage these tumours (27-8): most patients present in inoperable Stages III or IV. The only useful treatment for squamous tumours in Stages I and II is total cystectomy; the recurrence rate after partial cystectomy is high, but even total cystectomy has few 5yr survivors. No effective chemotherapy is affordable, except for those rare patients with high human chorionic gonadotrophin (β-HCG) levels. Ureteric diversion (nephrostomy, 27.14) gives symptomatic relief, but this tends to be shortlived and not without its problems. All you can often do is to palliate the patient (37.1). Death is likely from renal failure, due to obstruction of the ureters by the tumour.

Ultrasound (38.2i) will show a thickening or mass in the bladder wall.

Radiographs. An intravenous urogram (IVU) is helpful only where low-grade papillary transitional tumours predominate, because there may also be tumours of the ureter and renal pelvis.

Special tests
Measure the Hb and the blood urea. Examine the urine for microscopy and culture, and if you can, cytology, looking for irregular shed cells, and keratin.

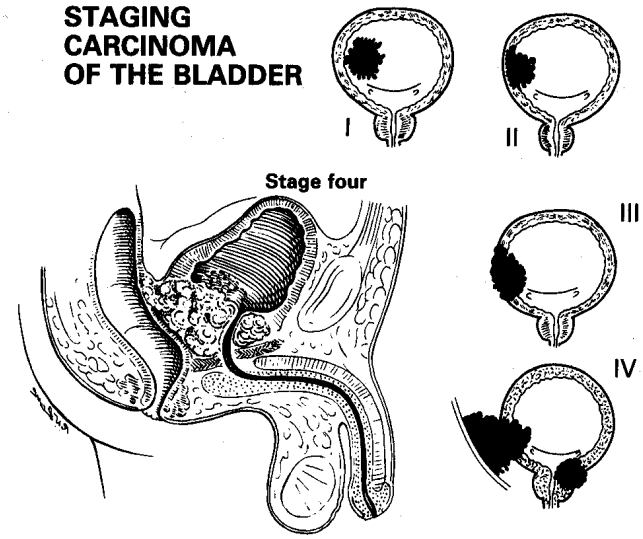


Fig. 27-8 STAGE CARCINOMA OF THE BLADDER by doing a cystoscopy and examining a patient under GA.
Stage I, the tumour is not palpable. Stage II, it is palpable, but is not larger than you would expect from cystoscopy. Stage III, it is mobile, but is larger than you would expect from cystoscopy. Stage IV, it is fixed. Stage V *(not shown)*, it is widespread.
After McNeill Love RJ. Bailey, & Love's Short Practice of Surgery, HK Lewis 15th ed 1971 Fig. 1415 with kind permission.

Staging (27-8)
Following cystoscopy, examine the patient bimanually under GA, with the bladder empty and the muscles relaxed. Try to confirm the diagnosis with a biopsy.

(1) **Stage I.** The tumour is sessile and not palpable. This needs cystodiathermy, or partial cystectomy for a transitional carcinoma. Total cystectomy, for an aggressive squamous carcinoma, is only justified if you explain the situation fully, and the resulting urostomy is manageable at home. It will spare much suffering, but is unlikely to provide a cure and may be socially unacceptable.

(2) **Stage II.** The tumour is palpable as a localized but definite thickening, which is mobile. It is <5cm in diameter, and is not larger than you expect from cystoscopy. Treat as for Stage I.

(3) **Stage III.** The tumour is mobile, >5cm in diameter, and is larger than you expect from cystoscopy. Cystectomy may be possible, but is highly unlikely to provide a cure.

(4) **Stage IV.** The tumour is fixed to the wall of the pelvis, or to the paravesical glands, or is infiltrating the vagina or rectum. Palliation only is possible.

(5) **Stage V.** There is widespread disease. Palliation only.

CAUTION!
(1) Confirm the diagnosis histologically, before advising radical surgery. Schistosomal granulomas (common in endemic areas) and tuberculosis can simulate small tumours.
(2) *Avoid a suprapubic cystostomy*, because it can cause a distressing, permanent urinary fistula if a malignant tumour is present.

27.6 Retention of urine

Retention of urine **in men** can be acute, chronic, or 'acute on chronic'. Six kinds of men suffer this way:
(1) A young man with a history of gonorrhoea, followed by a stricture or prostatitis. Sometimes, acute gonorrhoea alone is enough to cause retention, or he may have both.
(2) An old man with an enlarged prostate. The acute event causing urinary retention may be a pneumonia, a fractured femur, or just drinking too much beer!
(3) A man with painless retention caused by an acute neurological lesion, such as HIV transverse myelitis, injury or tumour of the spine, in which case the signs are obvious, but are often overlooked.
(4) A man with frank haematuria, from previous surgery (*e.g.* prostatectomy), trauma or a bleeding disorder.
(5) A man with a urethral injury, often from pelvic disruption.
(6) An older patient with a bladder tumour.
N.B. Old men may still have urethral strictures, and young men bladder carcinoma.

Retention of urine can happen **in women** because of:
(1) *detrusor* muscle failure complicating pelvic surgery, especially hysterectomy (23.15),
(2) a retroverted gravid uterus,
(3) an impacted fibroid in early labour,
(4) perineal trauma, including FGM,
and to both men and women as a result of:
(5) a neurological lesion particularly HIV neuropathy,
(6) an advanced bladder tumour,
(7) a rectal mass, which may be a faecal impaction!
(8) a horseshoe-type ischiorectal abscess (6.17).

Occasionally drugs may cause urinary retention, especially opioids and antipsychotics.

Acute retention usually presents in much the same way, whatever its cause, with acute discomfort, often late in the evening when the realization dawns that no urine is going to come out before bedtime. The bladder is usually distended to the umbilicus. No stricture is complete, and the final stage of the obstruction is probably congestion and oedema. This will subside if you drain the bladder suprapubically; then, if you try to pass a urethral catheter after 1-2wks, you will probably succeed. If you are going to perform a prostatectomy, and you can operate during the next few days, you can leave the suprapubic catheter in place until you do so. If there is a stricture, you can dilate this as soon as the acute oedema subsides.

SUPRAPUBIC CATHETERIZATION IS *MUCH* SAFER THAN A DIFFICULT URETHRAL CATHETERIZATION

First make sure that there really is retention of urine and this is not oliguria or anuria. If you cannot feel or percuss the bladder, the reason for the inability to pass urine must be in the ureters or kidneys, or be pre-renal. If in doubt, use an ultrasound to look at the kidneys and the bladder. One glance at the face will usually tell you if the retention is acute or chronic: acute retention is agonizing.

If the bladder is grossly distended, but not painful, the retention is either chronic or neurological.

(a) History
Has there been gonorrhoea, and how was it treated? Is it necessary to strain to pass urine? (suggests a stricture). Frequency, hesitancy, dysuria, nocturia? (prostatism).

(b) Examination
Look for heart failure, anaemia, and hypertension, which might be the result of an obstructive uropathy. Look for signs of HIV disease (5.5,6).

Examine the urethra from end to end, using your eyes and your fingers. Start at the glans. Exclude phimosis and stenosis of the meatus. Feel the urethra in the penis, and the perineum, for palpable thickening. Extensive strictures are associated with a large palpable area of scarring in the perineum. You may feel the distended proximal part of the urethra ending in a firm fibrous stricture. Examine the membranous urethra with your finger in the rectum. Look for scars on the scrotum and perineum. If there is a painful tender area in the perineum, it is probably a periurethral abscess complicating a stricture or a horseshoe ischiorectal abscess. Check for sensation in the ano-perineal area.

Examine the prostate rectally:
(1) The hardness and irregularity of carcinoma are usually easy to distinguish from the softer, smooth consistency of benign hypertrophy, although the gritty feeling of a calcified prostate may be misleading.
(2) A firm mass above the prostate is likely to be carcinoma of the bladder.
(3) Tenderness of the prostate is often difficult to assess, but a genuine prostatic abscess or acute prostatitis is usually exquisitely tender to palpation.
(4) An impacted stone in the prostatic urethra (uncommon; the meatus is the common site of impaction) or tuberculous prostatitis can be readily confused for carcinoma.

CAUTION!
(1) The size of a prostate is no indication as to whether it is causing obstruction or not, but it is useful to know its size when planning surgery.
(2) If the bladder is distended, the upper border of the prostate may be difficult to distinguish from the bladder base, and the prostate may seem enlarged, because it is being pushed downwards by the distended bladder. You may find later, when surgery is scheduled, that the prostate has disappeared! So if you do think it is enlarged, examine it again, after you have relieved the retention: *don't diagnose prostatic enlargement from one examination while in retention.*

Examine if the kidneys are palpably enlarged. Are the nerves to the bladder intact? Is there perianal sensation? Test the anal reflex during rectal examination, and feel for a patulous anal sphincter. If you suspect any neurological abnormality, examine the spine and legs thoroughly.

(c) Special tests
Later, examine the urine for sugar, protein, and pus. Diabetic and HIV neuropathy can cause retention, and proteinuria may indicate uropathy. Measure the Hb and the blood urea, and repeat this after 7 days' relief by catheterization if it was initially abnormally high.

Prostate specific antigen (PSA) is highly specific for prostatic carcinoma; it is very useful (27.22). Acid phosphatase will only tell you crudely about the presence of metastatic deposits: you should do both tests *before doing a rectal examination, or else there may be a false +ve result!*

(d) A plain radiograph of the kidney, ureter, and bladder may show stones or evidence of metastatic deposits in the pelvic bones, typical of prostatic carcinoma. *There is no need for a routine IVU*: reserve it for special indications, such as haematuria when the cause is not found on cystoscopy, or if you suspect some abnormality of the kidneys.

(e) Ultrasound. Look for the size of the bladder (when it is full), the thickness of its wall, any indentations or diverticula (38.2i), and the size of the prostate (38.2j). Examine the ureters and kidneys, looking especially for pelvi-calyceal dilation (38.2f).

(f) Relieving bladder obstruction
Pass a catheter (27.2). *Don't clamp it:* the pressure in the bladder is reduced by 50% by letting out only 100ml, so serial releasing of the flow is unnecessary and may even promote infection. It does nothing to prevent haematuria, caused by rupture of compressed bladder wall veins, which usually stops spontaneously.

If you fail to pass a urethral catheter, perform a suprapubic catheterization (27.7).
If this is contraindicated, and you are experienced, use pethidine and diazepam, or GA but *not ketamine* (which may cause bladder contraction). With deep relaxation, the sphincter should relax and a urethral catheter should slip in. If it does not, try a curved metal introducer to 'lift' a catheter into the bladder, *taking great care*. When it is in the bladder, remove the introducer.

CAUTION!
(1) Use an introducer with great care and gentleness: lubricate it generously when you put it into the catheter, and lubricate the catheter when you pass it into the urethra. This will help to prevent it being pulled out with the introducer.
(2) Make sure the introducer has a smooth curve. A kinked introducer will be difficult to extract.
(3) Make sure the introducer tip does not emerge through the catheter hole but sits snugly at its tip when you are introducing the catheter.
(4) *Never use force!*

If the urine comes out murky, turbid or smelly, treat this with the most suitable antibiotic available: *viz.* nalidixic acid, nitrofurantoin, trimethoprim, gentamicin or a quinolone.
N.B. Ampicillin and tetracycline are not usually very effective.

(g) Recovery diuresis: *danger of renal failure*
When you have relieved an obstruction to the urinary tract, the bladder and the kidneys may or may not recover. An early sign of recovery is a diuresis, which may amount to >5L/day.

If there is a recovery diuresis, measure the urine output carefully. If this is >200ml/h, replace this fluid loss by infusing IV fluids at 80% of the volume lost in the first 24h, then at 50% of the volume lost for the subsequent 48h. *Don't forget the potassium lost*: this may be as much as 35-40mmol/L urine produced. If possible, measure the serum $[K^+]$, and adjust the dose of potassium accordingly. Be guided also by the pulse and blood pressure chart. If you fail to appreciate the danger of this diuresis, renal failure may recur due to dehydration leading to poor renal perfusion, in spite of an apparently normal fluid intake.

(h) Difficulties with retention
If there are symptoms of prostatic obstruction with acute or chronic retention, but no large prostate, there are 2 possibilities (27.21):

(1) DYSKINESIA is a functional rather than a mechanical obstruction. You cannot diagnose it by the size of the prostate or by looking at the bladder neck. The bladder neck is not mechanically tight, but fails to open up during a voiding contraction. It can occur in HIV disease.
You can easily insert a catheter, which drains quantities of urine, and cystoscopy shows trabeculation (hypertrophic submucosal muscle fibres) of the bladder. Medical treatment with prazosin, an α-blocker, 0·5-2mg may help.

(2) BLADDER NECK STENOSIS is a mechanical obstruction due to fibrosis or previous prostatic surgery, or *schistosomiasis*. As with a urethral stricture, passing a catheter is difficult or impossible. Treatment is by incising the bladder neck, if possible endoscopically, deeply enough to divide all its circular fibres.

CYSTOSCOPY (27.3) is necessary to:
(1) exclude a urethral stricture,
(2) examine the size and nature of the prostate,
(3) show if there is bladder neck stenosis,
(4) assess the trabeculation of the bladder (evidence of longstanding obstruction) and presence of diverticula,
(5) demonstrate the presence of stones or carcinoma.
N.B. Never perform a urethral dilation in a woman unless there is a definite urethral stricture (27.9)*!*

27.7 Emergency (closed/blind) suprapubic cystostomy

If a patient has retention of urine, and you cannot pass a catheter, the alternative is to drain the bladder through the abdomen. As the bladder distends, it rises up above the pubis and strips the peritoneum off the abdominal wall.

This allows you to drain it without passing through the peritoneal cavity. Passing a catheter on an introducer is best if you have to continue drainage for more than a few days. Much less satisfactory is using a plastic tube and trocar or needle puncture, because there is nothing to stop urine leaking internally.

There are special kits to enable you to do this slickly. We describe what to do if you don't have these.

INDICATIONS
(1) Acute obstruction with a full bladder, such as that from an enlarged prostate, a urethral stone, or a stricture when catheterization has failed.
(2) Urethral rupture.

CONTRAINDICATIONS
(1) An empty bladder. *Don't try closed suprapubic cystostomy if there is extravasation of urine*.
(2) Carcinoma of the bladder causing retention, because a fistula track may form, which is very distressing, and carcinoma can spread to the abdominal wall.

So, *feel for a craggy rectal or suprapubic mass before you make a suprapubic puncture.* Get an ultrasound scan if in doubt.

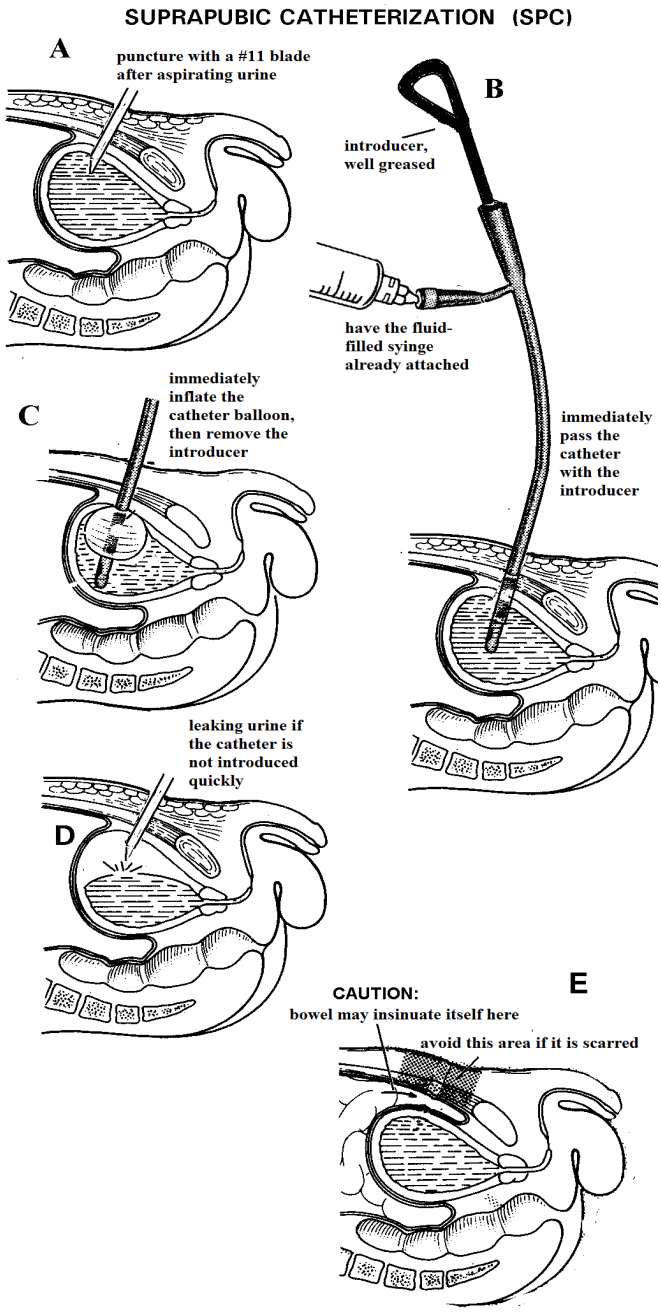

SUPRAPUBIC CATHETERIZATION (SPC)
A
puncture with a #11 blade after aspirating urine
B
introducer, well greased
have the fluid-filled syringe already attached
immediately pass the catheter with the introducer
C
immediately inflate the catheter balloon, then remove the introducer
leaking urine if the catheter is not introduced quickly
D
E
CAUTION:
bowel may insinuate itself here
avoid this area if it is scarred

Fig. 27-9 EMERGENCY SUPRAPUBIC CATHETERIZATION (CLOSED/BLIND SUPRAPUBIC CYSTOSTOMY). A, puncture the abdominal wall and bladder with a #11 blade. B, introduce the catheter with introducer inside and syringe attached. C, immediately blow up the balloon when the catheter is inside the bladder. D, urine extravasating if you don't blow up the balloon quickly enough! E, if the peritoneum is tethered by a previous operation scar, you may traverse the peritoneal cavity and damage the bowel.

CAUTION!

(1) For a closed (blind) suprapubic puncture, the bladder must be distended and palpable. If it is not, wait for it to fill, or perform a formal open cystostomy (27.8).

(2) The classical site for drainage is halfway between the pubis and the upper limit of bladder dullness.

N.B. Note that in children the distended bladder becomes intra-peritoneal, so closed suprapubic catheterization is dangerous.

If the patient is to have a prostatectomy later, perform the cystostomy (drainage) as high as you can, so that you can open the abdomen below later, without entering the cystostomy track.

PREPARATION

Make sure you have all the equipment ready that you need: this is important, because you will otherwise find urine flowing out uncontrolled when you stab open the bladder, and then have to perform an open cystostomy!

Have ready a well-lubricated catheter, already mounted on an introducer, with a filled syringe attached to the balloon channel.

INCISION (GRADE 2.1)

Scrub up and put on sterile gloves. Check the outline of the bladder by preference using ultrasound. Infiltrate the site of puncture with LA in the midline. Continue to infiltrate down to the bladder; when you get there, confirm it is distended by aspirating urine into the syringe. Keep the bladder steady by placing your left hand on its dome. Make a small cut with a #11 blade (4-1) in the midline halfway between the dome of the bladder and the symphysis pubis. Push this in the same direction as that taken by the needle you used to aspirate urine, till you feel you have punctured the bladder wall (27-9A).

Immediately when you see urine coming out, pass the catheter with its introducer into the bladder (27-9B) and *blow up the balloon* (27-9C). *Don't delay!*

CAUTION!

(1) *Avoid suprapubic scars*: if the peritoneum is adherent to the abdominal wall, you may injure the bowel. It is quite acceptable to make your puncture wound 2cm lateral to the midline, but *beware the inferior epigastric vessels!* Use ultrasound if possible to check the position of the bladder.

(2) Puncture the abdominal wall in the direction of the lower sacrum. *Don't direct the catheter too caudally; you may enter the retropubic space and fail to enter the bladder. Don't direct it too cranially; you may enter the abdomen and possibly injure the bowel.*

Drain the urine into a urine bag. Make sure there is a daily fluid intake of at least 3L/day: a generous fluid intake is the best way of preventing or clearing infection.

If there is a urethral stricture, drain the bladder for 1wk before you attempt to deal with the stricture.

Before removing a suprapubic catheter, clamp it.

You can then estimate the residual urine by measuring the volume which drains through the tube, after a good passage of urine *per urethram*. If there is no residual urine, you have succeeded.

DIFFICULTIES WITH EMERGENCY SUPRAPUBIC CYSTOTOMY

If urine fails to flow readily, first check that the catheter tube is patent by flushing it with sterile water. If there is still no urine flow, suspect extravasation of urine into the suprapubic space (27.12), which will result in spreading cellulitis or Fournier's gangrene (6.23); or urinary leak into the peritoneum. Perform an open suprapubic cystostomy (27.8) and leave an extravesical drain.

If there is heavy or prolonged bleeding, suspect a bladder tumour, or damage to the bladder neck or prostate. Abandon the procedure and perform an open suprapubic cystostomy.

If there is bowel content in the catheter, you have punctured small or large bowel! Perform a laparotomy and repair the perforation (14.3) and thoroughly lavage the abdomen; *don't forget to place a catheter formally in the bladder*, through a separate stab incision!

27.8 Open suprapubic cystostomy

METHOD (GRADE 2.2)

INDICATIONS

(1) Extravasation of urine (27.11).
(2) Rupture of the bladder.
(3) Intractable urinary clot retention.
(4) Extraction of a bladder stone or foreign body.
(5) An impassable urethral stricture.
(6) During open prostatectomy (27.20) or laparotomy for another reason.

INCISION

Make a 5cm midline vertical suprapubic incision, a little longer if there is excess fat. Divide the *linea alba*, and retract the rectus muscles. Use your forefinger, covered with a gauze swab, to push the connective tissue and peritoneum upwards, away from the anterior surface of the bladder. Dissect the loose fatty tissue away anterior to it.

The bladder may be empty as the result of extravasation of urine due to trauma or a stricture. A similar operation is needed for the removal of a stone or foreign body from the bladder (27.16).

Recognize the bladder by its characteristic pale appearance with some tortuous blood vessels on its surface. Aspirate it first, unless it is impalpable (as with trauma causing extravasation). Insert stay sutures, superiorly and inferiorly, at the proposed ends of your vertical bladder incision. They will make useful retractors when it sinks into the pelvis. Open the bladder with a longitudinal 5cm incision, take urine for culture, and explore the bladder with your finger.

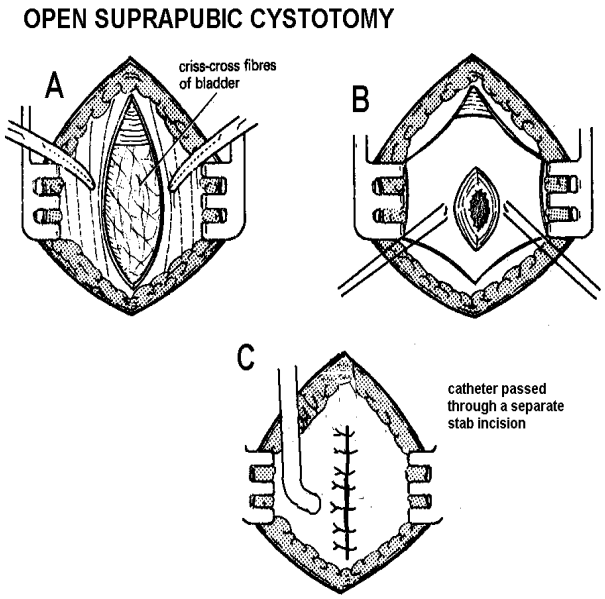


Fig. 27-10 OPEN SUPRAPUBIC CYSTOSTOMY.
A, midline incision (*avoid using a Pfannenstiel incision*). The cystostomy tube should emerge halfway between a patient's umbilicus and the symphysis. Part the rectus muscles to reveal the criss-cross fibres of the bladder. B, open the bladder between stay sutures. C, close the bladder in 2 layers. Make a separate stab incision for the catheter so that there will be less likelihood of a leak when it is withdrawn.
After Blandy J. Operative Urology, Blackwell 1978 Figs. 8.14-24 with kind permission.

If the bladder is not distended, and you should divert the urine flow, you cannot make a closed (blind) suprapubic cystotomy, so you have to use the open method.

CONTRAINDICATIONS

Carcinoma of the bladder (common in areas where *Schistosoma haematobium* is endemic), because it may lead to a permanent and distressing urinary fistula.

If you are going to leave a suprapubic catheter in place, pass a Foley catheter into the bladder through a separate stab incision above or to the side of the main one. Make it a snug fit and hold it in place with a purse-string suture.

Close the main bladder incision with 2 layers of 2/0 or 1/0 absorbable sutures. Close the wound with the catheter emerging through a long, oblique, midline track. Extend the wound proximally if necessary. If it is likely to be infected by contaminated urine, as it may be if you are operating for extravasation, insert an extravesical drain.

CAUTION! Make sure the suprapubic catheter emerges through a different incision laterally, so that the track closes easily, and will not interfere with an approach to the bladder later.

Change the catheter monthly, or 3-monthly if you have a silicone catheter. Once a track has been established after the first 10-14 days, you should have no difficulty replacing the catheter. *Replacing it earlier may be almost impossible*.

If the replacement catheter does not pass easily, introduce a guide wire along the track. Dilate the tract gently and then slide the catheter along the guide wire. To do this, cut a hole *longitudinally* at its end, so it slides along easily. *Don't cut the catheter transversely at its end,* because this creates a sharp edge which does not easily pass along an irregular track.

CAUTION! Don't leave a persistent urinary fistula without a catheter in place as this is distressing, causing a smell, permanent wetting of clothes, and chronic infection.

27.9 Urethral strictures

Gonorrhoea is the most usual cause of urethral stricture in men everywhere. Some strictures are the late results of *schistosomiasis*, prostatectomy, tuberculosis, trauma, or instrumentation of the urinary tract. Whatever its cause, you should try, if possible, to get a urethrogram, urethroscopy, and the release of the stricture with an optical urethrotome.

Strictures can be of any length from 0·5-10cm. The commonest sites for gonococcal stricture are:
(1) the bulbar urethra (27-18), and rarely
(2) at the junction of the penis and scrotum,
(3) in the glans penis.
Gonococcal strictures are the result of fibrosis in the corpus spongiosum. Meatal strictures are different (27.31). Traumatic strictures due to instrumentation occur usually in the prostatic urethra, but from external pelvic injury in the anterior urethra.

A urethral stricture increases the resistance to micturition, which causes the detrusor muscle of the bladder to hypertrophy. This may produce an adequate flow initially, but as time passes, sacculations and diverticula form in the bladder; it no longer empties completely, and the high residual urine it contains leads to frequency of micturition, and infection. Sensation is diminished, as its wall is increasingly replaced by fibrous tissue. Finally, the result is 'retention with overflow', and incontinence. Bilateral hydronephrosis develops as high pressure is transmitted to the ureters, and thus secondary renal failure (obstructive uropathy) results.

Apart from acute painful retention, and chronic painless retention with overflow incontinence, the many other complications of urethral stricture include:
(1) False passages.
(2) Periurethral abscesses (6.18) causing extravasation of urine, with gross distension of the penis and scrotum (sometimes leading to gangrene), and external fistulae.
(3) Infection of the urinary tract.
(4) Infection of the seminal vesicles, epididymes or testes.
(5) Chronic non-specific infection ending in elephantiasis.
(6) Obstructive uropathy ending in renal failure.
(7) Bladder neck stenosis, and detrusor failure. These are common and may explain why bouginage and external urethroplasty often fail.
(8) The results of straining, such as hernias or prolapse of the rectum.
(9) Stones in the urethra and bladder (27.16,17,18).
(10) Infertility and impotence.

Urethral strictures can be dilated, although they are never cured. *The problem with dilation is that you can very readily traumatize the longer male urethra further and worsen the stricture, or create a false passage by perforating it. This is a disaster.* Try, by all means, to treat strictures under direct vision with a urethrotome.

If this is impossible, and it is not feasible to leave a suprapubic catheter *in situ*, you may be justified in attempting dilation. However, *don't do this with rigid sounds, and don't do this for:*
(1) *Acute retention of urine,*
(2) *Prostatic or peri-urethral abscess,*
(3) *Extravasation of urine.*

Use sedation, but avoid GA as you will then not know how much you are traumatizing the urethra. Administer gentamicin IV pre-operatively. Preferably, only use soft filiform bougies. These are long, thin, flexible nylon rods which you introduce into the urethra till they reach the point of the stricture. Thread these into the urethra one by one, till one of the bougies passes into the bladder. The bougies have a thread on the distal end, onto which you can screw the follower of greater size. *Dilate the stricture by not more than Ch2 on each occasion!* Full dilation requires many repeated bouginages, until you can easily, and completely atraumatically, pass a metal sound.

The optical urethrotome is a very useful instrument to learn how to use, and useful to obtain. *If you are not experienced, use it with great care.* The aim is to make a cut anteriorly in the stricture (at the 12o'clock position) and occasionally at the anterolateral (4 and 8o'clock) positions if the stricture is dense. You can do this under LA if you introduce lidocaine jelly; leave it in the urethra for 5mins using a penile clamp. Generally leave a Ch16 urinary catheter *in situ* 7-10 days post-operatively and arrange follow-up dilations (best done with plastic sounds by the patient himself at home).

You can do a great deal of harm by enthusiastically dilating urethral strictures blind: leave this for an expert. In the absence of urethrotomy, it is best to divert the urine flow, and allow the expert to deal with a simple stricture, than to ask him to deal later with a complicated stricture which may prove impossible to negotiate.

Prostatic obstruction is the main differential diagnosis (27.19). Chronic retention distends the bladder greatly, but is painless, so that decompression is not needed so urgently as it is in acute retention.

Urethral strictures in women occur often in combination with vesico-vaginal fistula (21.18), after mutilating surgery, or in the elderly due to perineal contracture. The urethra is short and usually amenable to simple dilation.

Late complications

If there is a tender painful swelling in the perineum, this is probably a PERI-URETHRAL ABSCESS (6.18), which may or may not be associated with retention of urine. The diagnosis is not difficult, but you can easily overlook it in the presence of retention of urine.

If the testes and epididymes swell, this is an acute epididymo-orchitis. Treat non-operatively with ampicillin or trimethoprim.

If the perineum, lower abdomen or penis swell, this is due to EXTRAVASATION of urine (27.13). Multiple fistulae may develop with gross thickening of the peno-scrotal skin (27-12).

If stones develop, they are the result of infected stagnant urine, and may form in the dilated urethra proximal to the stricture. They will remain until removed by cystotomy or urethrotomy. Treat the infection, and arrange intervention for the stricture.

N.B. **If there is haematuria** without instrumentation, there may be a bladder tumour (27.5). Get an ultrasound scan (38.2i).

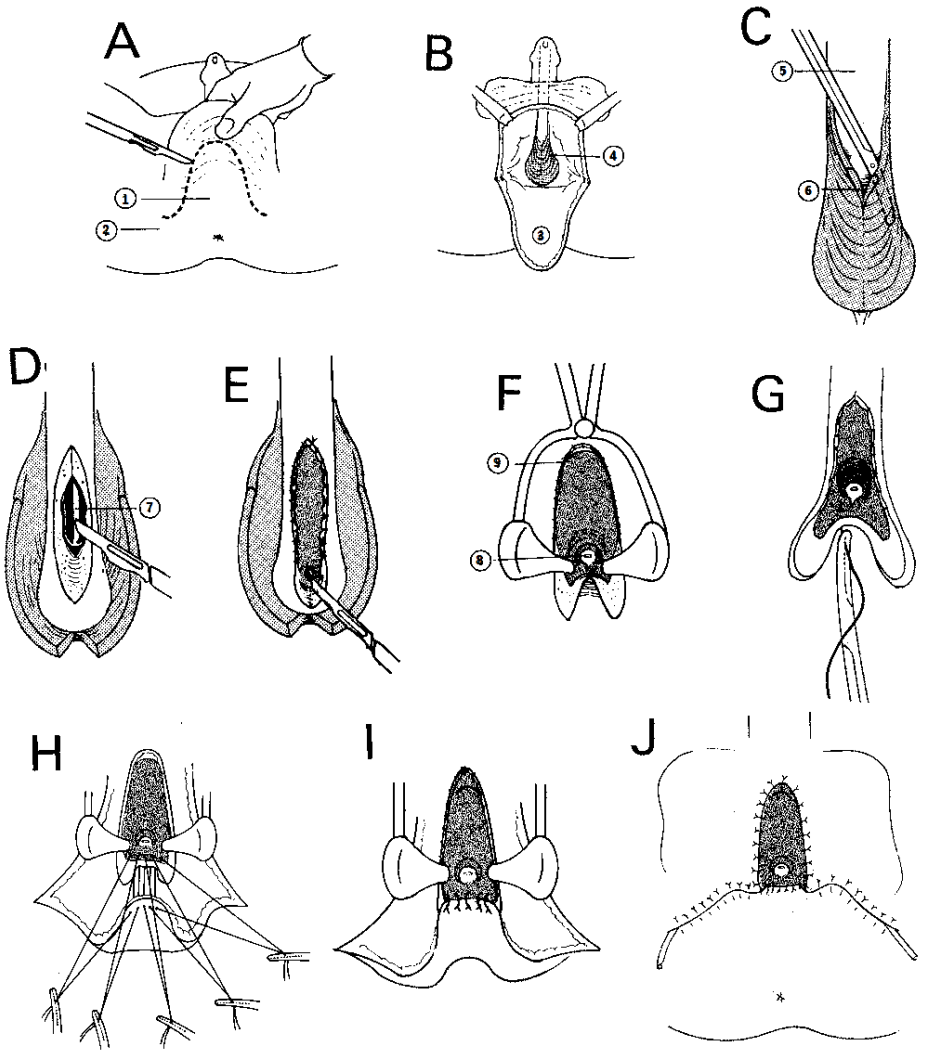


Fig. 27-11 PERINEAL URETHROSTOMY. This is the 1st stage of Blandy's urethroplasty.
A, outline the flap. (1) scrotal flap, (2) ischial tuberosities. B, allow the flap to fall down. (3) flap reflected, (4) *bulbospongiosus.* C, reflect the *bulbospongiosus* from the bulbar urethra. (5) urethra. (6) *bulbospongiosus* incised. D, open the urethra on to a bougie (7) just distal to the stricture. E, oversew the edges of the *corpus spongiosum* for haemostasis. F, inspect the urethra with a nasal speculum, and continue to incise it, until you emerge into healthy mucosa, and can see the verumontanum (8) proximally. (9) sutured edge of the *corpus spongiosum*. G, insert sutures at the edge of the divided urethra to evert it. H, lead 5 sutures through the apex of the flap. I, tie the top 5 sutures, bringing the flap to the edge of the opened-out urethra. J, likewise approximate the advancement scrotal skin flap to the opened urethral edge all round.
From Blandy J, Operative Urology, Blackwell, 1978 Figs14.43-6, with kind permission.

27.10 Impassable urethral strictures

A stricture which you cannot dilate or open is a difficult problem. If it is short and of traumatic origin, you may be able to excise it, and anastomose the ends of the urethra end-to-end. If however it is the result of inflammation, it is likely to be longer, and needs a formal urethroplasty in at least 2 stages, in which a new urethra is made with scrotal skin. This is a lengthy and difficult procedure and is work for an expert. There are, however, some simpler options:
(1) A permanent suprapubic cystostomy (27.8),
(2) A perineal urethrostomy. This is part of the 1st stage of Blandy's posterior urethroplasty.

This will result in a permanent orifice, through which urine is passed 'like a woman'. It will not affect potency, but it may be very embarrassing having semen coming out of 'the wrong place'!

The 1st stage of a posterior urethroplasty is not easy, but is much easier than the 2nd. Even the 1st stage may give much relief, and is much better than a permanent suprapubic catheter, but it is not easy to get a good channel which will not stenose; also, bleeding can be a nuisance.

It is feasible for an impassable stricture anywhere in the urethra, even as high as the verumontanum.

(a) Perineal urethrostomy (THE 1ST STAGE ONLY OF A POSTERIOR URETHROPLASTY) (GRADE 3.2)

INDICATIONS. Impassable strictures.

PREPARATION. Make sure the perineum is washed and perfectly clean. Put the patient in the lithotomy position. Shave the perineum, and prepare the skin with care.

METHOD. Make an inverted U-shaped scrotal flap with rather a flat apex to end just in front of the ischial tuberosities (27-11A). The key to the operation is access, so the flap must go far back. Cut through the skin and *dartos*, tying and coagulating vessels as you go, and allow the flap to hang down.

CAUTION!
(1) Allow a generous lining of fat on the flap.
(2) *Don't disturb the vessels in its base*.
(3) *Don't use diathermy on the flap*, or you may cause flap necrosis.

Pass a Ch24 bougie down to the tip of the stricture, and ask your assistant to hold it in the midline. Feel for it, and dissect down to it, until you see the *bulbospongious* muscle (27-11B). Dissect the muscle from the bulb and reflect it on either side (27-11C). Cut down on to the bougie (27-11D), and immediately insert a 4/0 continuous absorbable suture on either side of the cut *corpus spongiosum*, to prevent bleeding from this spongy tissue. Incise until you have completely opened the stricture and you reach healthy tissue; in a bulbar stricture you may have to cut almost to the bladder within a few mm of the verumontanum. Cut 1cm at a time, and control bleeding by continuing your haemostatic suture down each side of the split *corpus spongiosum* (27-11E). *Don't divide the external sphincter just distal to the verumontanum.*

CAUTION! Be sure to continue the incision the full length of the stricture. The only way to be sure about this is to pass your finger past the stricture, to make sure there are no strands of fibrous tissue remaining.

Inspect the stricture and the verumontanum with a nasal speculum (27-11F). Divide all fibrous bands until you see the verumontanum. This is normally a cystoscopic landmark, and is a posterior midline swelling in the urethral mucosa. It is just proximal to the external sphincter and the ejaculatory ducts open onto it. Then pass 5 interrupted 3/0 absorbable sutures through the flap onto the opened urethra (27-11G). Attach each haemostat to the drapes, so that they cannot be muddled up (27-11H). Push the flap towards the bladder. Tie one throw on each knot until it is tight. Reinsert the speculum, and check that the edge of the flap is neatly up against the defect in the urethra, before completing the series of knots. If not, readjust and replace the suture which was at fault. When you are sure the flap is in the right position, put several more throws on each knot, and cut their free ends. Withdraw the speculum and complete the work of suturing in the flap, trimming away surplus skin where necessary (27-11I). Use fine monofilament to bring the edges of the scrotum to the edges of the urethra, previously exposed (27-11J). Leave the catheter *in situ*.

POST-OPERATIVELY, treat with frequent baths or a douche, remove the catheter at 5 days, and the sutures, after premedication, at 14 days. Make sure that there are no cross-adhesions between the suture lines. If a tissue bridge has formed, separate it, and ask him to keep the passage open by inserting a sound daily in the bath.

(b) Difficulties with a perineal urethrostomy

Curiously, incontinence is uncommon.

If the tip of the scrotal flap necroses, take it down, trim it and resuture it; there is usually plenty of skin left.

If a haematoma forms, return to theatre, take down the wound, evacuate it, and secure haemostasis.

27.11 Urethral fistula

A proximal urethral fistula in the male is usually the consequence of a periurethral abscess, but may arise spontaneously, particularly with HIV disease. Multiple fistulae may involve the perineum, scrotum, penis, perianal region and inner aspects of the thighs (27-12). Sometimes a fistula forms between the urethra and the rectum.

Special tests. Try to delineate the stricture with a urethrogram (38.1) and urethroscopy.

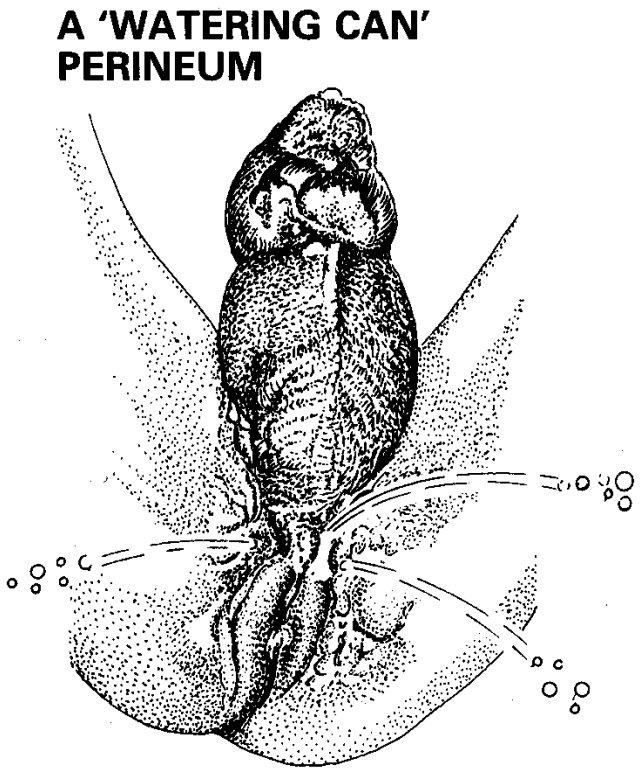


Fig. 27-12 A 'WATERING-CAN PERINEUM' is the late result of a complex gonococcal, or rarely a tuberculous stricture. Multiple chronically infected and epithelialized fistulae have involved the penis, scrotum, perineum and thighs. A slow ooze of purulent discharge is more usual than the shower of urine shown here.
Kindly contributed by Neville Harrison.

If the fistula is recent, divert the urine by an open suprapubic cystostomy (27.8), culture the urine from the bladder and use the appropriate antibiotic, and after 6wks clamp the suprapubic catheter to see if urine still leaks. If it does, repeat the process for a further 6wks, and if this fails, the patient needs an optical urethrotomy (27.9).

If the fistula is chronic, divert the urine by an open suprapubic cystostomy and excise the fistula track. If there are many, this may be impossible, forcing you to leave a permanent urinary diversion in place.
Consider the possibility of TB or carcinoma, and take a biopsy.

If there is a congenital recto-urethral fistula, it is usually associated with an imperforate anus (33.6).

A distal urethral fistula is usually the result of trauma, either from a crude circumcision, or from penile piercing with rings. Correction requires an operation similar to a hypospadias repair (33.9).

A urethral fistula in women is invariably associated with a vesico-vaginal fistula (21.18).

27.12 Extravasation of urine

The effects of extravasated urine are dramatic. The combination of urine and infection produces severe oedema of the scrotum and abdominal wall. Untreated, the skin over the scrotum, penis, and anterior abdominal wall may slough. This results in Fournier's gangrene (6.23) with consequent severe illness, toxicity, fever, dehydration, anaemia, and uraemia. If renal function is impaired, as it often is after a longstanding stricture, extravasation may be fatal.

Urine can extravasate from a stricture spontaneously especially with HIV disease, through a periurethral abscess, or as the result of bouginage or other types of trauma. The attachments of Camper's & Scarpa's, Buck's and Colles' fascia limit the spread of urine so that from a defect in the bulbous urethra it can track into the scrotum, up over the pubis and into the lower abdominal wall. However, from a defect in the more distal penile urethra the urine leaking is limited to the penis.

PREPARATION
Replace fluids IV, which may be life-saving, and correct electrolyte disorders. Treat with IV chloramphenicol, gentamicin and metronidazole. Counsel the patient that urinary diversion may be long-term. Exclude cardiac, renal, and hepatic causes of oedema.

METHOD
(1) DIVERT THE URINE FLOW so that it no longer leaks into the tissues. Perform a formal open suprapubic cystostomy (27.8).

(2) DRAIN THE URINE OUT OF THE TISSUES.
Make 5cm incisions on each side of the base of the penis. Insert your index finger, and open up the tissue planes widely towards the abdomen, and down the shaft of the penis. Then make 5cm incisions on the inferolateral aspects of the scrotum, and use your finger to open up the tissue planes as far as possible (27-13).

EXTRAVASATION OF URINE

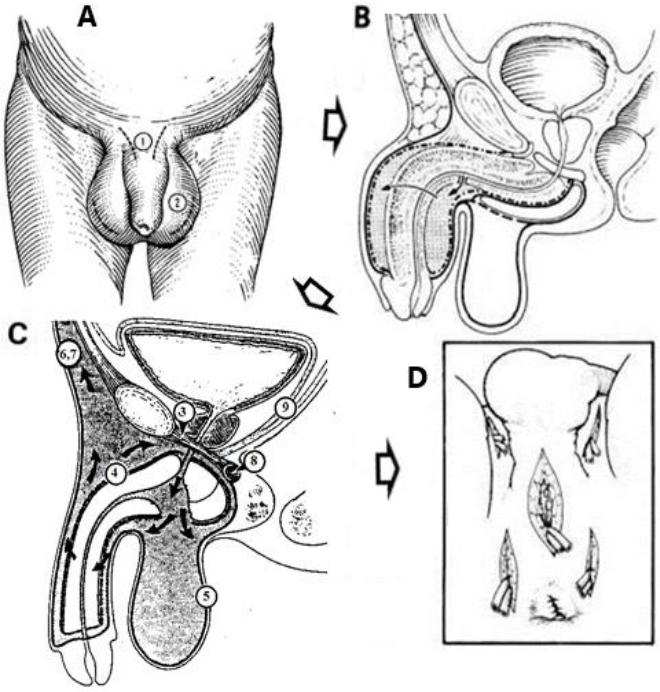


Fig. 27-13 EXTRAVASATION OF URINE.
Subfascial areas where extravasated urine may collect. A,D, incisions for drainage (1-2). B, drawing showing the directions in which urine can track. (3) triangular ligament. (4) Buck's fascia. (5) Colles' fascia. (6,7) Camper's (superficial) & Scarpa's (deep) fascia. (8) *transversus perinei* muscle. (9) Denonvillier's fascia.
A, from Dudley HAF (ed) Hamilton Bailey's Emergency Surgery. Wright 10th ed 1977 Fig 50.11 with kind permission.

Place 2 long corrugated rubber drains (4-14B) into the depth of each wound in each direction, and suture them in place. Dress the wounds with gauze and cotton wool. Arrange daily baths. The swelling will usually settle in about 5 days. Shorten the drains 5cm/day.

If you find any areas of necrotic skin and subcutaneous tissue, debride these radically, otherwise Fournier's gangrene (6.23) will result. When infection has subsided, close the skin incisions by secondary suture, and graft the bare areas unless it has healed spontaneously.

(3) LATER, DEAL WITH THE UNDERLYING STRICTURE. Allow inflammation to settle for at least 4-6wks; change the suprapubic catheter after 4wks.

27.13 Urinary tract stones

Stones in the urinary tract vary greatly in their prevalence. For example, they are common in North India and the Sudan, but are rare in East and Central Africa. In the 'stone belts' of Southeast Asia and South America, they are very common, even in children. You should be able to:
(1) Relieve the excruciating pain of renal colic.
(2) Make a nephrostomy for calculous anuria: this can be life-saving, but is rarely needed.
(3) Remove a ureteric stone (27.15).
(4) Remove a bladder stone in an adult (27.16) or child (27.17).
(5) Remove a stone impacted in a child's urethra (27.18).
Removing a stone from the kidney or renal pelvis, however, is a task for an expert.

(a) Stones are of 2 kinds:
(1) Primary or metabolic stones.
(2) Secondary stones resulting from obstruction or repeated infection.
Primary stones are most common in men of 30-50yrs, and usually form in the renal pelvis or else in the lowermost calyx.

The size and position of a stone determine what effect it has. If it is small, and remains in the periphery of the kidney, or in a calyx, it may cause few symptoms; if it enlarges, it may obstruct part of the kidney. A small stone <7mm diameter usually passes down the ureter, causes acute ureteric colic as it does so, and later is voided in the urine. If it is too big to pass, it may obstruct the upper end of the ureter, and cause hydronephrosis which will ultimately destroy the kidney.

Stones in the bladder don't usually return when you remove them, but those in the upper urinary tract do so with a 50% chance during the next 10yrs. The most useful preventive measure is a high fluid intake.

Most stones are radio-opaque, so learn where to look for them; an occasional exception is a urate stone in a child's bladder, but even these usually contain enough calcium to let you see them on a radiograph.

(b) Special tests. The presence of microscopic haematuria is the most useful test. If there are pus cells, or a patient's urine is alkaline, it is infected.

(c) Radiographs. Take a plain radiograph of the kidneys, ureters and bladder (a slightly oblique abdominal view).

You can easily miss a stone if:
(1) the radiograph is poor,
(2) the stone is only moderately radio-opaque,
(3) it is obscured by bone or dilated bowel.

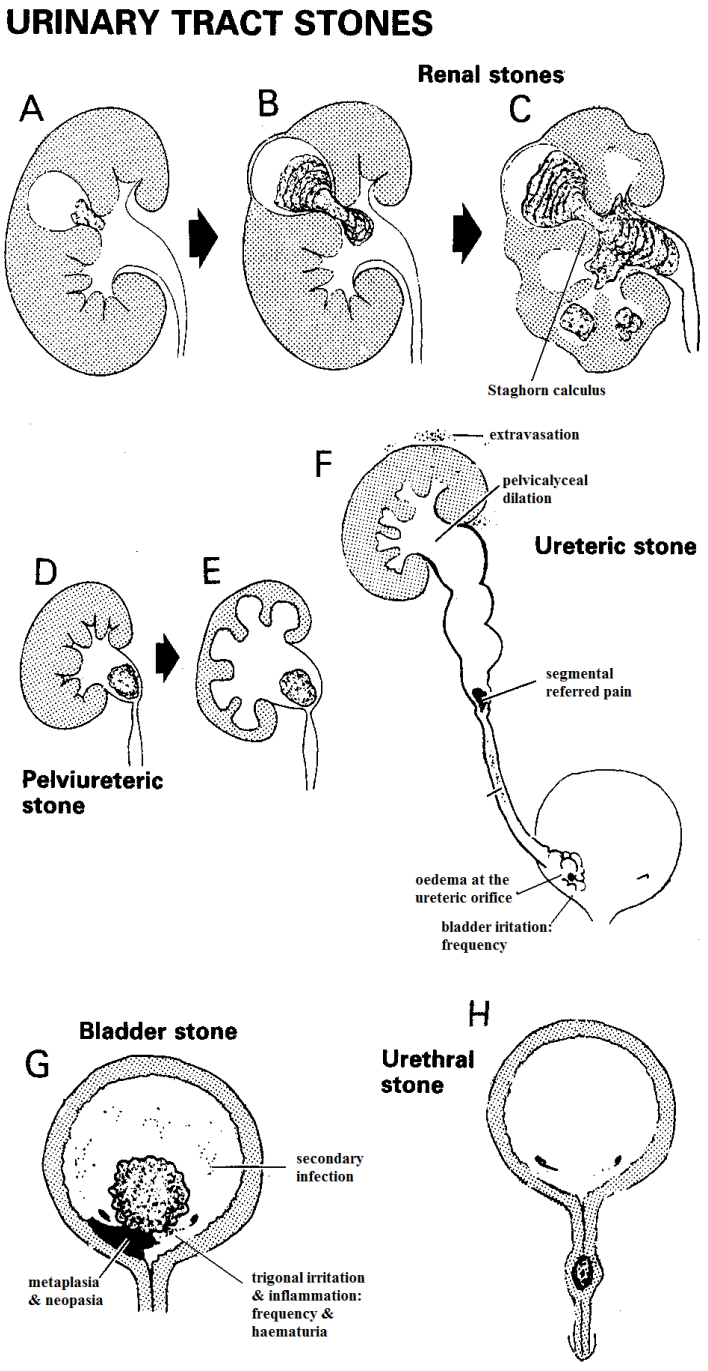


Fig. 27-14 STONES IN THE URINARY TRACT.
A, stone jammed in a calyx causing hydrocalyx. B, atrophied parenchyma over the dilated calyx. C, staghorn calculus formed, with several stones elsewhere in the kidney. D, stone impacted at the pelviureteric junction. E, calyces dilated and renal parenchyma compressed. F, stone in the ureter causing loin pain, segmental referred pain, haematuria, and frequency. G, bladder stone with squamous metaplasia ending in carcinoma. H, stone in the urethra.
After Blandy J. Operative Urology, Blackwell 1978 with kind permission.

Look for kidney stones opposite the second lumbar vertebra, and for ureteric stones, as the ureter crosses the tips of the transverse processes of the lumbar vertebrae, runs over the sacroiliac joint, and descends in a gentle arch to a point just medial to the ischial spine, from where it turns medially to enter the bladder.

Don't mistake a gallstone, a phlebolith (calcified thrombus) or a calcified lymph node for a urinary stone. Most ureteric stones are slightly elongated and *not round*. In a lateral view of the abdomen: gallstones are anterior; renal and ureteric stones overlie the lumbar spine.

(d) An intravenous urogram (IVU: 38.1a) will confirm the diagnosis. If no contrast medium is excreted on one side, that kidney has stopped functioning, or is excreting so slowly that you can be sure function is impaired. You will find the site of an obstructing stone that is not visible on a plain radiograph easily only if there is still some function remaining. Take exposures immediately at 1h immediately after emptying the bladder, so that contrast medium does not obscure a stone at the lower end of the ureter; then at 3, 12 and even 24h. Enough contrast medium may then have accumulated to show up the urinary tract, down to the site of the obstruction.

If contrast medium is concentrated in the kidney (a 'nephrogram'), but does not show up in the renal pelvis, a stone may have blocked the pelvi-ureteric junction (27-14E), and caused the contrast medium to be retained in the kidney tissue. This is a hopeful sign, because it shows that there is still good renal function.

N.B. Clots in the ureter, and small calculi, may cause colic especially if the ureter is narrowed by *schistosomiasis* or tumour.

(e) Ultrasound will demonstrate most stones easily in the bladder because of their 'acoustic shadow' (38.2i), but not so readily in the ureter or kidney. It may be helpful with the differential diagnosis and follow-up in checking for hydronephrosis. If a ureter is completely blocked, no spurt will be visible on Doppler from the ureteric orifices.

(f) Differential diagnosis

If there is moderate pain in the costovertebral angle, a high fever, chills, obviously infected urine, and an ultrasound shows that the renal pelvis and calyces are normal, acute pyelonephritis is present.

If there is a palpable tender renal mass, this is probably hydronephrosis. If in addition there is fever, toxaemia, and leucocytosis, it is probably a pyonephrosis.

If there is a dull ache, with occasional fever and pyuria, suspect that there is a stone which is not obstructing the urinary tract.

If there is anuria and renal failure, this can be due to the presence of bilateral stones, but it is more likely to be due to chronic interstitial nephritis or pyelonephritis.

CAUTION! Some stones cause no symptoms, even when they are large.

If there is right iliac fossa pain and fever, this may be appendicitis (14.1) or PID (23.1) in a female, especially with tenderness on rectal or vaginal examination.

(g) Treatment for urinary stones

If there is a small kidney stone (<7mm) which is peripheral in the kidney, and is causing no symptoms and no infection, leave it, but watch it carefully, to see if it gets stuck at the uretero-vesical junction and causes obstruction.

If a stone is obstructing the renal pelvis, try to remove it. The risk of hydro- or pyonephrosis is high. If there are stones on both sides, operate on the side with the better function first.

If there is renal colic for a few days, after which oliguria and then anuria gradually develop, this can arise from bilateral obstruction, or, more commonly, from the obstruction of a solitary functioning kidney. Catheterization of the bladder produces no urine. A plain film may confirm the diagnosis. The blood urea rises. The episode may relieve itself spontaneously as the result of the oedema in the ureter settling, and the infection being brought under control. Observe for 24-48h.

If you have a cystoscope and can pass a ureteric catheter, it may slide past the stone and produce urine; you can then leave it in place for 2-3 days, which will relieve the acute situation. Or the catheter may dislodge the stone back into the renal pelvis. If there is no rapid improvement, urgently fashion a nephrostomy (27.14).

If there is a stone stuck in the ureter, you should remove it extraperitoneally unless you can let the urine drain from above it (27.15).

If there is a uric acid stone, try to raise the pH of the urine. Make it alkaline with sodium bicarbonate tablets tds, or potassium citrate mixture 20ml tds. If possible, measure the serum urate. Treat with allopurinol if there are recurrent uric acid stones, or an elevated urate.

If the serum [Ca^{2+}] is consistently high, it suggests a parathyroid adenoma, or some other generalized disease. A raised urinary calcium is more common; advise against taking calcium supplements.

TO PREVENT RECURRENT STONES, encourage plenty of fluids and treat any associated infection.

27.14 Nephrostomy for calculous anuria or hydronephrosis

If there is obstruction of the upper urinary tract in the ureters or the pelves of the kidneys, life is only in danger if both sides are obstructed simultaneously, or there is obstruction in a solitary kidney. When this happens, no urine is passed and soon death comes from renal failure, unless something is done quickly.

(a) Ureteric obstruction can be the result of:
(1) *Schistosoma haematobium* causing strictures at the junctions of the ureters and the bladder, so producing hydronephroses.
(2) Stones obstructing the renal pelves (or a staghorn calculus on one side, and no function on the other).
(3) Mistakenly tying both ureters at a hysterectomy (23.15) or Caesarean section (21.11).
(4) Bilateral pelvi-ureteric junction obstruction.

A chronically obstructed kidney is usually large, so whenever you diagnose renal failure, always palpate for enlarged kidneys. Permanent relief of the obstruction requires expert surgery. Meanwhile, with luck, you may be able to keep a patient alive long enough, if you put a tube into one of the obstructed kidneys to decompress it. Chronic obstruction of this kind is not uncommon in areas where stones or *schistosomiasis* are endemic.

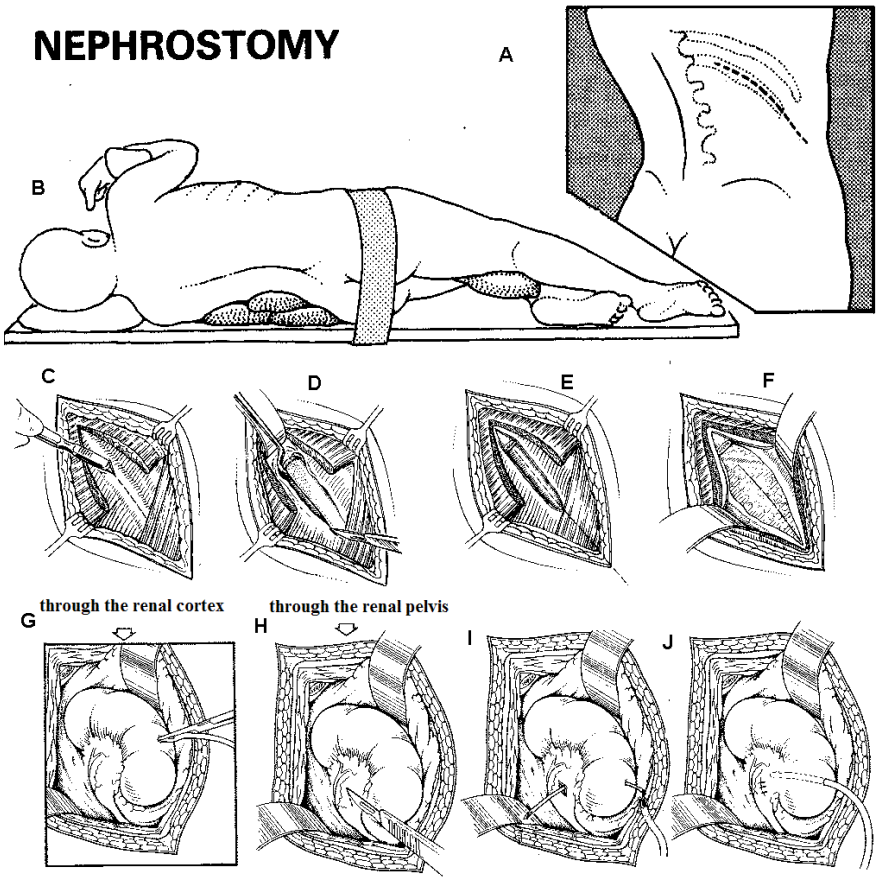


Fig. 27-15 NEPHROSTOMY.
A, expose the kidney by an incision over the 12th rib. B, ready for surgery, with sandbags under the loin and the arm supported. C, cut *latissimus dorsi* and strip the periosteum over the 12th rib. D-E, remove the rib. Alternatively, cut just below the 12th rib but *don't remove it.* F, expose the perirenal fat. G, to drain a kidney through its cortex, push a catheter into a tense fluctuant area. H, to drain a kidney through its pelvis, make a short incision in the posterior of the renal pelvis, well away from its junction with the ureter. I, pass a probe through this incision out through the cortex of the kidney, and tie a catheter to it. J, the catheter in place.

Open nephrostomy is not an easy operation, because the kidney is deep and difficult to get at. It is easier for schistosomal hydronephrosis of slow onset than it is for stones, because the kidney is always large.

N.B. If you have ultrasound, it is much easier to drain the kidney by making a percutaneous track and dilating it serially; you may then be able to use this track to remove stones. However, this does require some special instruments.

Having exposed the kidney, you can either push a catheter through a dilated calyx, if you can find one, or you can open the renal pelvis and pull a catheter through the kidney into it. If a stone is the cause, and you can easily remove it, and the patient's condition is good, do so.

(b) Nephrostomy (GRADE 3.2)
If there are bilateral stones, decompress the side on which there has been more recent pain or discomfort, because this is the side which is most likely to regain its function.

POSITION. Use the lateral position with the kidney to be operated on uppermost (27-15B). If your table has a kidney bridge, place this at the 12th rib. Then raise the bridge, so as to open up the space between the rib cage and the pelvis.

If you don't have a kidney bridge, introduce 3 or 4 sandbags or folded pillows into this space.

If you have a table that can be broken (the head or foot end can be lowered separately), use it to give you more room.

Flex the lower knee, straighten the upper knee, and put a pillow between them. Support the upper arm on a cushioned Mayo instrument table, to prevent the trunk rotating. Take a wide strap, or a long piece of wide adhesive strapping, and wrap this round the pelvis and trochanters, so that the pelvis will not rotate. Have the patient leaning forwards a little, rather than strictly on the side.

INCISION
Here we assume that you are going to remove the 12th rib. You can, if you wish, approach the kidney just below and parallel to it, without excising it, especially when the kidney is large, as with hydronephrosis. If necessary, make a short incision forwards from the last (12th) rib.

Mark the 12th rib with a felt pen (27-15A). Then clean an area about 20cm wide over the 12th rib, from the midline of the back to the umbilicus. Stand at the back, and make a skin incision starting at the lateral margin of the *sacrospinalis*. Cut along the line you have drawn over the 12th rib. Proceed anteriorly, and stop 5cm short of the umbilicus, at the lateral margin of the rectus sheath. A shorter incision will suffice if there is a marked hydronephrosis.

You will have to cut through much muscle. If possible, use a cutting diathermy, turned down low enough to cut through muscle and coagulate the vessels in it at the same time. Or, use a scalpel, and carefully control the bleeding points as you meet them.

Start by cutting the *latissimus dorsi* over the 12th rib, until you can see the rib clearly (27-15C). Then remove it subperiosteally with a scalpel, or cutting diathermy. Cut the periosteum down the middle of the rib as far as its tip. Using a periosteal elevator, push the periosteum off its raw surface down its entire length. Reflect the flaps of periosteum. Take a curved periosteal stripper, and gently insert it under the distal part of the rib. Slide it up and down, until the rib is completely clear of periosteum (27-15D,E). Cut the narrow strand of *external oblique* attached to the tip of the rib. Use rib shears, or bone cutters, to cut off the rib as close to its neck as is convenient. *Don't push towards the neck of the rib*; it is too close to the pleura! Smooth its stump so that it will not tear your gloves.

Cut the 3 muscles of the anterior abdominal wall in line with the skin incision. Cut the first 2, the *external oblique* and *internal oblique*, boldly. When you get down to the *transversus*, stop temporarily. The peritoneum is under it, and you don't want to risk opening it and flooding the peritoneal cavity with urine, or having bowel obscure your view.

Return to the bed of the rib, and use the tip of a scalpel to cut its lowermost half. Carry the incision down on to the remaining fibres of the *transversus* muscle. Split this in the direction of its fibres.

You will now see the peritoneum, with the liver and part of the colon under it (27-15F). Using a sponge on a holder, gently push the peritoneum down and away from you forwards and upwards. Use a self-retaining retractor to separate the rib cage above from the crest of the ilium below, and so open up the whole area.

Feel for the kidney up against the posterior abdominal wall. If you are not sure if it is the kidney, try moving it up and down. Use a scalpel to make a short incision in the fascia over it. Insert your fingers, separate the perirenal fat (which may be extensive in an obese patient), and feel the shape, size and consistency of the kidney. The tissues around it will probably be engorged and oedematous.

If the kidney is enlarged, soft and feels cystic, it is probably hydronephrotic, but it may be polycystic, in which case nephrostomy does not help. If it is hydronephrotic, it is probably safe to drain a dilated calyx through the cortex, without exposing the renal pelvis.

If the kidney looks and feels fairly normal, expose its pelvis, and put the drain there.

CAUTION!

(1) *Be careful not to damage the fragile and often flattened renal vein*, which enters the renal hilum anteriorly, and may cover part of the renal pelvis: this is why you should approach it from behind.

(2) The end of the catheter must go into the drainage system, and not into the kidney parenchyma itself.

(1) NEPHROSTOMY THROUGH THE CORTEX is easier, but does not provide such good drainage. Choose an area on the convex surface of the kidney, where the renal parenchyma is thinly stretched over a tense fluctuant area, and which feels as if there is probably urine under pressure close below it. To confirm that you have found a dilated calyx or pelvis, aspirate it with a fine needle and syringe. Be sure that you are not dealing with an isolated renal cyst.

Make a ½cm incision into the kidney capsule over its convex border, and then plunge a fine haemostat into the fluctuant area. If urine pours out, you are in the right place. Suck it out. Take a small catheter, hold its tip with a haemostat, and push this far enough into the kidney to get a good flow of urine (27-15G). Remove the haemostat and leave the catheter in. If blood oozes around it, insert a haemostatic fine absorbable suture.

(2) NEPHROSTOMY THROUGH THE RENAL PELVIS (PYELOSTOMY) drains a kidney better as it will drain all the calyces, but is more difficult, because you have to find access to the medial side of the kidney.

Turn the kidney forwards and medially, using finger dissection. When the perirenal tissues are oedematous and thickened, separating the kidney from surrounding fat is not difficult. You will see the tense distended renal pelvis as the most posterior of the structures at the hilum. Holding the kidney so as to expose the renal pelvis, confirm that urine is present by aspirating with a syringe and fine needle. Make a short incision in the renal pelvis, well away from its junction with the ureter. Urine should gush out (27-15H).

Pass a curved probe through this incision. With your other hand, feel for an area on the convex surface of the kidney, where its cortex feels thin. Carefully (to minimize bleeding) push the tip of the probe out through this point (27-15I). Tie the probe to a catheter, and draw it back and out through the kidney (27-15J). Close the pyelostomy opening with two fine absorbable sutures. If the kidney bleeds where the catheter emerges, apply a purse-string suture.

Bring the nephrostomy tube to the surface through a separate stab incision where it will not be occluded on lying down. Irrigate the tissues round the kidney, and close all the muscles over it in layers. Close the skin, and fix the nephrostomy tube. Finally, as an extra precaution, tape the nephrostomy tube to the skin. Connect it to a bedside collecting bottle.

If there is no area of thinned cortex, as may happen with a stone, remove the stone through an incision in the renal pelvis, and let the nephrostomy catheter drain from there.

If at any time you open the pleura, close it and put in a chest drain (9.1).

POST-OPERATIVELY, if urine drains freely, you have succeeded, and renal function should improve. Watch for the nephrostomy tube kinking or blocking. If it blocks, try irrigating it. You should replace, or remove, a silicone catheter after 3 months, and an ordinary one after 4wks. There may develop a massive recovery diuresis, so make sure that you replace the lost fluid IV (27.6).

He will need definitive surgery later, when his general condition permits.

27.15 Ureteric stones

The stone that obstructs the ureter originates in the kidney. Once it is free in the renal pelvis, it may pass into the ureter, and cause obstruction most likely:
(1) at the entry of the ureter into the bladder,
(2) at the pelvi-ureteric junction,
(3) in the lower ⅓ of the ureter, or
(4) in the upper ⅓ of the ureter.

A normal-sized stone may obstruct if there is a pre-existing stricture, typically due to *schistosomiasis*. A stone is usually rough, so that some urine can usually leak past it to begin with. Later, obstruction becomes complete, so that after some weeks or months, hydronephrosis or a hydroureter develop, which may become infected.

As the stone passes down the ureter, it causes severe ureteric colic: even a tiny stone causes agonizing sudden pain in the loin, radiating to the groin, perineum, and testis (or to a woman's labia). The patient vomits, sweats, and rolls about to get relief. If, at the same time, the urine is infected, there are fever and rigors. The urine may be 'smoky', but is seldom grossly blood-stained. There may be slight tenderness in the area of the referred pain, and there may have been attacks like this before. If the stone impacts, the severe pain of ureteric colic gradually subsides.

There is an 85% chance that the ureteric stone will be passed into the bladder, and then out through the urethra. So administer plenty of fluids, and treat the pain.

(a) Differential diagnoses include:
(1) Appendicitis (14.1), especially retrocaecal, but this will only give colicky pain in its early phase.
(2) Biliary colic (15.2).
(3) Torsion of an ovarian cyst or uterine fibroid.
(4) PID (23.1).
(5) Colic due to the passage of blood clot in the ureters, resulting from trauma, or a neoplasm.

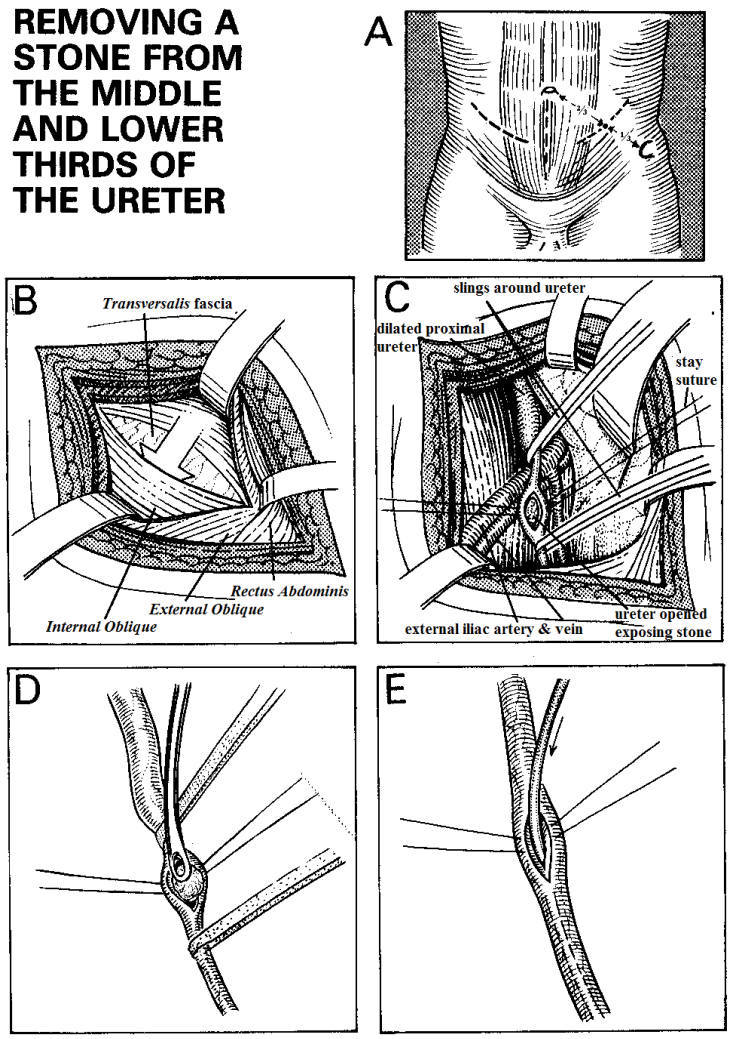


Fig. 27-16 REMOVING A STONE FROM THE MIDDLE AND LOWER ⅔ OF THE URETER.
A, centre the incision on McBurney's point; for the middle ⅓ carry it laterally in the line of the inguinal ligament, for the lower ⅓ carry it medially in the line of this ligament. Alternatively, for the lower ⅓ use a midline incision. B, divide the *external oblique* in the direction of its fibres. Cut the *internal oblique* to expose the transversalis fascia. C, sweep the peritoneum medially to the bifurcation of the common iliac artery. Find the ureter and the stone. Pass catheters, or tapes, round the ureter above and below the stone. Cut on to it with a small knife. D, carefully remove it with Desjardin's forceps. E, pass a thin paediatric feeding tube upwards and downwards to check patency. Leave the ureter open and insert a drain.
Kindly contributed by Samiran Nundy.

(b) Special tests (see 27.13)

(c) Management
Leave a stone of <7mm to pass spontaneously, unless there is some complication. A bigger stone is less likely to pass. An impacted stone may remain in the ureter for weeks or months, without necessarily causing obstruction.

(1) NON-OPERATIVE TREATMENT
Relieve the pain with opioids and NSAIDs. Repeat these as required. Administer plenty of fluids, and encourage walking about. Filter the urine to look for the stone(s). Repeat the ultrasound or radiographs if pain persists.

(2) INDICATIONS FOR SURGERY
(i) Symptoms persist, and serial radiographs taken at 6-8wk intervals show that a stone of >5mm is impacted. If it is not causing symptoms or obstruction, it does not necessarily have to be removed, but it is desirable to do so.
(ii) Pain comes and goes over days or weeks without any further descent of the stone.
(iii) Ultrasound shows ipsilateral dilation of the kidney.
(iv) An intravenous urogram shows a hydronephrosis or a hydroureter, or no excretion of contrast medium.
(v) Infection supervenes with fever, chills, rigors, pyuria, and toxaemia.

PREPARATION. Mark the side to be operated upon. Take a plain radiograph of the abdomen just before you operate to make sure that the stone has not moved.

FOR A STONE IN THE RENAL PELVIS OR UPPER ⅓ OF THE URETER, perform a nephrostomy (27.14) if the kidney is obstructed. *Don't try to remove a stone from the renal pelvis unless it is dilated.* Try to refer the patient.

FOR A STONE IN THE MIDDLE ⅓ OF THE URETER, perform a MIDDLE ⅓ URETERO-LITHOTOMY (GRADE 3.2).
Use the supine position. Start the incision ⅔ from the umbilicus to the anterior superior iliac spine, and carry it *laterally* for 7cm parallel to the inguinal ligament. Divide the subcutaneous tissues, and the *external oblique* aponeurosis in the direction of its fibres; likewise divide the *internal oblique*.

Divide the *transversalis* fascia, and sweep the peritoneum medially, until you reach the inner margin of the *quadratus lumborum* muscle, and the bifurcation of the common iliac artery into its internal and external iliac branches (24-16). You will see the ureter lifted up by the peritoneum. *Don't injure the spermatic vessels*, which lie lateral to the ureter.

Feel for the stone in the ureter. Carefully pass a long Lahey forceps round the ureter, and pass 2 fine catheters, or cloth tapes, above and below the stone. Exert gentle traction on these. This will prevent the stone slipping upwards or downwards. Cut longitudinally on the ureter onto the stone with a #15 blade. Remove it carefully with Desjardin's forceps. Wash the area free of grit with warm saline. Pass a small paediatric feeding tube up into the kidney, and down into the bladder, to make sure there is no other stone, or if you suspect a schistosomal stricture at the vesico-ureteric junction.

Leave the ureteric incision open. *If you try to close it, the sutures may well cut out and a stricture is likely to form.* Place a #12 catheter near this site, and bring it out through a separate stab incision. Close the abdominal incision in layers, using interrupted absorbable for the muscle, and monofilament for the skin. Connect the catheter to a closed drainage system.

CAUTION! Make sure you find the stone and place a sling around the ureter above the stone. If it slips upwards into the kidney, *don't try to remove it by extending the incision, or using a traumatic instrument*.
Close the incision and wait. Try to refer the patient, or try again when ureteric colic recurs, after making sure the stone is indeed in the middle ⅓ of the ureter!

POST-OPERATIVELY, the catheter will drain up to 1L of urine daily, but the volume will gradually diminish. By the 7th day the ureteric incision should close, and the drainage cease.

If the volume draining remains undiminished, there is an obstruction in the ureter distal to the site of the incision, or it is diseased locally. Wait another week, and investigate this with an IVU (38.1a).

FOR A STONE IN THE LOWER ⅓ OF THE URETER, perform a LOWER ⅓ URETERO-LITHOTOMY (GRADE 3.3).
Ideally, you can remove a stone at the lower end of the ureter with a cystoscope and a Dormia basket which traps the stone, but this is difficult. Otherwise, empty the bladder by passing a urethral catheter. Use the supine together with a slight Trendelenburg position. There are 3 possible approaches. Remaining outside the peritoneum, which should be your aim, is easier in the first.
(1) Start your incision ⅔ from the umbilicus to the anterior superior iliac spine and continue *medially* parallel to the inguinal ligament. Incise the *external* and *internal oblique*, and open the *transversalis* fascia.
Or, (2) Make a lower midline incision, starting at the pubis, and ending at the umbilicus. Incise the *transversalis* fascia.
Or, (3) Use a Pfannenstiel incision.

Carefully strip the peritoneum upwards with a gauze swab. Look for the ureter at the bifurcation of the common iliac vessels (23-16) and follow it downwards to the bladder. It is crossed anteriorly by the vas deferens. You may have to divide the superior vesical artery so as to let you sufficiently mobilize the bladder medially to allow you to see the vesico-ureteric junction easily.

Find the stone, and pass a fine sling or cloth tape under the ureter proximally to prevent the stone slipping upwards. Make a longitudinal incision over the stone, and remove it carefully. Leave the ureteric incision open and close as above.

(d) Difficulties with ureteric stones
If the ureteric colic resolves, but there is no evidence that the stone has passed, this is not uncommon. It has probably passed without notice, especially if it was small.

If a stone becomes impacted at the pelvi-ureteric junction, and there is only one working kidney, perform a nephrostomy urgently. Try to refer the patient.

If a stone is firmly impacted at the utero-vesical junction deep in the pelvis, it might be that this area is diseased, especially as a result of *schistosomiasis*. You can try to squeeze the stone upwards into a more accessible part of the ureter where it will be easier to remove, but this does not usually work. Alternatively, make an incision 2cm above the site of impaction, and try to remove the stone carefully with Desjardin's forceps (27-16D): this will, however, not deal with any disease. Take care: you can easily tear off the diseased ureter from the bladder; it may need re-implantation anyway. This is complex surgery.

NEVER CLOSE THE URETER

27.16 Bladder stones in adults

Bladder stones can be primary, or secondary owing to urinary obstruction, or vesico-vaginal fistula (21.18). Primary stones are still common at all ages, mostly, but not only, in the poor, in a 'stone belt' which includes North Africa, the Middle East, Iran, Pakistan, India, Burma, Thailand, Vietnam, Laos, Cambodia, southern China, and Indonesia. If you are not in the stone belt, and you do find a bladder stone in an adult, be sure to exclude distal obstruction. Otherwise, a fistula may form and refuse to heal.

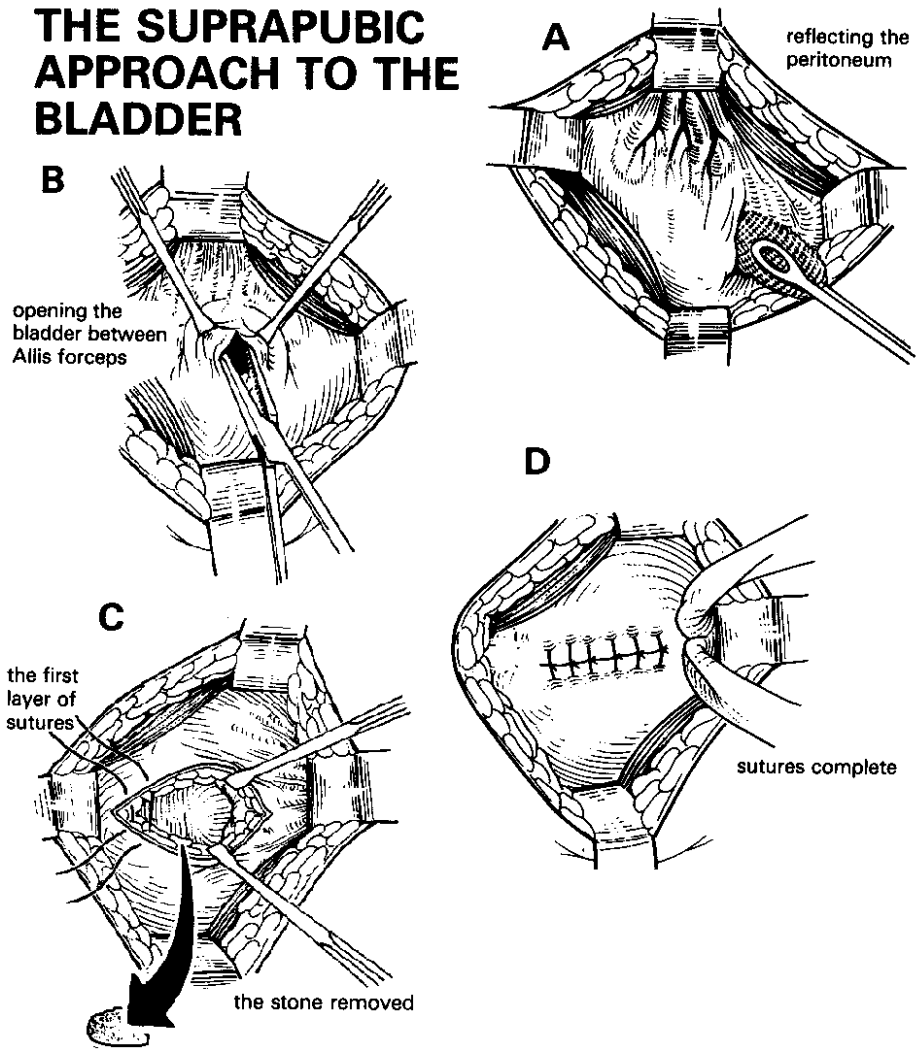


Fig. 27-17 SUPRAPUBIC APPROACH TO THE BLADDER FOR THE REMOVAL OF A STONE. In this view you are standing on the patient's left side, so that the bladder appears upside down.
A, displace the reflection of the peritoneum upwards. B, hold the bladder in Allis forceps and open it. C, the 1st step in closure. D, complete the 2nd layer of sutures.
After Flocks RH, Culp DA. Surgical Urology Yearbook Medical 4th ed 1975, Plates 69,70.

Most bladder stones in adults cause no pain, or slight pain in the perineum, or, if a stone is big, a 'bumping feeling' as the stone moves about. Nonetheless it is likely to cause intermittent infections and grow bigger. Otherwise, on standing up, a stone may fall onto the trigone, produce an intense urge to pass urine, but obstruct the urethral opening, making this impossible.

The operation to remove a bladder stone in an adult is similar to that for the 1st stage of a Freyer's prostatectomy, and open suprapubic cystostomy (27.8). Use absorbable suture to close the bladder.

N.B. For good results, it is important always to:
(1) Keep the bladder empty with an indwelling suprapubic or urethral catheter.
(2) Drain the retropubic space, so that blood and urine cannot accumulate.
When you have removed a bladder stone it does not usually recur, but nonetheless encourage a good intake of fluids.

(a) Ultrasound (38.2i) **and radiographs** confirm the diagnosis, because bladder stones in an adult are usually radio-opaque.

(b) Differential diagnosis

(1) Calcification of the bladder wall due to *schistosomiasis* (very common in endemic areas and gives no trouble). You may be able to confirm this by showing that the shadow is a different size when the bladder is full and empty, if it has not become rigid.
(2) Calcification in a uterine fibroid.
(3) A calcified mesenteric lymph node.

(c) Cystolithotomy (GRADE 2.5)

PREPARATION
Insert a urethral catheter and fill the bladder with fluid; you can use the same catheter for post-operative drainage. A steep Trendelenburg position will make exposure easier.

INCISION
Use a Pfannenstiel incision (11-4) because it is easier then to remain below the peritoneum and avoid opening it. Incise the skin and subcutaneous tissue transversely. Either part the rectus muscles to expose the peritoneum in the midline, or cut the rectus muscles transversely in line with the skin incision, little by little, until you see the inferior epigastric vessels in the deep surface laterally. This will give you better exposure, and you will be less likely to incise the peritoneal reflection over the bladder in error, which may spread infection into the peritoneum. Find the reflection of the peritoneum and displace this upwards (27-17A).

Grasp the bladder with stay sutures on either side of the midline, or with two Allis forceps, holding the entire thickness of its wall. Make a vertical incision in the bladder. *Avoid a transverse incision*: it will bleed more than a vertical one, and if you are too inferior you may damage the ureters. Suck away the urine as it gushes out.

Put your finger into the bladder to feel if the stone is lying free or is impacted in a diverticulum. Feel for a tumour or other pathology. Remove any free stones with your fingers, a scoop, or lithotomy forceps.
CAUTION! Repeatedly wash out the bladder before you close it. If you leave any stony fragments, they will act as the nuclei for the formation of more stones.

Close the bladder in two layers with continuous or interrupted 2/0 long-acting absorbable. Place a suprapubic catheter to provide drainage, and put an extravesical drain as well; bring these out through a separate stab wound.

POST-OPERATIVELY, remove the extravesical drain after 48h. Leave the suprapubic catheter in place for 8-10 days, to keep the bladder collapsed while it heals. Take a urine specimen for culture before you clamp the suprapubic catheter to see if normal voiding is possible. If so, remove it. An initial leak will stop in 24h.

(d) Difficulties with bladder stones in adults

If there is copious urine coming from the drain, and little in the catheter bag, the bladder closure is probably leaking. Check that the drain fluid is indeed urine by measuring its urea concentration, or by inserting dye *via* the catheter and seeing if it runs out *via* the drain. If the leak continues for 48h without diminishing, take the patient back to theatre, open up the wound and close the bladder wall again carefully in layers.

If a fistula develops, it will probably be the result of some obstruction to the urethra. Leave a urethral catheter in long enough for the fistula to heal. You will probably have to deal with the prostatic enlargement or urethral stricture later, though.

If the wound becomes septic, open it and drain it: *don't rely on antibiotics.*

27.17 Bladder stones in children

In some parts of Asia, smooth stones, mostly ≤5cm in diameter, form in the bladders of underprivileged children (mainly boys). Even an infant may suffer from them. They may be the result of vitamin A deficiency. When you have removed a bladder stone, it is unlikely to recur.

A child's mother will say that he cries every time he passes urine, and pulls at the penis as he tries to relieve the pain. Strangury, *i.e.* slow painful passage of small quantities of urine, makes life unbearable; sometimes he passes blood. Other symptoms include interruption of the urinary stream, frequency, dysuria, and suprapubic pain.

There are few physical signs: the bladder may be distended, and the foreskin red and swollen from being pulled. You may be able to feel the stone on rectal examination. It is likely to be made of urates, but it will probably contain enough calcium for you to see it on a radiograph. It will be readily visible on ultrasound (38.2i).

Removing a stone from the bladder of a child is not too difficult. When you have done so, there is no need to drain the bladder with a catheter, either suprapubically or through the urethra, if you have closed it securely.

ASHVIN (3yrs) had repeated urinary infections which had been treated with antibiotics on many occasions, but the symptoms always returned. He then saw another doctor, who remembered that repeated urinary infections in children should always be investigated, so he X-rayed Ashvin's bladder and was surprised to see a large stone. At operation, the stone was difficult to remove, and appeared to be lying in a diverticulum. After it was removed, he had no more urinary infections.
LESSON *Don't forget the possibility of stones in children*, especially if you are in a high-incidence stone area.

(a) Cystolithotomy in children (GRADE 2.5)

PREPARATION. Distend the bladder with water before you start, so that you can find it more easily. Pass a small plain catheter; then, inject 100-200ml of water into the bladder, depending on its size.

INCISION. Make a lower transverse subumbilical skin incision. Reflect the skin flaps 1cm on either side. Divide the *linea alba* strictly in the midline, without entering the peritoneal cavity.

Keeping the umbilicus in view to help you stay in the midline, make a vertical incision through the whitish aponeurotic fibres of the *linea alba*. Continue the incision down to the symphysis pubis, where you will meet the *pyramidalis* on each side. With a sponge on a holder, gently push the *rectus* muscles laterally, so that you can see the

posterior rectus sheath. Insert a small self-retaining retractor to keep the *rectus* muscles apart.

Feel for the distended bladder: it should be easily palpable as it rises out of the pelvis. Using a sponge, or your index finger, gently open the retropubic space. At the same time displace the peritoneum, so that you don't enter the peritoneal cavity. You should now be able to feel and see the distended bladder.

Insert stay sutures, and apply a haemostat on each side of the midline at the most easily accessible part of the bladder. This will prevent it slipping away. Get the sucker ready. Then with a scalpel, or cutting diathermy, make a 2cm incision in the bladder wall, between the 2 stay sutures. Aspirate the urine which squirts out.

With your index finger, feel through the hole in the bladder for the stone. Remove it with stone forceps or a sponge-holder. If the hole is not big enough, enlarge it. Having removed one stone, feel again to make sure that there is no further stone. Wash out the bladder.

Close the bladder carefully with continuous 3/0 long-acting absorbable on an atraumatic needle. Include all layers, and make the bites ≤5mm apart. The longer the incision, the more care you need in closing it.

CAUTION! Make a small stab incision just beside the wound and insert a small soft extravesical drain. *Don't forget to do this*. Even if you think you have closed the bladder securely, it may still leak. If urine extravasates, it may cause serious cellulitis.

Suture up the *linea alba* with 2/0 absorbable. Make sure that you have controlled all bleeding, and then close the skin. If you think that the bladder may leak, or if you have had to make a large incision, insert a Foley urethral catheter for 4-5 days. Otherwise, *don't insert one*. It is, however, helpful to insert a small suprapubic catheter and have it for a trial of voiding.

POST-OPERATIVELY, urine will probably pass without difficulty later that day. If urine leaks through the extravesical drain, insert a urethral catheter, and leave it there for a few days. Otherwise, remove the extravesical drain after 3-4 days. The child is unlikely to get another stone, but his siblings may.

27.18 Urethral stones in children

In some areas, bladder stones are common; these are mostly passed spontaneously in the urine. Occasionally, one impacts in the urethra, especially in boys. It is usually possible to pass urine around it, but pain, strangury, and dribbling are severe. Suspect this especially if there is a distended bladder, and you can feel a hard mass somewhere along the course of the male urethra. The patient may be able to show you exactly where the stone is stuck.

If ultrasound or radiograph suggests that it might be wedged in the neck of the bladder, try to feel it rectally. You may be able to push it back up into the bladder. If you fail, insert a well-lubricated sound into the urethra, until it strikes the stone. You may be able to push it back into the bladder, *but don't use force!* If this fails, ask your assistant to exert upward pressure on the stone, with the finger in the child's rectum, while you manipulate it with the sound. If you can move the stone back into the bladder, proceed to remove it suprapubically (27.17). Otherwise open the bladder and try to dislodge the stone out of the urethra from above: this may however be difficult.

If it is readily palpable in boys between the bulbous urethra and the *fossa navicularis* (27-18A), removal by manipulation under GA may succeed. *Don't use force!*

If it is impacted, remove it by external urethrolithotomy.

If it impacts at the external meatus or the *fossa navicularis* in boys, you may be able to 'milk' it free, or release it by a meatotomy (27.31).

If it has developed in a diverticulum (rare), you may have to cut down on this, and then close the diverticulum.

URETHROLITHOTOMY (GRADE 3.1)

Use the lithotomy position.

Thoroughly clean the genitalia, the medial surface of the thighs, and the perineum. Feel for the stone in the urethra, and steady it between the thumb and index finger of your left hand. Infiltrate with dilute adrenaline solution: this is a very vascular area. *If you don't control bleeding, everything will be obscured*.

Make a 3cm midline incision over the stone, on the ventral surface of the penis. Cauterize or tie off the bleeding points. Ask an assistant to retract the skin flaps with hooked retractors.

Incise onto the part of the urethra containing the stone. Make the incision just big enough to deliver it. Lift it out with stone forceps or a haemostat. Try not to break it.

Insert a small (Ch10 or 12) Foley catheter into the external urethral meatus, and up past the incision into the bladder. Inflate the balloon. *Don't try to close the urethra:* its edges will fall together and heal naturally. Close the skin. Remove the Foley catheter after 1wk.

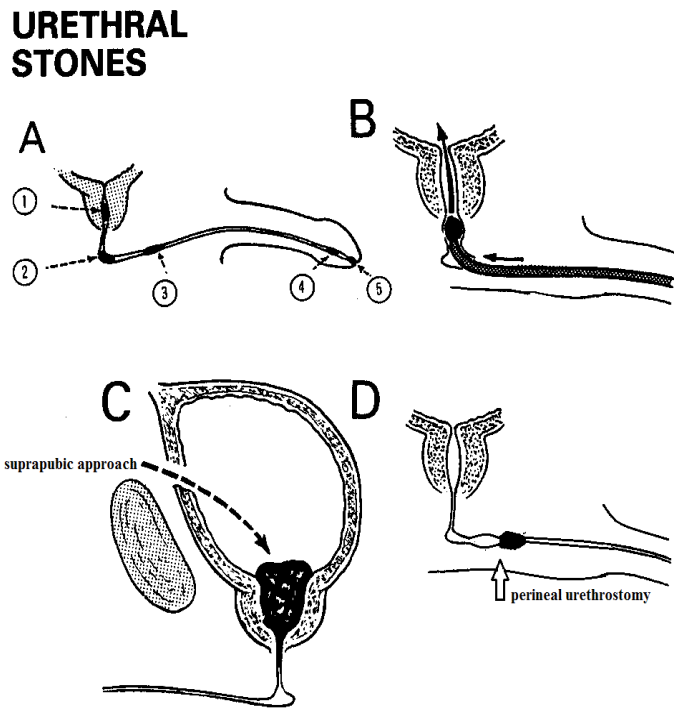


Fig. 27-18 URETHRAL STONES.
A, sites where stones can impact in the urethra. (1) prostatic urethra. (2) membranous (bulbar) urethra. (3) anterior (spongy) urethra. (4) *fossa navicularis*. (5) external meatus. B, remove a prostatic urethral stone by pushing it up with a sound. C, extract an impacted stone by the suprapubic approach. D, a perineal urethrotomy.
N.B. Urethral stones are very rare in women.

27.19 Prostatic enlargement

Urinary outflow obstruction can occur if the prostate is enlarged through hyperplasia, infection or carcinoma, or from bladder neck dysfunction or stenosis.

(a) Presentation
Before urinary flow is completely obstructed, there will be:
(1) Frequency of micturition which interferes seriously with sleep,
(2) Hesitancy (difficult voiding),
(3) Poor urinary stream.
Here you can use selective α-blockers, *e.g.* prazosin, and 5α-reductase inhibitors such as finasteride if they are affordable or available.

Then he may present with various types of complete obstruction:
(1) Acute retention of urine (27.6), perhaps precipitated by a recent drinking bout. (If you catheterize him, he will usually start to pass urine again, but the retention will probably recur).
(2) Chronic retention: the bladder remains distended when micturition is over, and he may dribble urine continuously and painlessly (retention with overflow).
(3) 'Acute on chronic' retention with a poor flow for some time, a large bladder which has recently become painful. This may progress to retention with overflow.

If a patient presents with retention, he may not be well enough for immediate surgery, because:
(1) the acute retention may be the final episode in a long period of obstructive uropathy; the renal function may be impaired and the urine infected.
(2) the factor that has precipitated retention could be a serious illness, such as pneumonia, or fracture of the neck of the femur.

If you try to do surgery while he is in a poor condition, he may not survive the operation. He is more likely to if you wait, drain the bladder for 1-2wks, and let him recover. Investigate the precipitating factor meanwhile.

If you expect to remove the prostate within 2wks, pass a urethral catheter and drain the urine into a closed sterile system. If you have to delay beyond 2wks, insert a suprapubic catheter (27.7) or change the urethral catheter every month.

It is important to try to distinguish between benign and malignant prostatic enlargement. Rectal examination is not entirely reliable; PSA is very specific (but levels also rise with prostatic TB), but acid phosphatase levels are helpful if metastatic disease is present. *Take blood before a rectal examination or 48h after, so as not to get a false +ve result.* Examination under GA gives a good notion of the local extent of prostatic carcinoma, and ultrasound (38.2j) is also very helpful.

(b) Indications for prostatectomy
(1) Difficult voiding and a deteriorating urinary stream.
(2) Frequency of micturition (especially dribbling, due to outflow obstruction, rather than irritation of the trigone).
(3) Acute retention of urine.
(4) Chronic retention with overflow.
N.B. Several medications are now available which can reduce benign prostatic enlargement or improve urinary function; they are expensive and may have significant side-effects.

Conditions which don't *by themselves* indicate prostatectomy include:
(1) Frequency and nocturia.
(2) Haematuria (which is quite common in prostatic hypertrophy).
(3) An increased residual urine (seen on ultrasound).

(c) Contraindications
(1) A patient whose general condition is very poor especially with minimal renal function which does not improve after catheterization.
(2) Severe urinary sepsis.
(3) Senility with dementia (rather than age alone). A very senile old man is likely to be permanently incontinent anyway, and will be better with permanent urethral or suprapubic drainage through a small Foley catheter.
(4) Carcinoma of the prostate is a contraindication to open prostatectomy, but is very suitable for transurethral resection if available. You may be able to manage a malignant prostate with oestrogens or anti-androgens and catheter drainage (27.22) and possibly orchidectomy.

27.20 Open pervesical prostatectomy

It is possible to perform a prostatectomy by the trans-vesical route, by the retro-pubic route or endoscopically *via* the urethra. Another method avoids opening the bladder, but is more difficult. It also needs good lighting, more help, and better post-operative care. Endoscopic resection needs much skill and an expensive resectoscope.

The advantages of the trans-vesical method are:
(1) You can look into the bladder to exclude diverticula, carcinoma, and stones.
(2) You can control bleeding more easily.
(3) When well done, mortality is low.
One of its disadvantages is that it normally requires large quantities of irrigating fluid, although we describe ways of doing without this.

As age advances, the lateral and median lobes of the prostate enlarge. These lateral lobes are joined anteriorly by a narrow anterior commissure, which is the most anterior part of the prostate. As the lateral lobes enlarge, they compress the normal tissues of the prostate around them to form a false capsule, and compress the prostatic urethra from side to side. Posteriorly the median lobe of the prostate enlarges superiorly and extends upwards into the bladder.

You can enucleate the enlarged parts of the prostate by inserting your finger in the plane between the lateral lobes and the false capsule, and shell them out. The prostatic capsule forms the wall of the urethra, and so *if you damage this capsule, the patient will probably get a stricture*. However, if you can master this operation, many elderly men in your community will be forever grateful: you may even find patients come to you from further afield.

N.B. Enucleating the prostate may be impossible if it is malignant, and anyway you don't remove all the prostatic tissue, so this operation is not *suitable for carcinoma.*

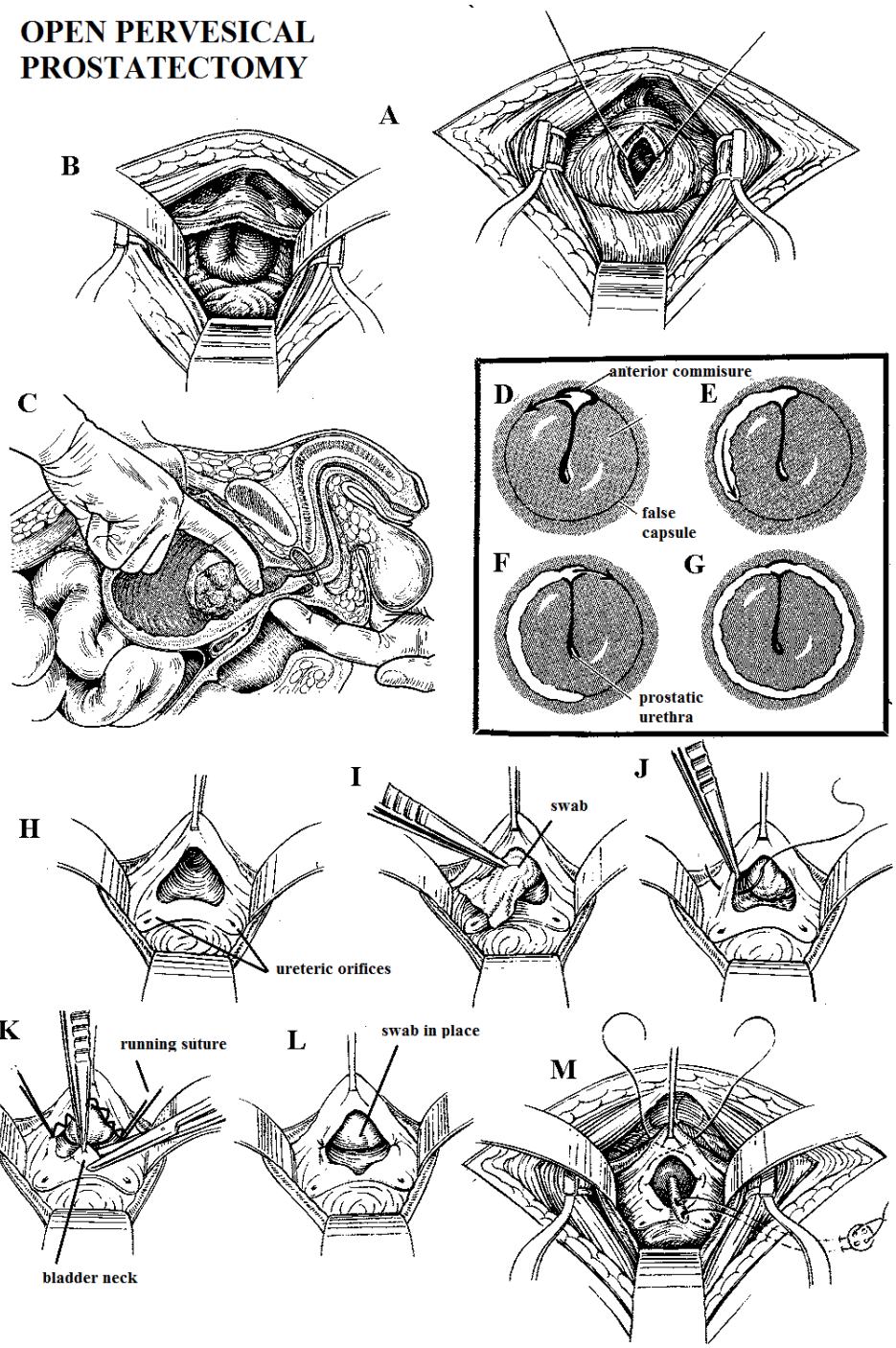


Fig. 27-19 OPEN PROSTATECTOMY.
A, open the bladder between stay sutures. B, inspect the bladder. C, enucleate the prostate by inserting your finger in the internal urethral meatus (you may need to elevate the prostate by an index finger of the opposite hand *per rectum*). D-G, use your right and then your left index finger to open up the plane between the gland and the false capsule. H, the empty prostatic cavity. I, mop out the prostatic cavity. J, place a running suture from 3 to 9o'clock positions to control bleeding. K, cut a wedge out of the neck of the bladder. L, the wedge complete. M, remove the swab and suture a Foley catheter in place.
A, Adapted from a drawing by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland. B-M, After Maxwell Malament, from a publication by Ethicon Ltd, with kind permission.

INDICATIONS
Benign prostatic enlargement (27.19).

CONTRAINDICATIONS
(1) Prostatic carcinoma (27.22) or fibrosis. If, however, you happen to find a carcinoma incidentally, you can open up a sufficient channel to relieve the obstruction.
Avoid a suprapubic catheter, as this risks spreading tumour along its track.
(2) Bladder carcinoma.
(3) Urethral stricture: *deal with this first*.
(4) Neurological impairment causing bladder or detrusor dysfunction. *There is no point in performing surgery if you end up needing a catheter anyway to drain the bladder!*
(5) Severe urinary sepsis: *treat this first*.
(6) Severe renal impairment and debilitation.

EQUIPMENT. Suction and bladder retractors are virtually indispensable. A three-way irrigating Foley balloon catheter is also almost essential. You should hesitate to perform open prostatectomy without these.

PREPARATION
Make sure you have examined the prostate digitally *per rectum*. If you suspect carcinoma, measure the acid phosphatase or PSA, check a pelvic radiograph for metastases, and take a biopsy *via the rectum* (27-20).
Try to perform a cystoscopy (27.3) beforehand. If you have difficulties getting past the prostate, be gentle: force will make it bleed. Look for:
(1) carcinoma of the bladder, especially in areas where *schistosomiasis* is endemic,
(2) bladder stones,
(3) fibrosis of the bladder neck,
(4) diverticula,
(5) benign enlargement of the prostate.
Preferably, have at least two units of blood cross-matched, and an IV infusion running. Treat the patient with prophylactic antibiotics suitable to the sensitivities in your area.

POSITION
Use the supine position and give the table a mild head-down tilt. If you are right-handed, stand on the left side, so that your right hand is in the most convenient position to enucleate the prostate, and so that you can, if necessary, put your left index finger into the rectum.

SOUNDING
If there is not already a catheter *in situ*, and you have not done a cystoscopy, pass a sound to make sure that there is no urethral stricture. If all is well, pass a catheter, and leave c.300ml fluid in the bladder to make it easier to find when you operate.

OPERATION (GRADE 3.4)
INCISION. This depends on whether there is already a suprapubic cystostomy scar. If not, make a Pfannenstiel (11.2) or, less satisfactorily, a 7cm midline incision immediately above the pubis longitudinally between the *rectus* muscles.

If there is a suprapubic cystostomy scar, dissection will be easier if you start in an unscarred part of the wound. Make an elliptical incision round the wound, excise the skin edges and the suprapubic track, and split the *rectus* muscles. Dissecting the peritoneum off the bladder will be difficult, so cover your right index with gauze.

Keeping the pulp of your finger in contact with the pubic symphysis, push your finger into the retropubic space. When you reach the prostate, rotate your finger through 180º and peel the peritoneum off the anterior surface of the bladder.

Insert stay sutures into the anterior wall of the bladder (27-19B), and then incise it in the sagittal plane.
CAUTION! Don't enter the peritoneal cavity. If by mistake you do so, immediately suture it.

Put two fingers of your right hand into the bladder. Feel inside to exclude neoplasms, to feel for stones and the orifices of diverticula. You can easily miss these. Feel the prostate and the internal urinary meatus.

If the prostate is enlarged, and you can easily get your fingers into the internal urinary meatus, enucleate the prostate as described below.

If the prostate is not enlarged, and there is a tight internal meatus which you cannot put your finger into, this is bladder neck fibrosis (27.21).

If the prostate is fibrous or malignant (27.22), and there is no clear plane of enucleation, *don't try to shell out the prostate* as this causes much bleeding, is difficult and unhelpful. Just remove enough tissue with scissors (or diathermy) to leave an adequate channel for the urine. Send this tissue for histology and screen for carcinoma as above. *You should have done this beforehand!*

ENUCLEATION OF THE PROSTATE. Remove the self-retaining retractor and put your index finger into the prostatic urethra. Use your left index finger to split into the recess between the anterior commissure (which should remain *in situ*) and the left lateral lobe of the prostate at the 10o'clock position. Open up the plane between the gland and the false capsule as far distally as you can. Keep in this correct plane to avoid copious venous bleeding. Separate the gland from the false capsule through at least 90º, and preferably 150º. Use your right index finger to repeat the procedure on the right side starting at the 2o'clock position, so as to free the prostate from within its false capsule. There is usually a residual attachment distally. Pull the prostate up into the bladder to make this taut. Divide the attachment with curved dissecting scissors.
CAUTION!
(1) Divide the attachment *close* to the prostate, or you may damage the internal sphincter which surrounds the membranous urethra, deep and superficial to the perineal membrane.
(2) Preserve the anterior commissure. Damage to either may lead to incontinence of urine or a stricture.

Remove the lateral lobes and the median lobe, by bringing it into the bladder with your index finger. If it is still lightly attached proximally to the mucosa of the bladder, separate it with scissors. Removing each lateral lobe separately may be easier. One will bring the median lobe with it.

If the patient is very obese, or muscular, you may be unable to reach the lower border of the prostate. You can push it upwards with your opposite index finger in the rectum while you enucleate the prostate from above. If you have to do this, cover your left hand with two gloves, and protect your forearm with a sterile towel under the drapes; otherwise, get an assistant to do this.

When you have removed the lateral lobes, feel the inside of the prostatic cavity, to make sure that no masses have been left behind (27-19H).

CAUTION! You can easily leave a large mass of adenoma behind, so compare one side with the other. Use your fingers, sponge holders, or vulsellum forceps to grasp and twist off any remaining pieces of prostate.

ENLARGE THE BLADDER NECK. First check the position of the ureteric orifices. Cut a wedge out of the bladder neck in the 6o'clock position level in between the ureteric orifices (27-19K,L). Excess mucosa of the bladder may overhang the prostatic cavity, and if left may produce a valve-like effect leading to retention of urine (27.21).

CONTROL BLEEDING. Put a tight gauze pack in the prostatic cavity (27-19I). After 3mins, take it out and assess the amount of bleeding. Insert a running suture inferiorly from the 3 to the 9o'clock positions (27-19J), taking care to avoid the ureters. Then put a purse-string absorbable suture in the floor of the bladder, around what was the internal meatus (27-19M). Blow up the balloon of a 50 or 75ml Foley catheter, until it fits snugly in the prostatic bed (usually 30-50ml is required). This will help to stop bleeding. Then tighten up the purse-string round it to hold it in place (27-19M) where it will remain if the balloon is inflated enough.

Alternatively, if bleeding is still brisk, tightly pack the prostatic cavity once more, and leave the pack in place for a full 15mins. Then remove it. If the prostatic cavity is still bleeding, remove the purse-string and the catheter, and irrigate the prostatic bed. Then re-insert the balloon of the catheter snugly in the prostatic bed.

Insert a #2 monofilament suture through the abdominal wall and the bladder, and then through the holes in the catheter to hold it in place. Knot it over a button outside the abdominal wall (27-19M). If the patient is confused post-operatively, this will prevent him from pulling out the catheter, even if the balloon bursts.

IRRIGATION. The purpose of irrigation is to remove blood clots, which encourage infection and block the drainage tube. It is, by far, best to use a 3-way irrigating Foley catheter. Introduce fluid down one channel, and let it drain through another.

If you have a shortage of drainable urine collecting bags, or you cannot be sure the bags are surveyed properly, it may be better to let the urine drain freely into a large narrow-necked plastic bottle.

Remove the catheter after 8-10 days. Alternatively, you can use 2 large (Ch20-24) ordinary Foley catheters, one urethral and the other suprapubic, passed through a separate stab incision.

Introduce fluid through the suprapubic catheter, and drain it through the urethral one. Remove the suprapubic catheter when the fluid is no longer bloody, usually >3-4 days. If you leave it longer, it tends to leave a track which leaks. You can improve diuresis by insisting the patient drink at least 4L water/day, or by using furosemide 40mg bd, but *make sure the patient still drinks plenty of fluids!*

You will need about 10L fluid for irrigation. This can be:
(1) IV saline, which is expensive and will be needed for other purposes,
(2) Sterile 3·8% sodium citrate (which is no better than saline),
(3) Mannitol,
(4) Sterile tap water or, better, distilled water. The disadvantage of this is that it may enter the circulation through the prostatic sinuses and cause water intoxication, and if it is not pyrogen-free, it may result in rigors.

Teach your nurses to milk the catheter hourly, until all the clots have gone, usually in 2-4 days. Continue irrigation just fast enough for the urine to be pale pink. It is usually necessary for 24-48h. You will need about 4L in the first day, and less the next day. You may need to continue irrigation until the 4th day.

CAUTION! Don't raise the irrigation bottle too high. If it is >60cm above the bladder, and haemostasis has been poor, the fluid in the bladder may enter the circulation, especially if the outflow catheter is obstructed. Keep the drainage bottle on or near the floor, to make use of gravity.

Deflate the balloon on the 3rd day, unless there is much bleeding; if so, wait until bleeding stops. The prostatic cavity will then become smaller naturally, and there will be less danger of secondary haemorrhage.

CLOSURE. Close the bladder with two layers of continuous absorbable suture. Insert a drain in the retropubic space, through a separate stab incision, below or at the side of the previous one, and secure it to the skin. Close the anterior rectus sheath with continuous absorbable suture. Close the skin.

N.B. Prostatectomy by a suprapubic approach avoids opening the bladder, but access is more difficult. Do this if you can get an expert to teach you.

DIFFICULTIES WITH OPEN PROSTATECTOMY

Besides the normal anaesthetic risks, and those listed below, the post-operative difficulties you may meet include epididymitis, septicaemia, deep vein thrombosis (relatively unusual in low- and middle-income countries), ileus (12.16), uraemia and oliguria, post-operative shock, and bladder tamponade.

If there is bleeding from the prostatic bed after return to the ward, within 48h of the operation (reactionary haemorrhage, not uncommon), all that is usually necessary is to keep the catheter clear by milking it hourly. Speed up the irrigation, and wash out the bladder. Start an IV infusion of saline. If the Hb falls <70g/L, transfuse blood. If copious frank bleeding persists, return to theatre, reopen the wound, and control bleeding from the prostatic bed (27-19J).

If there is a distended painful bladder and no urine drains, this is CLOT RETENTION. This is one of the most feared complications of prostatic surgery, and occurs within the first 72h. There has often been severe bleeding, and the blood in the bladder has clotted and obstructed the catheter, usually because the irrigation has been neglected. You may find urine leaking from the extravesical drain or even from the wound. Start transfusion if necessary. Inject 50ml sterile water or saline into the bladder and immediately aspirate it.

CAUTION! Don't inject more than 50ml, or you may burst the balloon, and *don't try this method more than once*.

If this fails to dislodge the clot, try using heparinized water; or deflate the catheter balloon, and push it further into the bladder and wriggle it about. *Don't pull the catheter out:* it may be impossible to re-insert without GA.

If all fails, return to theatre, remove the catheter under GA, and perform a urethro-cystoscopy if possible. Otherwise gently insert a new 3-way Ch24 catheter, using an introducer, and wash out the bladder through this.

If even this fails, re-open the bladder to remove the clots.

If there is bleeding 8-12 days post-operatively (secondary haemorrhage, quite common), it usually settles. Many cases are not severe, and will stop on their own.

If bleeding does not stop, re-insert a urethral catheter, making sure (by ultrasound if possible) that the balloon is sitting nicely in the prostatic bed and it is inflated maximally. Wash the bladder through this until the fluid is nearly free of blood. Leave the catheter *in situ* some days and repeat bladder wash-outs till the urine stays clear.

If bleeding persists, or you cannot re-insert a catheter, take the patient back to theatre and reopen the prostatic bed to control bleeding (27-19J).

If there is incontinence of urine, you can give reassurance that this is almost certain to improve during the next 3 months.

The symptoms of urge incontinence are common in patients who had these symptoms pre-operatively; they usually resolve spontaneously. *If you have damaged the membranous urethra by enucleating a prostate which did not have a clearly defined capsule* (27-19F), *incontinence may be permanent.* So, if the prostate feels fibrous or carcinomatous beforehand, *don't proceed*.

If a suprapubic fistula develops, it will close spontaneously unless the outflow is still obstructed. So drain the bladder with a urethral catheter for 10 more days. If the leak persists, go back to close the bladder wall again.

If a recto-urinary fistula develops, drain the bladder with a urethral catheter. If this fails, perform a diverting colostomy (11.6).

If there is a further episode of urinary retention some months or years later, perform a cystoscopy to see what is the cause. It may be a urethral stricture, regrowth of the prostate or bladder neck stenosis. You may have to make a wedge resection of the neck of the bladder. This is very unlikely to happen if you routinely excise a wedge of tissue from the back of the neck of the bladder when you remove the prostate.

If you find bladder diverticula, leave them: they will become smaller now that you have relieved the obstruction, unless they were very large.

If there is an inguinal hernia present, you may repair this (18.2) at the same time as the prostatectomy, if you are having good results and your complication rates of bleeding and blocked catheters are minimal. Otherwise repair the hernia later.

27.21 Bladder neck problems

There are 2 causes of urinary obstruction in which the prostate feels normal rectally, with no sign that it is enlarged, but in spite of this, urine cannot pass. Between 5-10% of cases of supposed prostatic obstruction are like this. The patient is usually younger than the others. If you can easily pass a sound, you thereby exclude a stricture.

Perform a CYSTOSCOPY: you will find that the bladder is obviously obstructed, as shown by trabeculation, a hypertrophied inter-ureteric bar (27-7A), and perhaps diverticula. But you cannot see any sign of an enlarged prostate. Instead, the posterior lip of the urinary meatus is unduly prominent (difficult to see with an ordinary cystoscope).

(a) Dyskinesia: Try to make this diagnosis before you operate, and use prazosin 0·5-2mg bd. (Beware: this is an α-blocker and may cause postural hypotension.) This can be a manifestation of HIV disease.

(b) Bladder neck fibrosis: you may find, when you open the bladder, that the prostate is not enlarged. Instead, there is a tight internal meatus, which you cannot put your finger into. One cause is *schistosomiasis*, affecting the trigonal submucosa.

(c) Bladder neck resection (GRADE 3.4)
METHOD
Get adequate exposure: you cannot expose the internal urinary meatus through a short incision. Approach the inside of the bladder as for open prostatectomy (27.20).

Put a self-retaining retractor into the bladder, open it, and tilt the head of the table downwards slightly. Use a Langenbeck retractor, or a bent copper retractor, to draw the anterior wall of the bladder against the pubis, so that you can see the internal urinary meatus.

Identify the orifices of the ureters. Make deep cuts in the bladder neck in the 5 & 7o'clock positions, sloping towards one another so as to excise a wedge of the bladder neck (27-7C, 27-19K). The cuts must go deep enough to divide the circular fibres of the neck of the bladder. This will then spring open, and the obstruction will be relieved.

If *schistosomiasis* is the cause, there will be more fibrosis, and you will be cutting fibrous tissue rather than muscle.
CAUTION!
(1) *Take great care not to injure the ureters*, as they enter the bladder. A wise precaution is to pass a Ch7 catheter (or a feeding tube) into each ureter.
(2) If you find diverticula, leave them alone.

POST-OPERATIVELY, leave a 3-way Foley catheter in place, or insert a urethral and a suprapubic catheter and irrigate the bladder as for a prostatectomy (27.20).

27.22 Prostate carcinoma

Prostate carcinoma is the commonest male cancer >65yrs.

(a) Presentation is commonly with:
(1) Bone pain, which is not necessarily in the back. 80% of patients have metastases when they present. Perineal pain suggests extensive local disease.
(2) Difficulty passing urine, acute or chronic urinary retention.
(3) Weakness in the legs, due to metastases involving the *cauda equina*.
Because prostate carcinoma is so common, it is fortunate that it can be controlled, for a time, by the cheap hormone therapy, diethylstilbestrol. You may, however, have difficulty persuading patients to continue to take it, because of its oestrogen effects.

Making the diagnosis on the basis of the clinical findings, a raised serum acid phosphatase and radiographic findings, is not wholly reliable; also, microscopic differentiation of carcinoma from benign hyperplasia can be difficult. PSA estimation is, however, highly specific.

(b) Rectal examination. A normal prostate feels smooth, symmetrical, and firm, usually with a median groove and mobile rectal mucosa. A carcinomatous prostate is hard, nodular, and asymmetrical; its median groove is often obliterated, and the rectal mucosa may be fixed to it. Late locally extensive disease may extend to the pelvic wall, form a band round the rectum, and fix the pelvic tissues. Sometimes, you can feel the spread of the tumour in the tissues round the prostate. If possible, confirm these findings by examining the patient bimanually under anaesthesia.

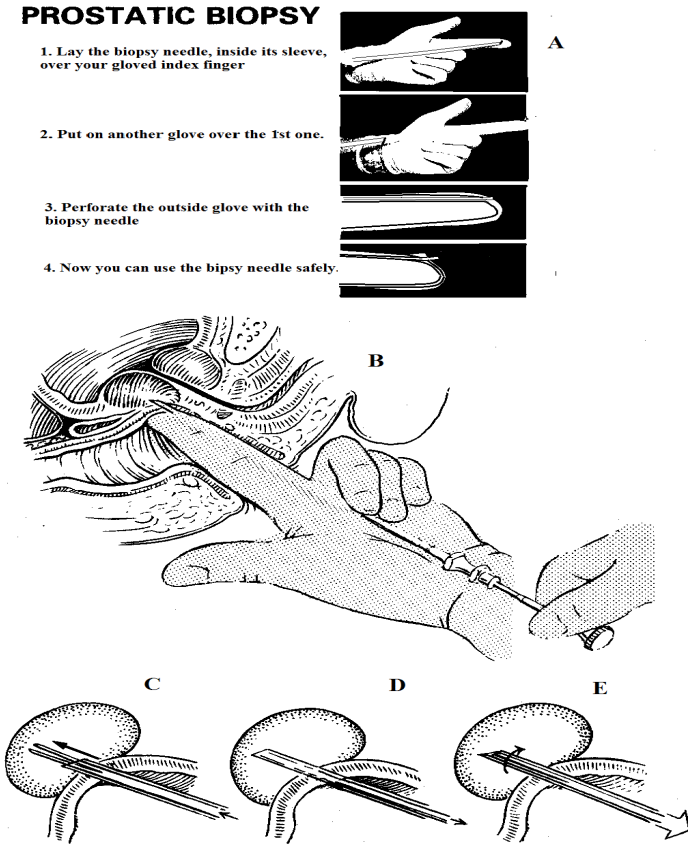

PROSTATIC BIOPSY
1. Lay the biopsy needle, inside its sleeve, over your gloved index finger
A
2. Put on another glove over the 1st one.
3. Perforate the outside glove with the biopsy needle
4. Now you can use the bipsy needle safely.
B
C
D
E

Fig. 27-20 PROSTATIC BIOPSY.
A, holding the sheathed (or Trucut biopsy) needle along the palmar surface of your left index finger, with the tip on the pulp, slip another glove over the biopsy needle and sheath, so that it is sandwiched between the two gloves. Insert your finger into the patient's anus. B, feel each lobe of the prostate, as if you were doing a rectal examination. Using your right hand, push the needle 0·5cm through the rectal mucosa, towards one of the prostatic lobes. Withdraw the stylet. C, insert the biopsy jaws, and push them into the prostate as far as they will go, still keeping the needle along the index finger of your left hand. Remove your finger. D, advance the outer sheath far enough to cover the biopsy jaws. E, rotate the needle, so as to break off the core of tissue that has been grasped, and withdraw it. Lift the core of tissue from the biopsy needle, with an ordinary injection needle, and put it into formalin. If you fail, repeat the process up to 3 times. Then do the same thing with the other lobe. (Using two gloves makes this procedure very much safer.)
After Ghei M, Pericleous S, Kumar A et al. Finger-guided transrectal biopsy of the prostate: a modified, safer technique. Ann Roy Coll Surg Eng 2005; 87:386.

(c) Radiographs. Look for lytic and sclerotic (typical but less common) metastases in the pelvis, and lumbar spine. Paget's disease and osteoarthritis produce similar bony symptoms, but show different X-ray changes.

(d) Ultrasound (38.2j) will demonstrate an irregular enlarged infiltrating prostate, but cannot detect an early carcinoma, except if you can use a rectal probe.

(e) Special tests. If the carcinoma is present, the PSA is >2U; where it has spread beyond the prostatic capsule, the serum acid phosphatase will be >3 King-Armstrong units. A normal level does not exclude it. A persistently raised level supports the diagnosis, and suggests metastasis. It has no prognostic value; its main use is in diagnosis. It falls in response to treatment, and rises when the disease reactivates.

CAUTION! Take blood before you do a rectal examination, or wait for 48h. If you take it immediately afterwards, you will get an abnormally high reading.

(f) Needle prostate biopsy (GRADE 1.3)
To confirm malignancy (or, rarely, TB), biopsy the prostate with a trucut biopsy needle (24-3) through the rectum (27-20). This is not difficult but you may miss the malignancy unless it is quite advanced. Ultrasound will help you guide the needle, but unless you have a rectal ultrasound probe, you will need to use a perineal route. In spite of passing through the rectum, serious infection is rare. You should, though, use a single prophylactic dose of gentamicin beforehand, and make sure the rectum is empty.

(g) Cystoscopy. Examination under GA is necessary with cystoscopy; this is particularly useful in distinguishing carcinoma of the prostate from carcinoma of the trigone of the bladder infiltrating the prostate. You may feel a grating sensation as you pass the cystoscope through the carcinomatous prostate, or you may see puckering of the apex of the trigone, or submucous nodules in the bladder (late signs).

(h) Differential diagnosis includes:
(1) benign prostatic hyperplasia,
(2) tuberculous prostatitis,
(3) bladder carcinoma infiltrating the prostate,
(4) bladder neck fibrosis (27.21),
(5) stricture of the prostatic urethra (27.9).

(i) Management
If the disease is still confined to the prostatic capsule at age <65yrs, there might be benefit from radical radiotherapy if available.

If there is severe bone pain from metastatic disease, localized radiotherapy may give much relief.

If the disease is advanced or you cannot access radiotherapy, treat with hormonal manipulation. The original drug, diethylstilbestrol, an oestrogen analogue, is cheap. Start with 1mg od, and if this fails to control the symptoms increase it to 5mg od. There is an 80% chance that there will be a good response.

However, there are many side-effects: fluid retention, gynaecomastia, impotency, hot flushes, osteoporosis, and itching. You cannot use this drug in the presence of heart failure.

Anti-androgens, such as flutamide 250mg tds, are more acceptable, but are more expensive, and interfere with the action of anti-coagulants and anti-convulsants. Other anti-androgens such as ketoconazole, cyproterone acetate, and spironolactone have too weak actions to be effective.

A good alternative is finasteride, a 5-α-reductase inhibitor, but this is even more expensive.

An alternative hormonal treatment is a subcapsular orchidectomy (27.26), which will avoid the need for life-long drug treatment and its side-effects. Most patients object to castration, but they may accept a reduction in size of their testicles! You must therefore discuss such treatment carefully.

If there is retention of urine, start hormonal treatment and pass an indwelling Foley catheter. Leave it in for at least 3wks before trying to remove it, and if then there is still urinary retention, consider leaving it in for a further 3wks. Hormonal treatment will usually make the prostate shrink enough to allow micturition. If, unusually, it does not, try to arrange endoscopic resection of the prostate.

If this is quite impossible, retain a urethral or suprapubic catheter. *Don't try to remove the prostate by the open method* (27.20): this is difficult, because it does not shell out properly, leaves tumour behind and risks spreading it, and there will be severe bleeding.

If the urine becomes infected, treat with appropriate antibiotics; in itself infection will not influence the outcome of the carcinoma.

27.23 Epididymo-orchitis

(a) Acute epididymo-orchitis presents with an acute painful swelling of one testis and epididymis. A few cases are viral: the important viral cause is mumps which occurs in adolescents. Occasionally it may be due to brucellosis. The testicle is acutely tender, may be associated with a hydrocoele, lies in its normal position, with pain partially relieved by lifting the testicle in a scrotal support.

DIFFERENTIAL DIAGNOSIS AND TREATMENT
Suggesting testicular torsion (27.25)**:** a sudden severe pain in the groin or lower abdomen, associated with an exquisitely tender testis lying horizontally in the scrotum.

Suggesting acute epididymo-orchitis: a history of urethral discharge; either only the epididymis is affected, or the epididymis more than the testis, usually unilaterally. If possible, analyze the discharge.

Treat with doxycycline 100mg bd for 2wks. If acquired after anorectal intercourse, use ciprofloxacin 500mg bd for 2wks.

Suggesting epididymo-orchitis secondary to urine infection: there is no urethral discharge, but pain, frequency and burning of micturition. If possible, culture the urine and use an appropriate antibiotic.

Suggesting *schistosomiasis*: small 3-5mm nodules in the tail of the epididymis. The *vas deferens* is usually palpable. Treat with one dose of praziquantel 40mg/kg.

Suggesting *filariasis* (34.14)**:** tender swelling of the cord, epididymis and testes, with oedema and inflammation of the scrotal skin. The *vas deferens* is usually *not* palpable. Treat with doxycycline 200mg od for 6wks.

Suggesting mumps orchitis: the testis is affected but the epididymis appears normal. Usually unilateral but occasionally bilateral, not necessarily simultaneously. Ask if others in contact at school have had similar illness. The boy will settle without treatment, but if both testes are affected, he may become infertile, especially if he is a young adult.

(b) Chronic epididymo-orchitis is common in the sexually active and in elderly men with urinary outflow difficulties. It is the result of:
(1) Previous untreated or imperfectly treated attacks of acute epididymo-orchitis, which is usually gonococcal or chlamydial.
(2) Non-specific urinary infection (usually due to *E. coli*).
(3) *Schistosomiasis* which is usually confined to the tail of the epididymis, causes little pain, less swelling and only mild tenderness.
(4) Tuberculosis, which results in a firmer swelling.

DIFFERENTIAL DIAGNOSIS AND TREATMENT
Suggesting testicular tumour: painless swelling of the testis (27.28).

Suggesting tuberculous epididymitis: swelling with low-grade pain affecting the epididymis and later the testis, not necessarily but often associated with tuberculosis elsewhere, which does not resolve with the usual antibiotics.

Suggesting bacterial epididymo-orchitis: chronic urinary symptoms predominate: use the appropriate antibiotics related to urine culture.

Suggesting *schistosomiasis* or *filiarisis*: a globular mass in the spermatic cord, which may extend along its whole course; this suggests fibrosis which may not respond to medical treatment.

If there are severe recurrent attacks of pain which don't settle, even when the urinary infection is controlled and the flow good, consider orchidectomy (27.26) taking the epididymis necessarily with the testis.

27.24 Hydrocoeles in adults

The cause of many hydrocoeles is unknown; they may be one of the manifestations of *filariasis.* A hydrocoele in an adult is different from one in a child, which is almost always a patent *processus vaginalis* (18.5). You must be able to differentiate a hydrocoele from an inguinoscrotal hernia by being able to get above the hydrocoele. Normally a hydrocoele transilluminates (if the skin is not too dark and the light bright).

If a hydrocoele is small, leave it, but if it is so large it makes walking or sex difficult, or is an embarrassment, operate. If it is only a modest size, turn it inside out (*i.e.* evert it out of the *tunica vaginalis*), so that the fluid which it secretes drains into the lymphatics; if it is very large, excise its secreting surface. Aspirating smaller hydrocoeles every 3-6 months is popular with many patients but is not a cure, and may result in septic complications. As an alternative, sclerotherapy is useful, but may also result in septic complications. To prevent recurrence, insert 1ml 2% phenol with 10ml lidocaine into the hydrocoele cavity for every 200ml aspirated.

The scrotum is famous for its tendency to bleed post-operatively, develop a haematoma and swell up bigger than it ever was before! Complete haemostasis is important. Sharp dissection causes less bleeding than blunt dissection. A continuous locking absorbable suture over the cut edge of the tunica is more reliable than interrupted sutures.

The sac may be tense, enlarged and tender if there is a scrotal abscess (6.21); occasionally it is not tender but contains a necrotic brownish paste associated with an atrophic or absent testis. The cause of this is unclear, but may be related to *filiariasis*, and is found in Northern India, known as 'kichad'. In either case, incision and drainage suffice.

Very occasionally in a lax hydrocoele, the testis is abnormal and may contain a tumour: in this case use an inguinal approach (27.26), and be prepared to perform an orchidectomy.

(a) Eversion of a hydrocoele
(JABOULAY OPERATION) (GRADE 2.1)

INDICATIONS. Moderate-sized thin-walled hydrocoeles.

METHOD. Hold the scrotum in such a way as to stretch the skin over the hydrocoele. Make a curved incision (27-21A). Carefully deepen the incision through to the *tunica vaginalis* and pierce this to let out the fluid.

The incision should only be just big enough for you to deliver the testis through the opening. When you have done this, evert the *tunica vaginalis* and suture it behind the testis, in such a way that the testis cannot return into its sac (27-21A).

CAUTION! Make sure you evert the whole sac. If it has an upward prolongation and you fail to evert this, the hydrocoele will recur. To evert it, put a haemostat into it, pull it inside out completely, and pass a mattress suture through it. Make sure there is no bleeding from the *tunica vaginalis*; insert another row of sutures if necessary. Stretch the layers of the *dartos* to make a pouch for the testis: this will allow any fluid to get absorbed and not re-collect. Close the wound in 2 layers, taking the *dartos* muscle in the first, and the skin in the second, with continuous short-acting absorbable suture, to secure haemostasis.

(b) Excision of a hydrocoele sac (GRADE 2.2)

INDICATIONS. A large hydrocoele with a greatly thickened wall, perhaps covered with a layer of cholesterol crystals. For enormous hydrocoeles, *e.g.* in *filariasis*, perform a reduction scrotoplasty (27.34).

METHOD. If the hydrocoele is very large, resect the redundant scrotal skin, but leaving enough skin remaining to re-create a scrotum. Excise the entire sac of the hydrocoele, except for a cuff 1cm deep around the testis and epididymis (27-21C). The cut edges will bleed profusely. Pass a simple continuous haemostatic suture along the whole cut edge.

CAUTION!
(1) The cleft between the testis and epididymis may be greatly enlarged by the extension of the hydrocoele, so take care not to injure or remove the epididymis with the *tunica vaginalis.*
(2) Operate gently and control bleeding before you close the skin.
(3) Avoid diathermy because you can overheat the spermatic cord and cause thrombosis of the blood vessels, resulting in testicular infarction.

If mild bleeding persists, insert a drain through the wound. Close it in 2 layers, as above.

If the scrotal sac remains large and floppy, secure it to the anterior abdominal wall between 2 pieces of gauze, for 48h.

(c) Difficulties with hydrocoeles

If the patient is a neonate or child, operate through the groin because this type of hydrocoele is actually usually a hernia with fluid in a patent *processus vaginalis* (18.5).

If there appear to be bilateral hydrocoeles, start with the bigger side first; the apparent second hydrocoele may subsequently disappear.

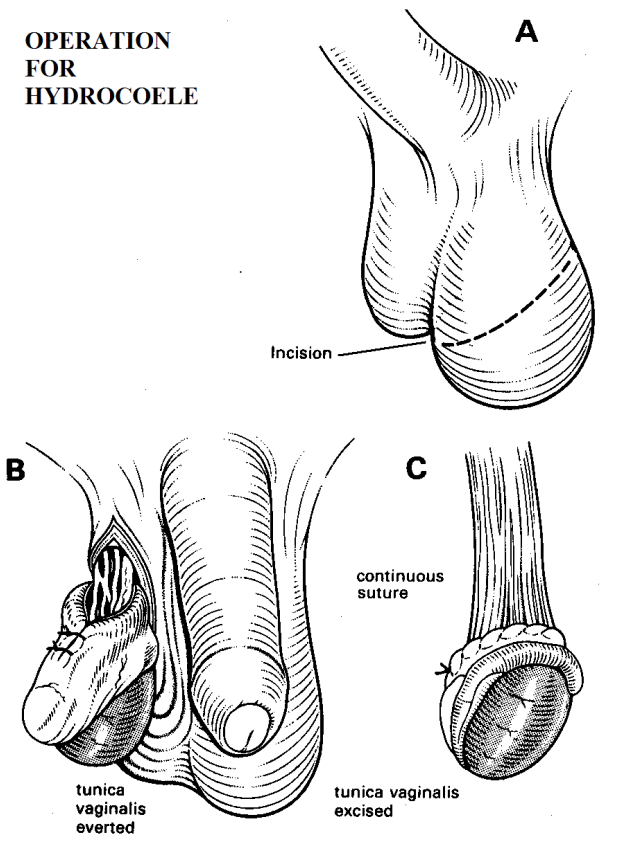


Fig. 27-21 OPERATION FOR HYDROCOELE IN ADULTS.
A, make an elliptical incision, and excise the skin within the ellipse, after which you can turn the *processus vaginalis* inside out (B), or you can excise it (C).

If when you open the sac you find bowel, this is an indirect inguinoscrotal hernia (18.2). Start again through the groin to identify the sac at the internal ring, and proceed as for a hernia repair. Use your scrotal incision as the site for a drain, because of a high risk of bleeding in such cases.

If when you open the sac you find altered blood (an old haematoma), drain it but *don't attempt eversion or excision*. Leave in a corrugated drain (4-14B) and give prophylactic cloxacillin 500mg qds; the scrotum will eventually shrink as it heals by fibrosis.

If you find a malignant-looking testis, start again through the groin and clamp the testicular vessels, and proceed to orchidectomy (27.26). Get histology done.

If the scrotum swells alarmingly post-operatively, suspect a haematoma. Open the wound, evacuate the blood and clots, and leave in a drain. Use prophylactic cloxacillin 500mg qds.

27.25 Testicular torsion

Torsion of the spermatic cord (strictly speaking) is a surgical emergency which needs operation without delay. *It is never wrong to operate to exclude torsion*.

Occasionally, the *tunica vaginalis* ends abnormally high up the spermatic cord, so that the cord can twist and obstruct the blood supply to the testis and epididymis. When this abnormality (the 'bell-clapper' testis) is present, the testis usually hangs transversely, and does so on both sides.

Unless you can untwist the cord, the testis will necrose, become purple-black, and fill the *tunica vaginalis* with a blood-tinged fluid. If you don't relieve the torsion before this happens, the testis will atrophy. If you are going to save it, you must operate within 6h of the start of symptoms.

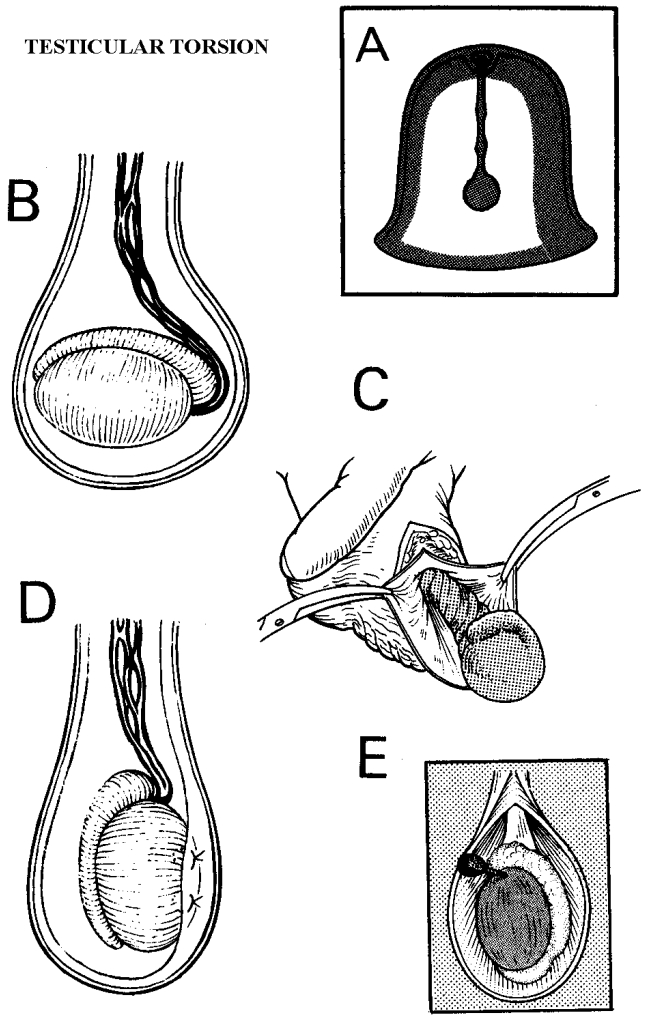


Fig. 27-22 TESTICULAR TORSION (or strictly speaking, the cord). Occasionally, the *tunica vaginalis* ends abnormally high up the spermatic cord, so that it can twist and obstruct the blood supply to the testis and epididymis. This abnormality is the 'bell-clapper' testis. A, is a bell with its clapper. B, is such a testis, with a long intravaginal spermatic cord hanging horizontally. C, torsion of the spermatic cord. D, the cord untwisted and the testis anchored to prevent recurrence. E, torsion of the appendix testis, which has no function.

(a) Presentation
Torsion can occur at any age, but is more common in the first year of life and in adolescence (12-19yrs).

Typically, a teenage boy wakes with sudden severe *pain in the groin or lower abdomen* (owing to the surface neurotome representation of the testis), rather than in the testicle. Often, there is severe nausea and vomiting, and rarely a fever. The testis becomes tender and swollen, and the skin of the scrotum may become red.

(b) Differential diagnosis
Suggesting epididymo-orchitis: the slower onset of pain in an adult with history and signs of past or present sexually transmitted infection, infected urine, or prostatic symptoms (27.23). Usually a mild fever is present. The testis lies normally but may be quite swollen and tender.

CAUTION!
(1) *Don't diagnose anyone as suffering from epididymo-orchitis under 30yrs* especially if there is no history of sexual contact. If there is any doubt, operate. *You will not do harm by exploring orchitis, but antibiotics will not relieve torsion! Don't rely on a Doppler test*: the presence of blood flow shows the testicle is still viable, but not that it has not twisted!
N.B. Except for the orchitis of mumps, orchitis is very rare in young boys.
(2) Treat painful *testicular* enlargement in an infant or neonate as torsion.

(c) Manipulative reduction is temporary and is never adequate or definitive treatment. *Don't try it without putting local anaesthetic into the spermatic cord*. It is only appropriate in the 1st 2h of symptoms, but may buy you time if you cannot operate within 6h. The testis twists medially in 66% of patients and laterally in 33%. Even if it is successful, torsion may recur, so proceed to operation and fixation early.

(d) Orchidopexy for testicular torsion
(GRADE 2.3)
INCISION
Make a vertical incision in the scrotum, over the area of tenderness. Cut through the subcutaneous tissue and fascial layers down to the *tunica vaginalis.* Open it. You will find it filled with blood-tinged fluid, and you will see the twisted spermatic cord. Untwist it.

If there seems no chance that the testis will survive, check that it is really infarcted by cutting into it: if it does not bleed, transfix the spermatic cord and remove the testis (27.26).

If you are not sure if the testis is viable or not, wrap it in a warm moist swab and inspect it again after 5mins. Bright bleeding when you incise the *tunica albuginea* is a promising sign. If you are in doubt, preserve it, especially if the symptoms have lasted <12h, and it has twisted <1½ times.

Once you are confident there is some life in the testis, fix it to the *tunica vaginalis* to prevent recurrence (27-22D). Align the testis with its head placed superiorly. Anchor it laterally with 2 non-absorbable sutures.

CAUTION! Whatever the viability of the twisted testicle, you must *always* anchor the contralateral testis in the same way: the anatomical abnormality is usually bilateral.

Close the *dartos* and skin in 2 layers with continuous short-acting absorbable suture.

(e) Difficulties with torsion of the testis

If you find that the testis is not twisted, but instead there is a small twisted structure attached to it, this is TORSION OF AN APPENDIX TESTIS (27-22E). These are the remains of the Müllerian duct. Tie off the twisted structure and excise it.

If in an infant and especially a neonate, you find that the whole *tunica vaginalis* with its contained testis and spermatic cord is twisted (supravaginal torsion), deal with it in the same way.

If a maldescended testis strangulates, **y**ou can mistake it for a strangulated hernia (18.6).

If the torsion reduces spontaneously, advise that it can recur and that bilateral orchidopexy is still necessary.

If you find only one testis, the other having been lost to neglected torsion, perform an orchidopexy on the remaining testis.

27.26 Orchidectomy

You may occasionally need to remove the testes, either because of infarction, chronic suppuration or a tumour (a sarcoma, seminoma or teratoma, 27.28). This is much safer than biopsying it, which may spread the tumour.

Or, in treatment of prostate carcinoma (27.22), you can perform a bilateral subcapsular orchidectomy. This removes the testicular function whilst retaining the semblance of testicles.

Don't mistake mumps orchitis or epididymo-orchitis for a tumour. This causes rapid enlargement, and some pain (which is minimal in the case of a tumour). Mumps orchitis may cause little pain, so if you are in doubt, wait for a few days rather than remove the testis.

You will have to exert some traction on the cord, but beware of its upper end slipping out of the clamp and retracting out of sight. Take great care to secure haemostasis before you close the wound. If possible, apply diathermy to the smaller bleeding vessels, and tie off the larger ones. Close the skin with continuous horizontal mattress absorbable sutures.

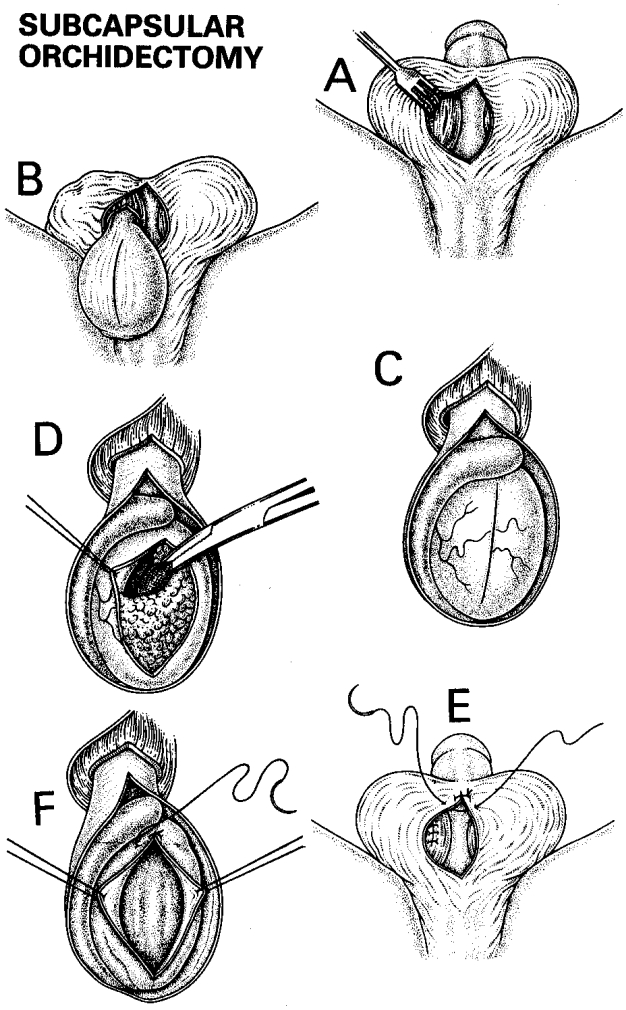


Fig. 27-23 SUBCAPSULAR ORCHIDECTOMY FOR PROSTATE CARCINOMA. A, make a longitudinal incision in the patient's scrotum. B, evaginate the testis. C, incise the *tunica albuginea*. D, separate the substance of the testis from the inner surface of the *tunica albuginea*. E, close the *tunica*. F, close the skin.
After Rob C, Smith R. Operative Surgery, Butterworth 1981 p.667-8 with kind permission.

(a) Subcapsular orchidectomy for prostatic carcinoma (GRADE 2.3)

Raise the scrotum, and incise the stretched skin and *dartos* muscle, to expose both testes (27-23A). Evaginate each testis with its coverings, and incise its *tunica vaginalis* vertically to expose the testis and epididymis (27-23B). Incise the visceral *tunica* vertically over the globe of the testis. Use sharp and blunt dissection, to separate the substance of the testis from the inner surface of the *tunica albuginea* (27-23C). Control bleeding carefully at the upper testicular pole. Remove all testicular tissue, and close the *tunica* with continuous 3/0 absorbable sutures (27-23D). Close the scrotum in 2 layers with continuous 3/0 short-acting absorbable sutures, without inserting a drain. After 2-3wks, blood clot in the *tunica* will become organized to form a small palpable nodule, not unlike a small testis.

(b) Orchidectomy for infarction or chronic suppurative infection (GRADE 2.2)

Expose the testis by incising through the scrotum (27-23A), deliver the testis, clamp the spermatic cord and ligate it with a strong suture. If it is very thick and oedematous, ligate it twice with a fixation suture and divide it. If the area is infected, leave the wound in the scrotum unsutured to drain freely, dress it loosely, and close it by delayed primary suture.

(c) Orchidectomy for testicular tumour
(GRADE 2.5)

You will need to remove the cord with the testis, so open up the inguinal canal as for a hernia with an inguinoscrotal incision (18-7A). Pick up the cord within its covering of *cremaster*. Apply a soft bowel clamp to it (27-24A), before you do anything else. Deliver the testis from the scrotum by pushing it up from below. If the tumour is large, you will have to extend the opening in the external inguinal ring.

If you feel a hard irregular mass which is not chronic epididymo-orchitis, doubly transfix and tie the cord proximal to the clamp as near the internal ring as you can, and excise the testis.

N.B. Don't cut through the scrotum as you will then open up a different lymphatic drainage field for the tumour.

You should try to administer adjuvant chemotherapy if testicular malignancy is confirmed (27.28).

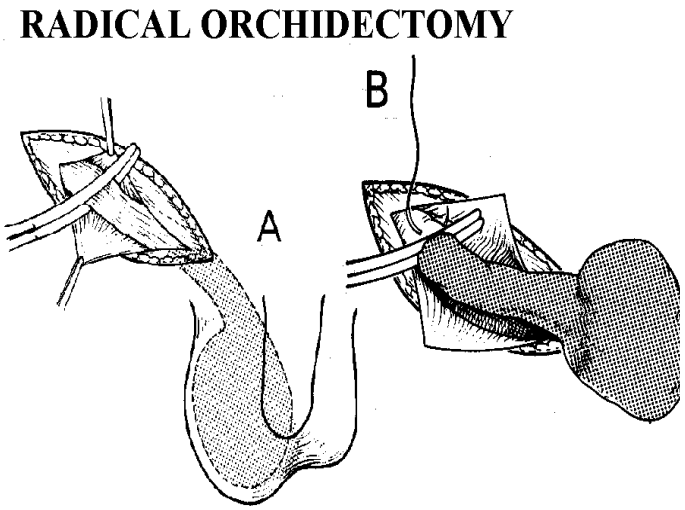


Fig. 27-24 TOTAL ORCHIDECTOMY FOR TUMOUR.
A, apply a soft (non-crushing) bowel or arterial clamp to the cord before you do anything else. B, make an inguinoscrotal incision and remove the cord with the testis through the groin.
After Blandy J. Operative Urology, Blackwell 1978 Figs 15.37,40 with kind permission.

27.27 Undescended or maldescended testes

About 3% of neonates and 0·5% of older boys have a testis missing from the scrotum, and in 20% it is missing on both sides. An incompletely descended testis lies along the track of descent of the testis: the common sites for it are in the inguinal canal, or inside the abdomen. A maldescended testis, however, may lie in the supra-inguinal pouch, just superior to the external ring deep to the membranous part of the superficial fascia, in the perineum, or on the medial aspect of the thigh. The distinction between incomplete descent and maldescent may be difficult.

A testis which is absent from the scrotum will produce hormones but not *spermatozoa*. So if neither of the testes is in the scrotum, there will be normal secondary sex characteristics, including potency, but infertility. The less complete the descent, the greater the chance of infertility. If a testis is absent on one side only, there will probably be fertility, but the misplaced testis is more easily injured. Spermatogenesis is normal in an incompletely descended testis and in a maldescended one for up to 2yrs. This is also the age at which nearly all 'retractile testes' will have settled normally into the scrotum. So wait until 2yrs before you advise orchidopexy. If neither of the testes is in the scrotum by this time, orchidopexy may still produce adequate fertility. If one testis descends, fertility may still improve with orchidopexy. Its main effect, however, remains psychological.

Maldescended testes are usually functional, which can be brought down more readily. Unfortunately, the evidence for orchidopexy improving fertility is still inconclusive.

If neither testis is present in a neonate scrotum, the possibilities are commonly:
(1) Retractile testes;
(2) A genuine undescended or maldescended testis; or rarely,
(3) An intersex state. These are complex and include true hermaphroditism and the adrenogenital syndrome.

If the testes tend to lie at the external ring or even a little within it, especially in the cold, but can be manipulated downwards (retractile testes), consider this normal. By puberty they will probably be permanently in the scrotum. Advise the parents to check for this.

If at any stage the testes *cannot* be manipulated into the correct position in the scrotum, you should perform an orchidopexy, especially above the age of 2yrs.

If there is a hernia and an undescended testis on the same side, perform an orchidopexy at the same time as the herniotomy; it will be much more difficult later.

(a) Special tests
If you cannot feel the testis in the groin, you might be able to see its position with ULTRASOUND (38.2).

(b) Orchidopexy for undescended testis
(GRADE 2.4)
METHOD
The important part of the operation is getting enough cord length; the method of fixation is less important. Aim to mobilize the spermatic cord to obtain more length, and then to fix the testes in their normal places. Deal with incomplete descent and maldescent in the same way.

To mobilize the spermatic cord, make a 5cm incision from just lateral to the mid inguinal point to the root of the scrotum. Open the inguinal canal from the external to the internal ring. Find the spermatic cord containing the spermatic vessels and vas. Use sharp dissection with fine instruments to mobilize the cord and testis from all surrounding structures, including the *dartos* muscle. If there is a hernia (common), dissect off the sac, divide it and ligate it as in a herniotomy (18.5).

If you now have enough length of cord to bring the testis down into the scrotum, put a stay suture through the lowest point of the testis.

To fix the testis in the scrotum, insert your finger into the scrotal sac to open it up. At the point where the testis will reach easily, incise gently through the scrotal skin but not through the *dartos*. Then insinuate an artery forceps between skin and *dartos* and so make a pouch big enough to accommodate the testis.

Make a small hole in the *dartos* and catch the suture you placed in the testis, and pull this with the testis through into the pouch. The trick is not to make the hole in the *dartos* too big so that the testis pops out again! Make sure that the cord is not twisted. Fix the testis with monofilament in the *dartos* pouch, outside the muscle which prevents it retracting back inside. Close the scrotal skin with chromic catgut.

CAUTION!
(1) Be sure to discuss with the parents what you can achieve by operation on an incompletely descended testis.
(2) Take great care not to damage the blood supply of the testis.
(3) At the end, explain the outcome of the operation.

(c) Difficulties with orchidopexy
If, at operation, you cannot bring down the testis fully, and you have mobilized the spermatic cord and vas as much as you can, and have removed any hernia sac present, open the internal inguinal ring further on its medial side and open the peritoneal cavity. Dissect the cord from the peritoneum covering it for about 5cm. This is not easy, so *don't attempt it unless you have had some experience* because you might devascularize the testis.

If you fail to bring down the testis fully, a 2-stage procedure will be necessary. Fix the testis as far down as you have been able to bring it.

If there are bilateral incompletely descended testes, consider carefully whether you wish to tackle this side as well. There may be a need for further mobilization at a later stage, but this is unlikely to improve fertility.

27.28 Testicular tumours

Nearly all tumours of the testis are malignant. In Africa, most of them under 16yrs are rhabdomyosarcomas; seminomas are very rare. Elsewhere, most are seminomas or teratomas or a combination of both, and are usually seen between 20-45yrs.

Presentation is with:
(1) A large, usually painless, testicular swelling.
(2) An abdominal mass.
(3) Gynaecomastia and breast tenderness (rare), in which case gonadotrophin production by the tumour may result in a +ve pregnancy test.

(a) Diagnosis
The testis is large, hard, smooth, heavy, and not tender. It loses its normal sensation early. On standing, it usually hangs lower than the normal one, unlike testes with inflammation or torsion, which are usually pulled higher. The vas, prostate and seminal vesicles are normal, and the cord likewise until late, but occasionally (10%) a hydrocoele is present. Early, the epididymis is normal; later, it is flattened or hidden in the tumour.

Feel for deposits above the umbilicus on the same side, in the liver, and above the clavicles. X-ray the lungs. Get an ultrasound of the abdomen looking for para-aortic nodes (38.2h) and liver metastases (38.2b).

(b) Differential diagnosis:
(1) epididymo-orchitis (27.23),
(2) hydrocoele (27.24),
(3) haematocoele following trauma,
(4) testicular torsion (27.25),
(5) epididymal cyst,
(6) tuberculoma (rare),
(7) syphilitic gumma (rare).

(c) Management
Don't delay the operation. Under GA, palpate the abdomen for para-aortic masses. Perform a radical orchidectomy through an inguinal incision (27.26).

If there is a seminoma, arrange post-operative radiotherapy to the upper abdominal para-aortic nodes as soon as possible, even in the absence of demonstrable metastases, though there is an 80% chance of cure by orchidectomy alone. If there is a teratoma, treatment with post-operative cyclophosphamide, vincristine, methotrexate and dactinomycin gives a 95% chance of complete cure. Use this even if there are metastases. Get expert advice!

CAUTION!
(1) If the presentation is with symptoms which might be due to metastases, *don't fail to examine the testes. You can easily miss a small primary tumour.*
(2) *Don't remove the testis through the scrotum*.
(3) *Don't try to biopsy the lesion*.

27.29 Circumcision

Recommending circumcision to reduce HIV spread in a region where HIV prevalence and incidence of penile carcinoma are high, is controversial. It may be useful before sexual activity starts, because the exposed glans develops, with time, an epithelium supposedly more resistant to spread of the virus. An argument against circumcision is that the foreskin is useful raw material for plastic surgery. *Never do it when there is hypospadias present* (33.9).

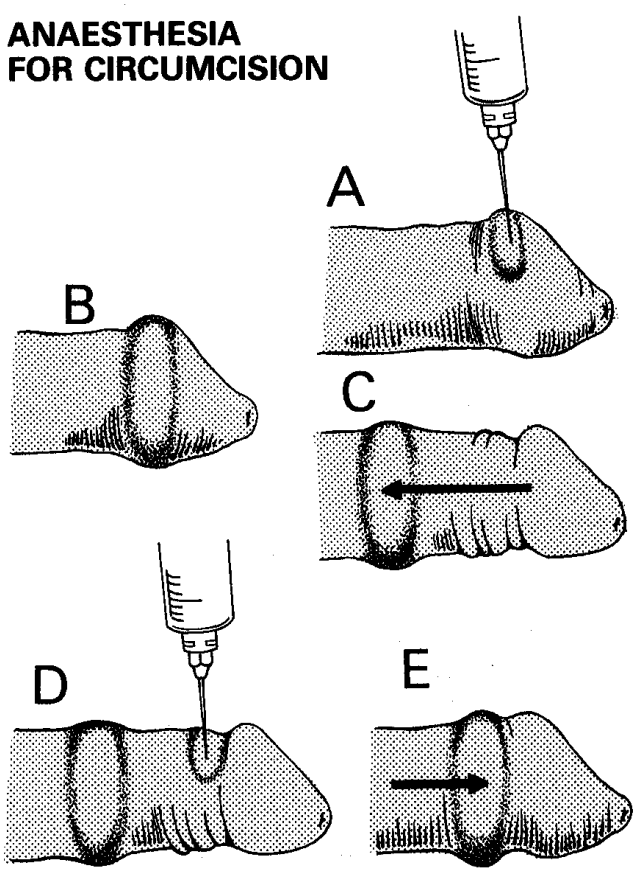


Fig. 27-25 ANAESTHESIA FOR CIRCUMCISION FOR PATIENTS >15YRS.
A,B, with the foreskin forward, infiltrate a ring of LA without adrenaline at the site of section. C, pull back the foreskin. To do this, you may have to infiltrate a little more and make a dorsal slit. D,E, infiltrate another ring of solution, at the site of section, just behind the glans. Pull the foreskin forwards again, and all is ready for circumcision to start.
***N.B. Under 15yrs and in HIV disease, use a caudal block or ketamine*.**

A STORY FROM A CITY HOSPITAL
Horrible shrieks used to be heard from the theatre whenever circumcisions were being done. A caring surgeon put Fig. 27-25 on the wall, whereupon they stopped completely, to the gratitude of the staff and the patients! 10yrs later this event was still remembered.

There may be a significant risk, in some cultures, that a child has circumcision done crudely by traditional practitioners under non-sterile conditions. You may be able to advertise a course in good surgical technique for traditional practitioners or nurses!

Consider carefully if the risks of surgery are justified. It is usually reasonable to delay the procedure till a child is over 2yrs. Often you can free up an apparently tight foreskin, and mobilize it over the glans with gentle traction.

(a) Indications for infants
(1) Phimosis.
(2) Irreducible paraphimosis.
(3) Religious requirements.

(b) Contraindications for infants
(1) Child <24h old or premature.
(2) Penile anomaly, including hypospadias.
(3) Family history of bleeding disorder.

(c) Indications for adults
(1) Phimosis; recent balanitis, such as that due to diabetes.
(2) Paraphimosis.
(3) A suspected malignant lesion confined to a small area of the foreskin.
(4) Copious penile condylomata.
N.B. Consider carefully if circumcision is indicated in HIV+ve patients.

ANAESTHESIA
(1) In a neonate, sucking on the mother's breast with some sugar on the nipple is usually good preparation. Topical anaesthetic cream is useful if you are performing many circumcisions. Wait for it to take effect!
(2) If the boy is <15yrs, use ketamine.
(3) If >15yrs and he is HIV-ve, use LA (27-25). You will have to cut the inner and outer skin of the foreskin, so you will have to infiltrate them both. With the foreskin forward, infiltrate a ring of anaesthetic solution *without adrenaline* at the site of section (27-25A,B). Pull back the foreskin (27-25C). To do this, you may have to infiltrate a little more solution and make a dorsal slit in it. Infiltrate another ring of solution at the site of section just behind the glans (27-25D). Pull the foreskin forwards and do the circumcision (27-25E). Or, perform a dorsal nerve block at the base of the penis at 2 and 10o'clock positions with 1ml of 0·25% bupivacaine or 1% lidocaine, about ½cm below the skin.
(4) A caudal block is an alternative <5yrs: using a 0·9mm spinal needle, under sterile conditions, introduce up to 4ml 1% lidocaine through the sacral hiatus into the sacral canal to the level of S3.

TIMING
Neonatal circumcision is best done at day 6 of age, and before day 10. Check that the child has passed urine, and look carefully for hypospadias or epispadias.

RESOURCES
Consider carefully if, because of financial incentives from programme donors, the resources for performing circumcisions are being diverted from other essential surgery!

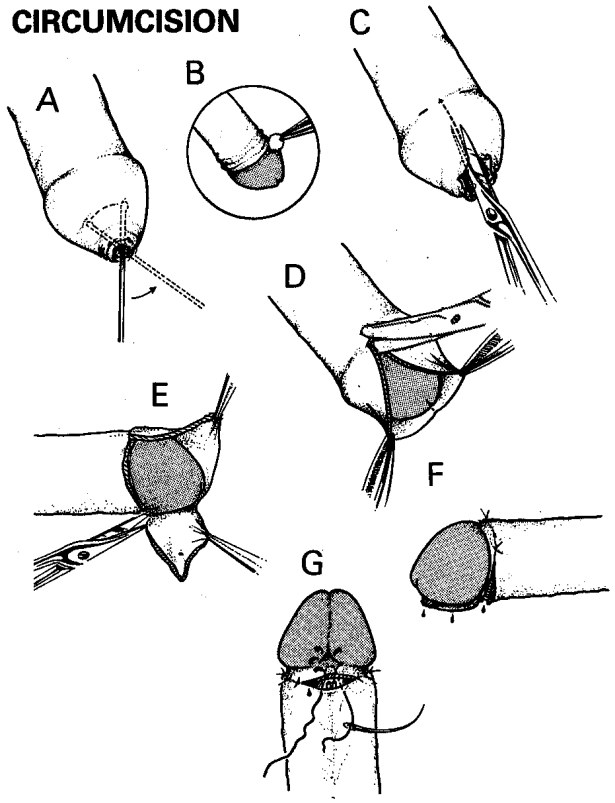


Fig. 27-26 CIRCUMCISION.
A, free up the foreskin. B, retract the foreskin and clean the glans. C, make a dorsal slit. D, cut the outer skin only with scissors. E, cut the inner layer with scissors leaving a 2mm fringe at the corona. F, suture the fringe of foreskin to the skin of the shaft. This will control most of the bleeding. G, control frenal bleeding with this suture.

METHOD (GRADE 2.1)
Use a warmed room for neonates, and have good lighting available.

Insist on adequate bathing pre-operatively. If adhesions or a tight phimosis prevent you pulling the foreskin back, use a probe to free up the foreskin from adhesions to the glans (27-26A). Retract the foreskin if you can (27-26B), clean thoroughly underneath it and then pull it forwards again. With the penis in its normal relaxed position, feel for the bulge of the corona of the glans. Hold the foreskin laterally on both sides with haemostats, and make sure the space between foreskin and glans is free with the probe.

Use a haemostat to crush a midline portion of the dorsal foreskin and then blunt-ended scissors to cut a slit in exactly this position up to the level of the bulge in the corona (27-26C). If you have been unable to clean underneath the foreskin, do so now.

CAUTION! Make sure that the point of the scissors is not in the meatus.

IN A NEONATE, you can use the Plastibell device, GOMCO or Mogen clamps. *Be careful, however, about using any special circumcision clamps and tools which don't allow you to see the glans. Never use diathermy.*

CIRCUMCISION DEVICES

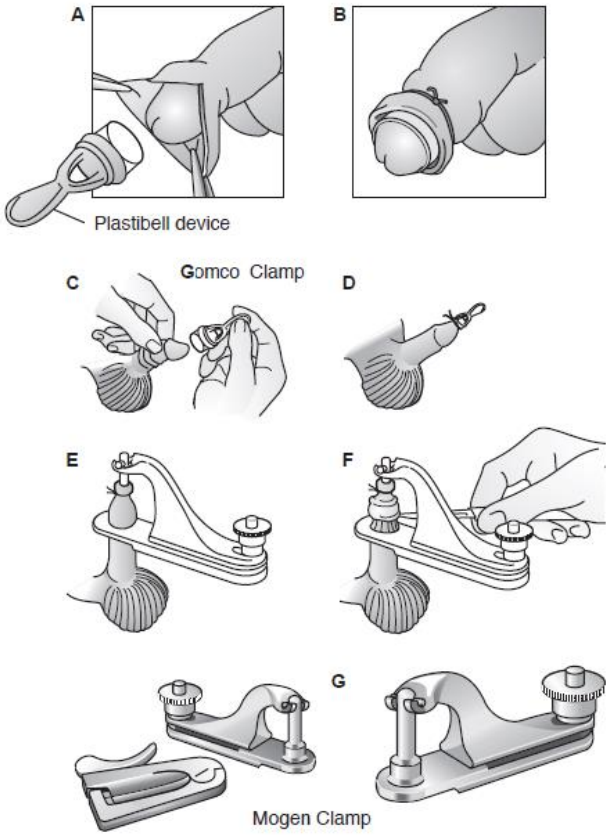


Fig. 27-27 NEONATAL CIRCUMCISION DEVICES.
A, applying the Plastibell device. B, suture tied over the foreskin. C, GOMCO clamp. D, apply the lubricated bell over the glans. E, tie the foreskin to the bell handle. F, fit the bell in the yoke of the clamp. After tightening the thumbscrew, cut off excess foreskin with a scalpel. G, Mogen clamp.

The **Plastibell** is a plastic bell with a groove around its edge. Place it under the foreskin and over the glans, and then tie a suture in the groove. The suture occludes the blood supply to the foreskin, which ultimately drops off along with the bell. It therefore may be more painful.

Make the dorsal slit long enough to accommodate the device (27-27A). Select the Plastibell cap that best fits the glans (6 sizes are available). A cap that is too small will not let you remove sufficient foreskin and a cap that is too big will cause you to remove too much foreskin. A proper fit is a cap that fits halfway down the glans. *The groove must always be distal to the corona.*

Separate the two sides of the dorsal slit incision to expose the glans, put the Plastibell device over the glans, and then close the foreskin over it. *Don't pull the foreskin too tightly*, as this might result in compressing the urethra. Then tie the string securely around the foreskin in the groove at the ring-shaped base of the bell (27-27B).

Some devices have a handle that helps secure the device and you must snap this off once you have secured the device in place. You can cut off any excess foreskin beyond the suture, taking care not to cut the suture itself.

The child goes home with the bell part of the device still attached. It will fall off with any foreskin left beyond the suture in 6-12 days.

If the device does not fall off during this interval, you must remove it promptly. Sometimes oedema will trap the plastic ring on the penis, so you will need a guide and ring cutter to cut the ring off.

If the child has difficulty urinating, it may be because the ring has slipped proximally from the glans onto the penile shaft; this can result in venous obstruction and a compartment syndrome leading to necrosis of the glans.

N.B. The advantages of the Plastibell are that you don't cut the frenulum and there is no free incised edge of foreskin that can bleed.

The **GOMCO clamp** (27-27C) is a 3-part device (base plate, bell, lock). The clamp crushes c.1mm of the foreskin circumferentially, while the bell protects the glans by separating it from the inner preputial mucosa during removal of the foreskin. The bells come in 3 sizes; for a correct fit, the edge of the bell should reach the frenulum and minimally extend over the corona, slightly stretching the preputial skin. (For a newborn, use a clamp with a 1·3cm hole.) A dorsal slit is not always necessary.

Place the bell inside the foreskin and over the glans, lubricated with a little lubricant jelly (27-27D), and remove the two haemostats. Make sure that the apex of the dorsal slit is visible above the rim of the bell. *Too small a bell may injure the glans and fail to protect the corona.*

Slip the handle of the bell through the circular opening of the base plate, without letting the foreskin slip off. Tie the foreskin in place (27-27E). Check to see that the foreskin is not twisted and remains relaxed proximal to the corona. *If it is taut, you can remove too much foreskin or even cut into the glans; if it is too loose, you will remove too little foreskin!* You may need to adjust the base plate: this is the time to do so.

Make sure that the crossbar at the top of the bell sits squarely in the yoke of the clamp so pressure is evenly distributed around the bell (27-27F). Tighten the thumbscrew until snug to crush the foreskin between the bell and base plate; then cut the foreskin at the base plate using a scalpel (27-27F). Carefully remove any remaining tissue in and around the groove that connects the clamp and bell. Leave the clamp secured for 5mins and then unscrew it to remove it.

N.B. You need care in adjusting the bell and base plate correctly, so not too much or too little foreskin is cut off: don't try repeating the procedure with the clamp. If the clamp is old and worn, it will not give a neat closure!

If too little foreskin is removed, proceed to a formal circumcision.

If too much foreskin is removed, you may need a skin graft to cover the defect.

The **Mogen clamp** (27-27G) technique is the quickest, and therefore least painful. You don't need to make a dorsal slit, although this may help to visualize the glans.

Use your thumb and first finger of the non-dominant hand to grasp the foreskin firmly below the tip of the haemostat and push the tip of the glans out of the way.

Slide the Mogen clamp anteriorly to posteriorly just above your fingers to protect the glans when applying the clamp. Place it at the same angle as the corona with the hollow side facing the glans, so that you remove more foreskin dorsally than ventrally. Before locking the Mogen clamp shut, manipulate the glans to be sure it is free of the clamp. Then close and lock it. Cut off the foreskin distal to the clamp with a scalpel.

CAUTION! The glans is not well protected during clamping and cutting. However, since the clamp only opens 3mm, the chance of trapping the glans is minimal.

FORMAL CIRCUMCISION IN OLDER CHILDREN AND ADULTS

After performing a dorsal cut of the foreskin, cut the outer skin only round the corona (27-26D). Cut the inner layer with scissors, leaving a 2mm fringe at the corona (27-26E). One of the purposes of the foreskin is to provide enough skin to allow the penis to erect, so when you perform a circumcision, *be careful not to take off too much skin*, or the pubic hair will later be drawn up the root of the erected penis. On the other hand, *don't leave too much skin*, or there is the risk of recurrent phimosis.

Use 3/0 absorbable individual ties to control bleeding, and suture the fringe of the foreskin to the skin of the shaft of the penis (27-26F). Finally, control bleeding from the frenal vessels with a special encircling suture (27-26G). Dress the wound with petroleum jelly gauze. No dressing is needed after 24h.

CAUTION!

(1) *Don't cut the glans*.

(2) *Don't use diathermy on the penis*, it can cause gangrene of the whole organ.

(3) *Never use adrenaline in a local anaesthetic for the penis*, this too can cause gangrene.

(4) *Avoid LA in HIV+ve patients*, as post-operative vasculitis and infection may result in gross necrosis (6.21).

DIFFICULTIES WITH CIRCUMCISION

If you cannot separate the foreskin and the glans, because the cleft between them is obliterated, you may find yourself dissecting the glans from the thick adherent foreskin. If necessary, work slowly and carefully with a sharp scalpel. This will leave a raw area. Allow this to granulate on its own, and *don't try to graft it with split skin*.

If an adult has post-operative priapism, sedate him (27.32), but *don't use propofol*.

If there is bleeding post-operatively, find the vessels and insert 1-2 more interrupted absorbable sutures.

If a cutaneo-urethral fistula develops, either from direct injury or because of a periurethral abscess, you can close this if you use a *Dartos* fascia graft.
Measure the fistula, and pass a urethral catheter; decompress the phallus and apply a proximal tourniquet. Insert stay sutures to expose the fistula, and close it using skin flaps (as for hypospadias, 33.9). Then mobilize the *Dartos* fascia to place a graft with suture lines *not* superimposing on each other, using fine 4/0 long-acting absorbable sutures. *Don't use diathermy*.

If a crooked erection develops, there may have been excessive skin removed, or excessive scarring. Excise the contracture and apply a skin graft.

27.30 Phimosis and paraphimosis

(a) Phimosis, in which the orifice of the foreskin is too small for it to be retracted over the penis, improves with the application of topical steroids (betamethasone 0·1% cream) over 6 months; this is successful in 90% of patients in producing an easily retractile foreskin but may fail if there is already scarring present, and especially if there is balanitis xerotica obliterans.

(b) Paraphimosis is where the foreskin has been forcefully retracted, is swollen and stuck behind the glans, so that it cannot be brought forward again. It is common in adolescence, and is fairly common between 8-14yrs. Part of the foreskin is tight, so that it becomes oedematous distally. The oedema may be severe. If it has been present for >6h, the base of the penis may be oedematous also. Try to get the foreskin into its normal place over the glans.

(c) Reduction of paraphimosis (GRADE 1.2)

METHOD
Squeeze the swollen foreskin between the thumbs and index fingers of both your hands, so that the fluid which is making it swell goes up into the tissues of the shaft. If the swelling is severe, wrap layers of gauze coated in sugar over it and squeeze them. When the foreskin is in its normal place, any residual swelling will usually subside in 24-48h. This is easier if you can inject hyaluronidase and lidocaine solution (1500U in 10ml water) into the oedematous foreskin.

After 2-3mins of firm squeezing, when the swelling is much reduced, push the glans proximally with your thumbs, and draw the foreskin over it with your fingers.

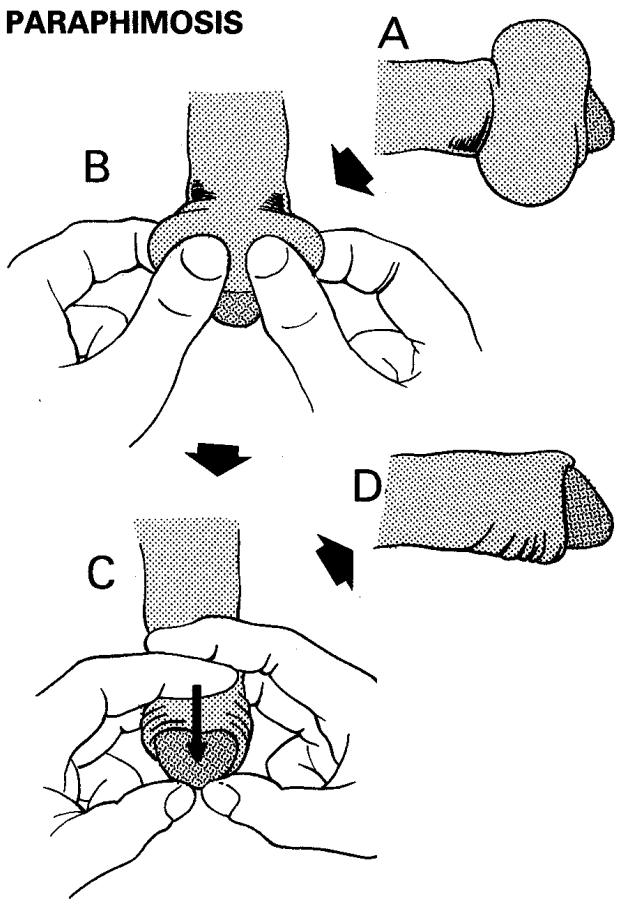


Fig. 27-28 PARAPHIMOSIS.
A, paraphimosis as the patient presents. B, use the thumbs and forefingers of both your hands to squeeze the swollen foreskin. C, slide the roll of foreskin forwards over the now thinner glans. At the same time push the glans back. D, successful reduction.

(d) Difficulties with paraphimosis

If you fail to reduce the paraphimosis, the constricting band is too tight, so proceed to circumcision (27.29). Where the paraphimosis is chronic, there develops a fibrous band just distal to the corona: you must excise this to get a good cosmetic result (a straight penis).

27.31 Meatal stricture

Strictures of the meatus have quite a different cause and prognosis from the gonococcal urethral strictures (27.9). You may see them in children or adults, and they may be congenital or acquired. The most important acquired cause is infection associated with instrumentation and catheterization. In adults a meatal stricture may be due to the skin disease called balanitis xerotica obliterans (BXO, *lichen sclerosis atropica*). This also involves the foreskin, so there is usually an associated phimosis also.

Dilation should nearly always precede meatotomy.

If a meatal stricture develops in a child, give the mother a plastic rod to keep it dilated. The rounded plastic containers used for disposable needles are very suitable. Ask her to lubricate it with vegetable oil. If necessary, perform a meatotomy.

If a meatal stricture develops in an adult, first try dilation.

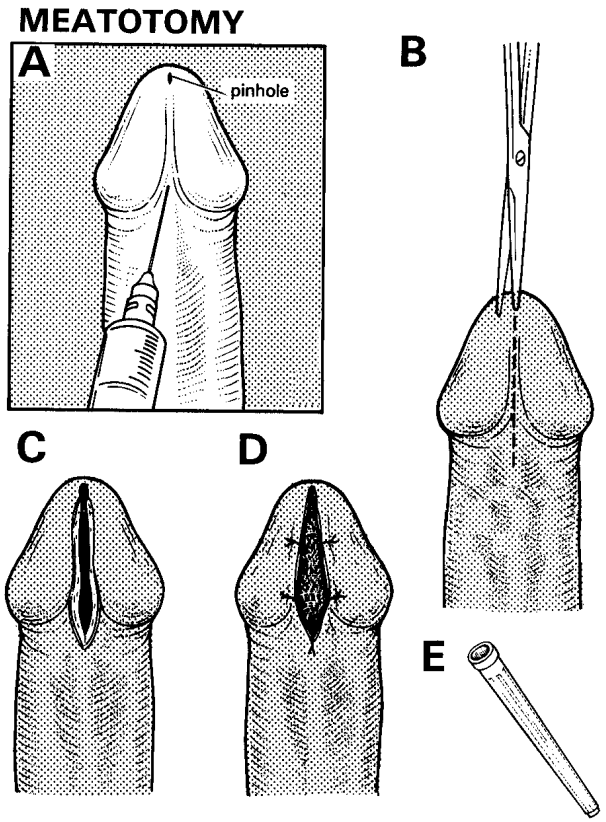


Fig. 27-29 MEATOTOMY.
A, although GA is preferable, you can infiltrate the frenulum and glans with lidocaine. B, introduce one blade of a pair of fine scissors into the urethra. C, cut 1·5 cm beyond the stenosis. D, suture the mucosa to the skin. E, plastic cover of a disposable needle makes a good dilator.
Kindly contributed by Neville Harrison.

MEATOTOMY (GRADE 1.4)

Either use ketamine, or use LA directly into the frenulum and the glans immediately under it. Alternatively, use a ring block of the base of the penis (27.29).

Put one blade of a pair of fine-pointed scissors into the meatus, and cut its ventral aspect in the midline. To stop the stenosis recurring, cut 1·5cm beyond the meatus, and suture the mucosa to the skin with long-acting absorbable. Use the first 2 sutures on either side as retractors to open the wound, and expose the mucosa of the urethra. Insert a suture at the apex of this incision to control bleeding from the frenal artery. The meatus may re-stenose, so use a dilator regularly.

If there are signs of BXO, hydrocortisone cream will help to prevent recurrence. Apply it into the meatus from the nozzle of a small tube, which will then act as a dilator. You should continue this for 3 months.

27.32 Priapism

If there is a sustained painful involuntary penile erection, either rigid or merely turgid, this is priapism, which is a urological emergency. If this is secondary to sickle cell disease (in which it is common), leukaemia, use of sildenafil or similar medication, or some neurogenic cause, such as paraplegia, it usually settles with sedation and without impairing subsequent erections. The danger is that if priapism from any cause persists too long, the *corpora cavernosa* may become ischaemic and fibrotic, so that the patient becomes permanently impotent.

So treat him early by:
(1) Administering an ice-cold saline enema: do this twice if the first time fails.
(2) Using sedation *(not propofol)*; always try this first.
(3) Irrigation of the *corpus cavernosum*.
(4) Injection with an adrenergic drug.
(5) Creation of a shunt between the *corpus cavernosum* and the *corpus spongiosum* (27-31D).

(a) Sedation. First try heavy sedation with pethidine and chlorpromazine. This will usually provide a cure, especially if the priapism is due to sickle cell disease. *Don't persist too long with sedation and anaesthetics*.

(b) Irrigation. Infuse lidocaine around the base of the penis to make a ring block. Introduce two 19G needles through the glans penis into each *corpus cavernosum* (27-30A) and use one for aspiration and one for irrigation with saline. Aspirate 5-10ml of blood: if it is bright red, this indicates a high-flow obstruction, which implies a fistula already exists between the *corpora.*

There is no danger of a compartment syndrome and this type of priapism will resolve spontaneously. If the blood is dark and venous, there is a low-flow obstruction and risk of ischaemia. Unless detumescence occurs within 30mins, proceed with injection of adrenergic drugs.

(c) Adrenergic drugs. Use an adrenergic drug sooner rather than later: through one of the needles already inserted, inject 1ml of 1:10,000 adrenaline diluted in 1L 5% glucose or 0·9% saline every 5mins up to 1h till detumescence occurs. Massage the penis to distribute the drug through both *corpora*.
If this is not effective, especially if priapism has been present >4h, proceed to make a fistula.
CAUTION! Monitor the blood pressure every 5mins.
Never use adrenaline in the subcutaneous penile tissues!

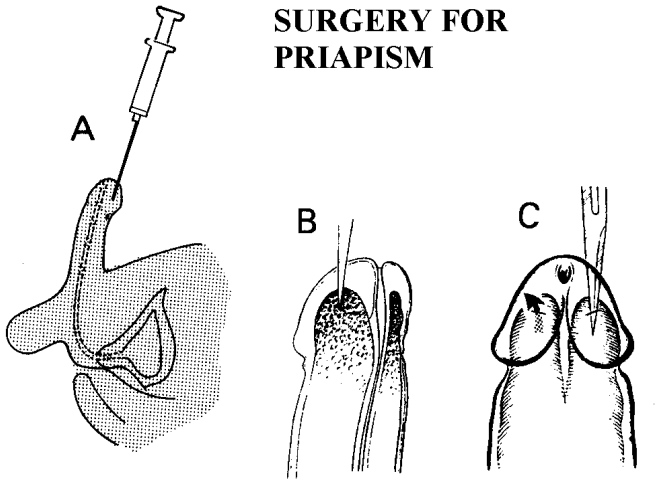


Fig. 27-30 SURGERY FOR PRIAPISM.
Aim to create fistulae, which will allow the *corpora cavernosa* to drain into the glans penis. In priapism the turgid *corpora cavernosa* project up under, and into, the glans penis. A, aspirating the *corpora cavernosa* through the glans. B and C, use a sharp knife to make an incision on each side between the glans and the *corpora*. Alternatively, use a needle and syringe, or a trucut biopsy needle.
After Rob C, Smith R. Operative Surgery: Urology, Butterworth 1981 p.584 with kind permission.

(d) *Corpus spongiosum* shunt (GRADE 2.4)
Introduce a trucut biopsy needle (24-3) in the closed position through the same skin wound, and push it through the glans to the coronal septum (between the glans and the *corpora cavernosa*), taking care to avoid the urethra. Note that the ends of the erected *corpora cavernosa* project well into and under the glans. You may need considerable force.

Open the biopsy needle by extending its obturator blade through the septum, and close it by pushing the sheath over the fenestrated tip, twist it, and remove it. You should withdraw tissue consisting of fibrous septum, and the contents of the *corpus cavernosum*. Repeat the manoeuvre in another site close by and then do the same thing with the other *corpus cavernosum*. By doing this, you will create 2 fistulae on each side.

The penis should now become flaccid rapidly, and remain so. Control brisk bleeding from the puncture site by pressure or with a figure-of-8 absorbable suture. *There is no need for a pressure dressing, nor for an indwelling catheter.*

If you don't have a trucut needle, make cuts (27-30B,C) on each side with a #11 blade, rotate the blade when within the *corpus* and withdraw it.

27.33 Penile carcinoma

Squamous cell carcinoma of the foreskin is common in India and Africa. The patient is ≥40yrs, and is almost always uncircumcised. Presentation is with a swollen and often infected foreskin, or with phimosis secondary to it. The tumour spreads until the whole foreskin is involved, after which it invades the *corpus spongiosum*, and later the *corpora cavernosa*. It also spreads to the inguinal lymph nodes, which ultimately ulcerate, so that he dies from sepsis, toxaemia, or sudden haemorrhage from the femoral vessels. The carcinoma does not obstruct the urethra completely, nor is it painful at first, so that commonly presentation is late.

In all but the earliest lesions, which can be treated by radiotherapy if available, you will have to amputate the penis, either partly or completely. A partial amputation is usually possible, and although it is not easy to do well, it is not nearly as difficult as a complete amputation. This is a difficult, bloody, major operation.

Partial amputation is very effective, and the prognosis is good, even if you have to perform a block dissection of affected inguinal nodes (17.8).

After a partial amputation, it is still possible to urinate comfortably. After a complete amputation, it is necessary to squat to pass urine. If you fashion the perineal meatus carefully, it will function well, and is unlikely to stenose.

The method of complete amputation described here leaves the *crura* of the *corpora cavernosa* attached to the bone, which simplifies surgery. Recurrence in the residual *crura* is rare. *There is nothing to be gained by doing a block dissection of unaffected inguinal nodes prophylactically*.

(a) Examination
Feel the shaft of the penis carefully to determine the exact extent of the tumour. If necessary, split the foreskin under GA, so that you can examine the glans adequately. Feel the inguinal nodes. They will probably be enlarged by sepsis, so you may find it difficult to know if they have metastases in them or not. Treat with cloxacillin and review if the nodes remain after 2wks. Fine needle cytology is very useful here.

(b) Biopsy
Ulceration alone does not always mean carcinoma is present. So always take a biopsy, and wait for histological proof before you amputate the penis. *Don't amputate until you have received the report*. Surgery is not urgent, because the tumour is very slow-growing.

(c) Differential diagnosis: includes condyloma acuminatum and the following:

Suggesting chancroid: an irregular undermined painful ulcer caused by *Haemophilus ducreyi* with a grey base. Enlarged groin nodes. Often associated with HIV disease.

Suggesting primary syphilis: a round or oval painless ulcer, often found under the foreskin, but which does not penetrate or destroy it. Enlarged groin nodes. Serological tests may be -ve early, but are always +ve later.

Suggesting venereal warts: small multiple lumps 1-3mm in diameter, covered by epithelium.

Suggesting donovanosis (*granuloma inguinale*): a slow-growing lesion which may destroy the foreskin and parts of the shaft of the penis. The lesion is usually flatter and redder than carcinoma.

(d) Management
The ulcer is almost always infected, so treat with cloxacillin 500mg qds for 1wk pre-operatively.

If the growth is limited to the foreskin and is freely mobile over the glans, make a circumcision and follow up closely.

If it has involved the foreskin and the glans, or the shaft of the penis, take a biopsy, and as soon as the diagnosis is confirmed, perform a partial amputation 2cm proximal to the lesion. If there is a recurrence, it will probably be in the inguinal nodes, not in the stump of the penis.

If the inguinal nodes don't seem to be clinically involved, wait. 'Normal' nodes are palpable, and sepsis may cause some enlargement and tenderness. If they are palpable, and are clinically infected, wait. If they are palpable and clinically cancerous, biopsy the primary and get fine needle cytology on the nodes, if possible. If there is cancer in the nodes, amputate the penis, with a simultaneous bilateral block dissection of the groin (17.8) after allowing any severe sepsis to settle.

PARTIAL AMPUTATION OF THE PENIS (GRADE 3.1)
Aim to fashion the urethral orifice carefully, so that a stricture does not develop. *Remember the anatomical description of the penis assumes it is in the erect position* (27-31B).

Cut a long ventral flap based proximally. Make its width equal to ½ the circumference of the penis. Cut a shorter 2cm dorsal flap. Dissect both flaps back to their bases.

Dissect the *corpus spongiosum* away from the *corpora cavernosa*, until you reach the planned level of section. Divide the *corpus spongiosum* 2cm distal to the level where you intend to section the *corpora cavernosa*.

Pass transfixion sutures of #1 absorbable through each of the *corpora cavernosa* 0·5cm proximal to the intended level of section. Divide the *corpora*, dissect proximally for 0·5cm, and then tie the sutures medially and laterally. Cut a small circular slit in the ventral flap, and pull the urethral stump through it. Leave the end of the urethra protruding. It is less likely to stricture if you do this.

Leave adequate spaces between the sutures joining ventral and dorsal flaps to allow blood to drain and prevent a haematoma forming. Split the distal end of the urethra longitudinally.

Evert each half, and suture it carefully so that it everts onto the ventral flap. Leave a narrow self-retaining catheter in place for 5 days. Epithelium will grow over the raw surface of the *corpus spongiosum*.

Warn him that, despite your efforts, you may need to perform periodic dilations.

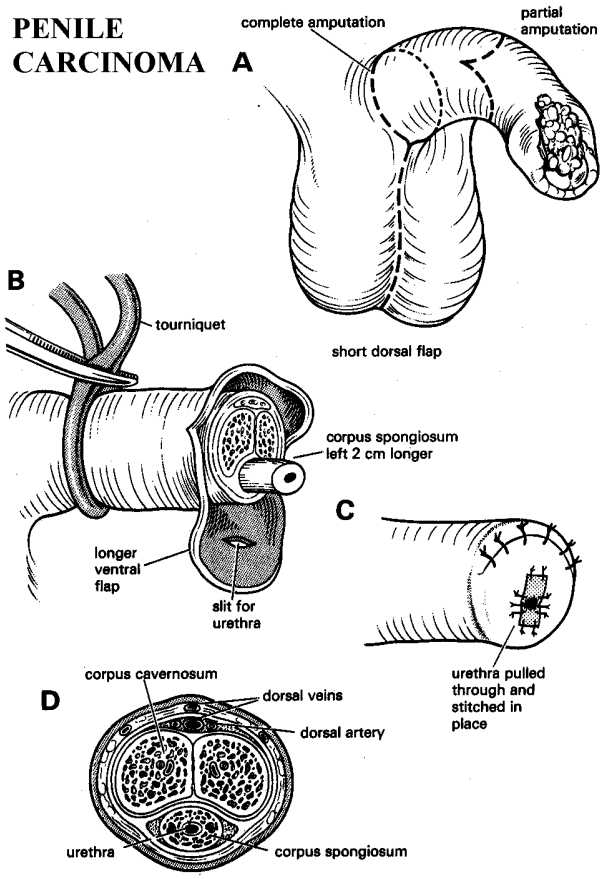


Fig. 27-31 PARTIAL AMPUTATION for penile carcinoma.
A, incisions for partial and complete amputation. B, flaps and the amputation. C, repair. D, cross-section of the penis.

MODIFIED COMPLETE AMPUTATION OF THE PENIS (GRADE 3.5)
INDICATIONS
Carcinoma of the penis which you cannot excise with a 2cm proximal margin by partial amputation.
N.B. This is a difficult operation!

METHOD
The ulcer is almost always infected, so treat with cloxacillin 500mg qds for 1wk pre-operatively.

Use the lithotomy position and pass a Ch20-24 metal dilator to define the urethra. Make a racquet-shaped incision round the base of the penis (27-32B). Extend the arm in the midline posteriorly for about 5cm, between the two sides of the scrotum (27-32A) (extend it further towards the perineum later).

Dissect deeper, clamping all vessels; this area is very vascular. Find, clamp, tie, and divide the large dorsal vein of the penis (27-31D).

Continue dissection until the shaft of the penis is free of subcutaneous tissue. Extend the incision posteriorly to where the scrotum hangs from the perineum, about 4-5cm in front of the anus. Separate the testes with their covering *tunicae vaginales*. Ask your assistant to retract them laterally with tissue forceps placed subcutaneously, first on one side and then on the other.

Dissect the *corpus spongiosum* on its ventral and lateral aspects, as far as the bulb which lies on the perineal membrane. Find it by feeling the expansion round the bougie. Remove the bougie, and cut the *corpus spongiosum* 4cm distal to the bulb. Separate it from the *corpora cavernosa* and retract it.

Now free the *corpora cavernosa* until they diverge as the *crura*, at the inferior border of the *symphysis pubis* (27-32C).

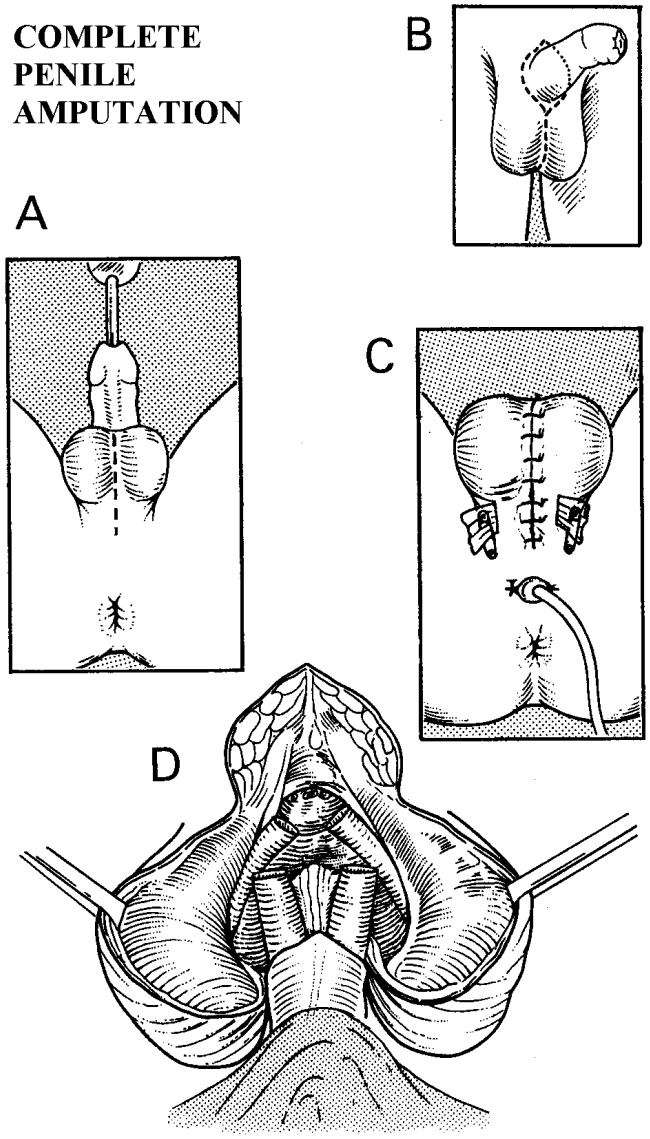


Fig. 27-32 COMPLETE AMPUTATION OF THE PENIS.
A, pass a sound. B, make a racquet-shaped incision round the base of the penis, and carry it vertically downwards in the midline of the scrotum. C, free the *crura* from the pubic bones. D, close the perineum round the stump of the urethra.

Transfix each of them with #1 absorbable and divide them 0·5cm distally. Only some connective tissue will now remain. Divide this and remove the penis.

Cut a transverse 1cm hole in the perineal skin, 2-3cm anterior to the anal verge. Deliver the stump of the *corpus spongiosum* through it, so that it protrudes about 2cm. Suture the base to the skin, using 2/0 or 3/0 monofilament sutures. *Don't try to evert the stump*, or the urethra may form a stricture. Leave the stump long because it tends to retract. Epithelium from the urethra and skin will grow and cover it.

Insert corrugated rubber drains (4-14B) through 2cm incisions laterally in the scrotum, and suture these to the skin. Or, better, use suction drains.

First close the wound in the midline using 2/0 monofilament. Then suture the anterior part of the wound. If the scrotum would hang down too much, trim off some skin and subcutaneous tissue before you suture it.

When you have finished, the scrotum will lie more anteriorly than usual. This allows good skin cover, and is less likely to get in the way when urinating through the perineal urethrostomy.

POST-OPERATIVE CARE. Pass an indwelling catheter, and remove it at 7-10 days. Apply much cotton wool padding, and a T-bandage pressure dressing. Remove the dressings and the drains >48h. Then start baths bd.

27.34 Penoscrotal elephantiasis

Lymphoedema of the penis and/or scrotum may occur as a result of:
(1) filariasis,
(2) onchocerciasis,
(3) donovanosis (granuloma inguinale)
(4) *schistosomiasis.*

When chronic, this produces the appearance of elephantiasis which may involve:
(1) the outer skin of the penis (but not its inner layer or its shaft),
(2) the scrotum,
(3) the testes which have hydrocoeles, but are otherwise normal; or, often, all of the above.

If the scrotum is grossly swollen (27-33A), try to excise it. The penis will either be buried in it, or separate, but covered with much thickened skin. This is a very satisfactory operation.

(a) Differential diagnosis: giant hydrocoeles (27.24), which may be present with elephantiasis, and hernias (18.4). In elephantiasis the texture of the skin of the scrotum is altered; it pits on pressure, it cannot be moved over the deeper tissues, veins are not visible, and the mass cannot be reduced.

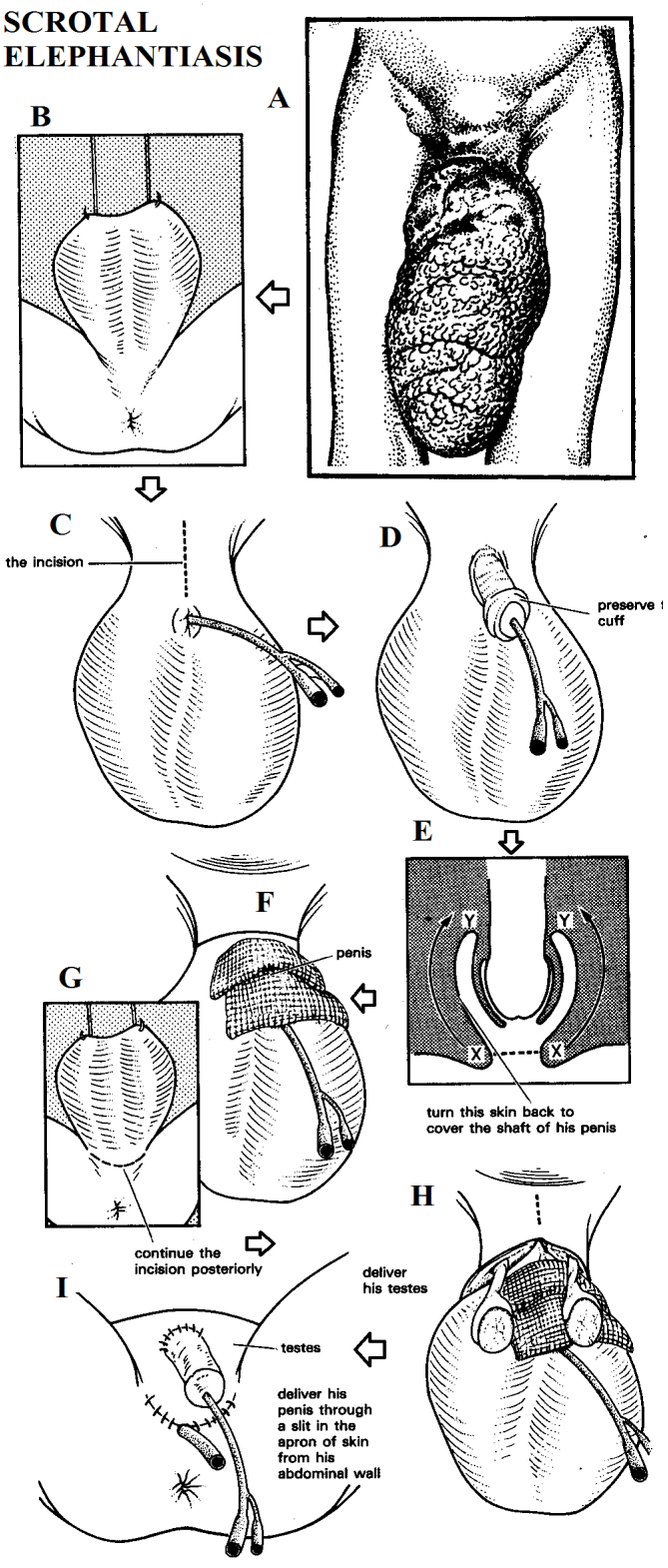


Fig. 27-33 ELEPHANTIASIS OF THE SCROTUM.
A, the appearance at presentation. B, suspend the scrotum (optional). C, catheterize the urethra and make the first incision. D, deliver the penis. E, preserve a cuff of foreskin to cover the penis. F, cover the penis with gauze while the operation continues. G, carry lateral incisions posteriorly. H, find the testes and deliver them. I, deliver the penis through the skin, and remove most or all of the scrotal skin. *Partly after Bowesman, C. Surgery and Pathology in the Tropics, Livingstone, 1960 with kind permission.*

(b) Treatment depends on the extent of the elephantiasis.

If there is elephantiasis of the foreskin (27-34), *don't make a standard circumcision*, or you will remove its inner normal layer. Instead, dissect off the thickened outer layer, and fold the inner one back over the shaft of the penis.

If the elephantiasis is mild and early, a limited operation may be all that is necessary. For example, you may only need to remove a dorsal strip of thickening on the penis, and close the resulting defect.

PREPARATION
Clean the skin thoroughly. If the scrotum is enormous (27-33A), either operate with the patient sitting and the legs over the edge of the table, or arrange a hook, and a block and pulleys, in the theatre ceiling before the operation starts (27-33B), so that you can raise the scrotum.

Catheterize the urethra (27-33C). If the catheter is difficult to insert, you may have to wait until you have exposed the penis. You will find that a catheter will be useful in locating the urethra, when you come to operate on the perineum.

Bleeding can be a problem. *Don't apply a tourniquet to the base of the scrotum to control bleeding*. Instead, use a long needle, such as a lumbar puncture needle, to inject the tissues with diluted adrenaline solution.

CAUTION! Never use adrenaline on the subcutaneous tissues of the penis; you can if necessary use it in the *corpora* (27.32).

REDUCTION SCROTOPLASTY (GRADE 3.1)
INCISION
If there are large hydrocoeles, tap them.

Make a midline incision downwards, from the pubic symphysis, to just above the foreskin (27-33C).

Carefully deepen the incision, until you reach the shaft of the penis (27-33D). Make a circular incision around the external preputial orifice, and preserve the internal layer of the foreskin, or the cuff of skin with which the penis communicates with the exterior (27-33E). Use it later to cover the penis. Clamp the cuff just beyond the glans, and divide the skin distal to it. Cover the raw isolated penis with saline swabs (27-33F), while you deal with the scrotum.

Make 2 lateral incisions round the root of the scrotum, to meet one another posteriorly in the perineum (27-33G). Carefully deepen these lateral incisions, until you reach the spermatic cords on each side. If necessary, find the cords at the external inguinal rings, and follow them to the testes, and deliver them (27-33H).

If the testes are of normal size and there are no hydrocoeles, *don't open the* tunicae vaginales.

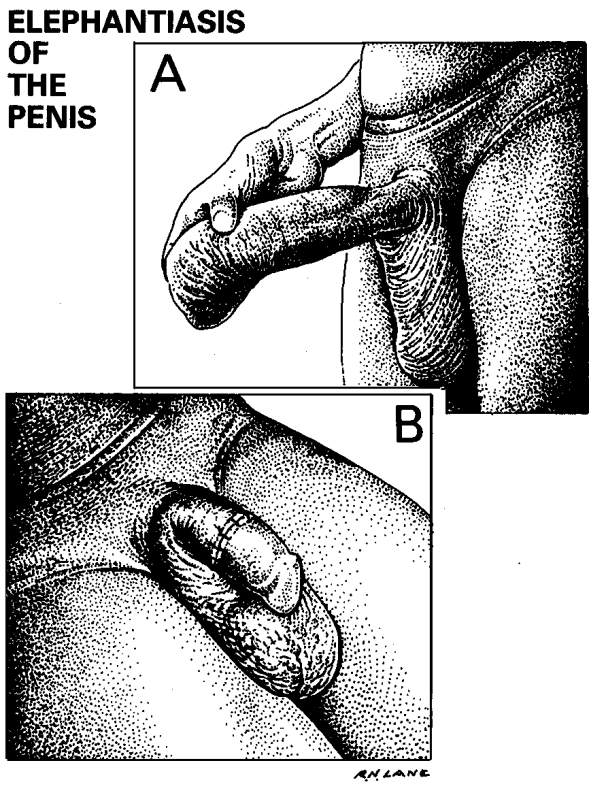


Fig. 27-34 ELEPHANTIASIS OF THE PENIS.
A, appearance at presentation; the scrotum is not involved. B, after a 'basal circumcision'. The skin of the inside of the foreskin has been used to cover the shaft.
After Bowesman C. Surgery and Pathology in the Tropics, Livingstone, 1960 with kind permission.

If there are large hydrocoeles, you may have to drain them first (if you have not already done so). Open them, and evert their sacs and suture them behind the testis (27-21).

If the sacs are thick, excise part of them.

Turn the scrotum up on to the abdominal wall. Identify, tie, and divide the many large veins that run from the scrotum. There is one large central one running up from the scrotum under the urethra.

Remove the bulk of the scrotum with a short amputation knife. Excise all thickened oedematous tissue. Either, make a new scrotum from the apron of normal skin that was dragged down by the mass.

Or, bury the testes in pockets, under the skin on the adductor aspects of the thighs. These pockets will be easier to make if you stand on the opposite side of the table. Push a long pair of scissors 15cm into the subcutaneous tissues of the thighs, *not deeper*, and create a pocket with a 5cm mouth. You may meet and need to tie the superficial external pudendal vessels and their 2 companion veins. Control bleeding before you insert the testes. Close the perineal part of the wound loosely, with a drain at its lowest point.

Remove the clamp from the cuff of skin that was the foreskin, trim away the part that was crushed, and roll the rest back to cover the shaft of the penis. Deliver this through a slit in the apron of skin dragged down from the abdominal wall (27-33I). Suture this to the skin of the shaft of the penis, starting with a single central suture, and proceeding laterally on both sides. Graft any remaining raw areas with grafts from the thigh, and dress them with petroleum jelly gauze.

Leave the catheter in place for a few days, to prevent the urine contaminating the wound. Any redundant tissue that you may have left will probably get smaller as time passes.

27.35 Kidney tumours

Renal adenocarcinomas present between 40-70yrs, as haematuria (60%), an enlarged kidney (20%), or with symptoms of secondary spread, such as general ill health and bone pain. Otherwise, pain is not a major feature, unless there is haematuria and clot colic.

If the tumour has not spread outside the renal capsule (<50%), the 5yr survival is 30% and the 10yr survival 7%. If it has, there are few 5yr survivors.

(a) Special tests
A good-quality intravenous urogram will demonstrate most renal masses. Look for displacement, deformity, and destruction of the calyces of the patient's kidney. The tumour is usually in the upper or lower poles.

(b) Ultrasound (38.2f) readily distinguishes solid from cystic lesions. If there is haematuria, perform a cystoscopy (27.3). Look for 'cannonball' metastases on chest radiographs.

(c) Differential diagnosis:
(1) Renal cysts (the commonest cause of a renal mass) and hydronephrosis. The kidney is palpable but haematuria is unusual.
(2) Polycystic kidney (a mass and haematuria).
(3) Hydatid renal cyst (15.12).
(4) An enlarged spleen.
(5) Other tumours of the kidney and large bowel.
(6) In children, a neuroblastoma displacing the kidney downwards.

N.B. Not all renal cysts and hydronephrotic kidneys are palpable, and the absence of haematuria does not exclude a carcinoma.

(d) Management
If there are no obvious metastases, arrange a nephrectomy. It may reverse some of the systemic effects of the tumour (anaemia, myopathies, etc.), even if there are metastases.

If there is a transitional cell tumour of the renal pelvis, it is likely to be associated with similar tumours in the bladder. These tumours usually project into the renal pelvis, so that you can see them on a retrograde urogram. Nephro-ureterectomy is indicated.

If it is a squamous carcinoma (HYPERNEPHROMA), it is likely to be associated with chronic infection, and a curative nephrectomy is seldom possible.

If a child has a nephroblastoma (WILMS TUMOUR), look for other congenital anomalies (eye defects, hemihypertrophy, and urogenital anomalies) which are sometimes associated. After Burkitt's lymphoma, this is the commonest solid tumour of childhood in Africa. The tumour arises from embryonal kidney cells and spreads locally through the capsule of the kidney to neighbouring nodes (often fairly late), as well as to the liver, lungs, and sometimes the bones.

The young child presents with a fairly rapidly growing and usually painless mass on one side of the abdomen. In 70% of cases this is <2yrs, and is seldom >6yrs. In 5% of cases the mass is present at birth, and in 5% it is bilateral. There is rapid weight loss and fever in 50% of cases. Haematuria is late.

(e) Staging and prognosis. Nephroblastoma is always fatal without treatment. The survival figures below are for a combination of nephrectomy, radiotherapy, and chemotherapy. Long-term cures for Stages I-III are common.

Stage	Definition	5yr Survival
I	Tumour is confined to the kidney, and can be removed *in toto*.	95%
II	Tumour penetrates the capsule, but can be entirely excised.	85%
III	Tumour not completely resectable, but confined to the abdomen.	85%
IV	Tumour widespread, with metastases beyond the abdomen.	5%

N.B. In bilateral disease (5%), palliation only is possible. *Don't try to remove the kidney*, unless you are skilled. Diagnose the condition clinically, and organize investigation, nephrectomy, and chemotherapy, and perhaps radiotherapy. If this is not available, it is acceptable to rely on nephrectomy and chemotherapy.
It is not acceptable to leave the kidney and to treat with chemotherapy only.

If you are sure of the diagnosis from the IVU or ultrasound, start chemotherapy to reduce the volume of the tumour: this makes surgery easier, and you then know if the tumour is responding to medication.

Treat with vincristine 1·4mg/m² (maximum dose 2mg) IV and repeat after 2 days. Arrange surgery 10-12 days later if the kidney is of resectable size. Otherwise, continue downstaging chemotherapy with nutritional support, prophylactic anti-TB therapy, antibiotics and antihelminthics also to prepare for surgery. You will need to cross-match blood and prepare the bowel.

CAUTION! Sometimes the tumour spreads into the inferior vena cava.
After nephrectomy, continue the chemotherapy regime monthly for 4-6 months after a gap of 2-3 days.

27.36 *Schistosomiasis* (*bilharziasis*) in the urinary tract

Schistosomes are trematode blood flukes infecting $>2.10^8$ people; there are several types but *S. haematobium* in the Middle East and Africa causes urogenital disease, though other types cause mainly liver and intestinal disease. The adult male fluke encloses the cylindrical female to mate, assuming an elongated shape able to live inside veins by attaching to their walls by suckers. The fertilized female then detaches itself and migrates to narrow veins, preferentially in the vesical plexus, laying >200 eggs daily (27-35). The *S. haematobium* egg has a terminal spine, unlike the *S. mansoni* egg which has a lateral spine.

When these eggs are excreted in the urine, they can hatch in fresh water and attach themselves to certain snails (*Bulinus truncatus*) which live in slowly flowing shallow water. These snails can then produce 10^5 free-swimming forked-tailed *cercariae* which have an enzyme that allows them to penetrate human skin, and so continue the life-cycle. People, especially women, using infected water are susceptible; it is rare <5yrs. The incidence may vary enormously from village to village. A protective soap may prevent invasion of the *cercariae*. It is the eggs that cause the trouble, initially producing an immune dermatitis ('swimmer's itch') and a Type II serum sickness called Katayama fever; later when they migrate into the tissues eliciting a Type IV immunological response, a granuloma results, the severity of which depends on the number of eggs deposited and the host's immune response.

Although the eggs of *S. haematobium* predominantly affect the urinary tract, they can also travel into the lungs, spinal cord and brain. The granuloma is accompanied subsequently by fibrosis and impaired collagen synthesis. The eggs can survive ≥10yrs and so chronic disease is common. *Schistosomiasis* affects >200 million people worldwide, and causes up to 200,000 deaths annually.

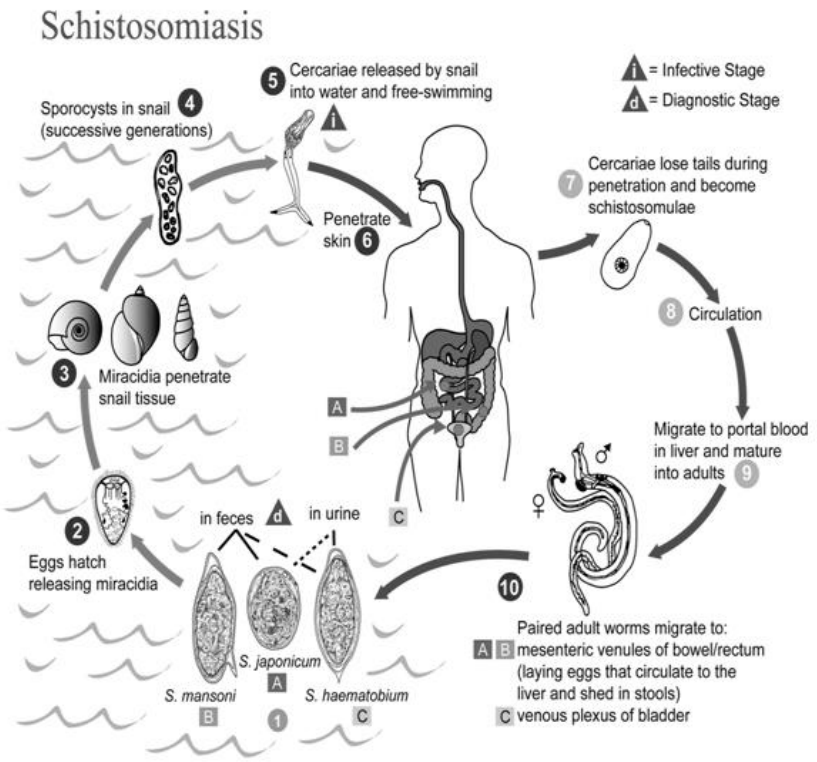


Fig. 27-35 THE LIFE-CYCLE OF THE *SCHISTOSOMIASIS* FLUKE.
After http://www.cdc.gov/parasites/schistosomiasis/biology.html and wikimedia.org.

The results are:
(1) dilated cystic ureters,
(2) thickened pipe-stem ureters,
(3) ureteric strictures all with possible stones (27.15),
(4) active cystitis,
(5) chronic cystitis (as with radiotherapy),
(6) bladder wall thickening and ulceration,
(7) bladder neck fibrosis (27.21),
(8) a contracted low-volume bladder,
(9) bladder carcinoma (27.5),
(10) active or chronic prostatitis (27.19),
(11) epididymitis and chronic spermatic cord inflammation (27.23),
(12) rarely, urethral stricture, dilation and fistula.

Such pathology may obviously lead to renal failure as well as infertility, or death from malignant infiltration. You should feel for an enlarged kidney, a pelvic mass, a hardened prostate or lumpy spermatic cord and epididymis. Active cystitis usually presents between 5-15yrs with painful micturition, frequency and terminal haematuria. This type of haematuria (27.4) is really the hallmark of *schistosomiasis*.

(a) Special tests
Examine urine or semen carefully for *schistosoma* eggs, as well as red cells and pus cells (especially in bladder carcinoma where you may find strands of tumour tissue in the urine). You may need a cystoscopic or epididymal biopsy, however, if all the eggs have migrated into the tissues.

The bilharzia fixation antibody test (BFAT) will show if there has been infestation in the past, but *does not necessarily imply the disease is active at present.*

In an endemic area, macroscopic haematuria in a patient under 30yrs correlates with *schistosomiasis* in 90% of cases.

Check the Hb and urea levels.

(b) Ultrasound may show hydronephrotic kidneys, dilated or tortuous ureters, a thickened bladder wall >5mm, possibly with ulceration or irregular tumour growth.

(c) Radiographs. Plain radiographs may show:
(1) an enlarged kidney,
(2) linear calcification in a dilated ureter,
(3) patchy ureteric calcification,
(4) secondary ureteric stones,
(5) dense calcification in the cystic wall, sometimes with defects in the calcification where a tumour has developed.

IVU will demonstrate the size, tortuosity and stricture of ureters most clearly. They may have multiple bubble-like filling defects. The ureters can look like small bowel! Also, you will be able to see the size of the bladder clearly.

(d) Cystoscopy. Appearances are striking: you may see in active cystitis:
(1) reddened congested oedematous patches especially on the posterior wall, as well as raised yellowish tubercles; and in chronic cystitis:
(2) 'sandy' patches,
(3) nodules, polyps, ulcers, scarring and tumours,
(4) cystic degeneration in the bladder wall,
(5) narrowed or patulous ureteric orifices.

(e) Treatment
Praziquantel 60mg/kg stat or 10mg/kg bd for 3 days is most effective where fibrotic effects are not yet advanced. You may need to treat repeatedly for re-infection.

Where the ureter is irreversibly damaged, reconstructive surgery is necessary: this is complicated because excision and spatulated end-to-end anastomosis is rarely feasible. A re-implantation of the ureter into the bladder is usually necessary, and sometimes, if the ureteric stricture is very proximal, with a bladder extension (Boari) flap. Otherwise the whole ureter needs replacing with an ileal segment.

You can treat bladder neck fibrosis with excision (27-19K) but a contracted bladder needs an augmentation cystoplasty and a urethral fistula may need a urethroplasty.

27.37 Other urological problems

If there is a foreign body in the urethra, try to remove it with as little damage as possible. Use GA.

Ideally, identify the foreign body with a cystoscope using the 0° objective to look down the urethra. Failing this, locate it in the penis by palpation and with radiographs.

Try to disimpact its distal end from the wall of the urethra. Use alligator forceps and, perhaps, a large bore cannula. If necessary, perform a urethrotomy (27.12), and cut down on the urethra through the ventral surface of the penis.

If the foreign body is far back in the urethra, try to dislodge it into the bladder, and if you cannot remove it endoscopically, do so through a small suprapubic cystotomy.

If it is a pin, you may have to remove it head first unless you can secure the sharp tip in a biopsy forceps, so put its point through the wall of the urethra, and turn it round.

If the opposing surfaces of the glans and foreskin are acutely inflamed, this is acute BALANITIS. Test the urine for sugar to exclude diabetes. The primary treatment of balanitis is better hygiene. Demonstrate how to retract the foreskin, and do this at least tds, to wash with soap and water and to apply a mild antiseptic, such as chlorhexidine or cetrimide and hexachlorophene. Avoid systemic antibiotics. If there is associated phimosis, arrange for circumcision.

If pain and swelling develop with explosive rapidity in the penis or scrotum, and there is severe illness, suspect that there is severe penoscrotal necrosis and infection (6.21,23: Fournier's gangrene), which needs immediate debridement.

If a painful perineal swelling develops, this is probably a periurethral abscess (6.18).

If milky urine is passed, this is CHYLURIA due to a fistula between the lymphatics and the urinary tract. Sometimes chyle comes out with blood (chylohaematuria).

Where *Wuchereria bancrofti* is endemic, chyluria is not uncommon and this is the most common cause; elsewhere it is rare, and due to tuberculosis, *schistosomiasis*, ascaris infestation, hydatid disease, malignancy, ureteric stone or trauma. The urine is characteristic; if left to stand, it separates into 3 layers: a top layer of fat, a pinkish layer of clots, and a bottom layer of debris in the urine. You can see chylomicrons in the urine under the microscope, and microfilariae in 25% of cases.

Chyluria debilitates, through a persistent loss of fat, protein, electrolytes, and vitamins in the urine. Treat with a single dose of diethylcarbamazine 6mg/kg with albendazole 400mg, or ivermectin 150μg/kg.

If this fails, you can lavage 5-10ml of 1:10000 silver nitrate solution into the renal pelvis after passing a ureteric catheter under direct vision by a cystoscope with the patient in a Trendelenburg position. This can be done weekly up to 4 times. It is effective in 70% of cases.

If there is erectile dysfunction, examine the penis for any structural abnormality. Ask if there is ever a sustained erection: although 'wrong time, wrong place, wrong sexual partner' is often the cause, it is not necessarily so.

The underlying problem may well be:
(1) HIV disease,
(2) obesity,
(3) vascular disorders (not only the classical Leriche syndrome caused by aortic bifurcation occlusion),
(4) medication (especially cimetidine, bendrofluazide, β-blockers and antidepressants).

Sildenafil 25-100mg 1h prior to sexual activity is most effective; intra-penile injections and prosthesis are very much second best, and can be subject to significant complications, particularly priapism (27.32), and infection.

If there is an atonic or hypotonic bladder, either from motoneurone disease, spinal injury (54.2,7), stroke or other neuropathy, but with continence, the bladder needs help to be emptied. The least risk of introducing infection is by training the patient in intermittent self-catheterization. *You must educate him on sterile procedures!* An alternative in certain circumstances may be a *rectus abdominis* detrusor myoplasty (54-21).

28 The eye

28.1 Introduction

There are c.285 million visually impaired people in the world, of whom >39 million are blind. WHO estimates that 43% of the visually impaired are so because of a lack of spectacles while 30% have cataract. Major causes of blindness in the world are cataract (50%), corneal infections (particularly trachoma, 25%), glaucoma, vitamin A deficiency, and *onchocerciasis*.

Washing the face regularly and the use of azithromycin every 3 months in children under 12yrs reduces the incidence of trachoma. In the industrial world 0·2% are blind, but in low-income countries blindness is 10 times more common. You can treat cataracts, arrest glaucoma, and prevent trachoma and vitamin A deficiency. It is unfortunate therefore that ophthalmology scares most doctors, who imagine that treating the eye must be impossibly difficult. *This is not true*: you can diagnose 90% of eye diseases with a torch and an ophthalmoscope.

Nonetheless, the eye may be difficult to examine, particularly in a child, and if the eyelids are swollen or the eye painful, the patient may forcefully resist examination. *Don't give up*, because the signs of serious trouble may be subtle. Danger signs are: haziness of the cornea, inequality of pupil size (especially if associated with reduced visual acuity), and circumcorneal redness.

Ideally someone in your district should be able to perform cataract removals. Surgery inside the eye, however, is difficult, so try to learn these operations by apprenticeship from an expert; they are not described here.

(a) Anatomy

The eye lies within the orbit, a V-shaped enclosure, designed to protect the eye from trauma. Its blood supply comes from the ophthalmic artery, a branch of the carotid artery. Six muscles are attached to the eye and wall of the orbit; the IIIrd, IVth and VIth cranial nerves pass through a fissure in the superior part of the orbit to supply the muscles and the Vth cranial nerve gives sensation to the eye.

A septum is attached to the rim of the orbit and the eyelids are attached to this. The eyelids protect the corneal surface and are made from skin, muscle, tarsal plate (28-18) and conjunctiva.

The eye itself can be divided into:
(a) the external eye: lids, conjunctiva, sclera,
(b) the anterior segment: cornea, iris and lens,
(c) the posterior segment: vitreous and retina, optic nerve.

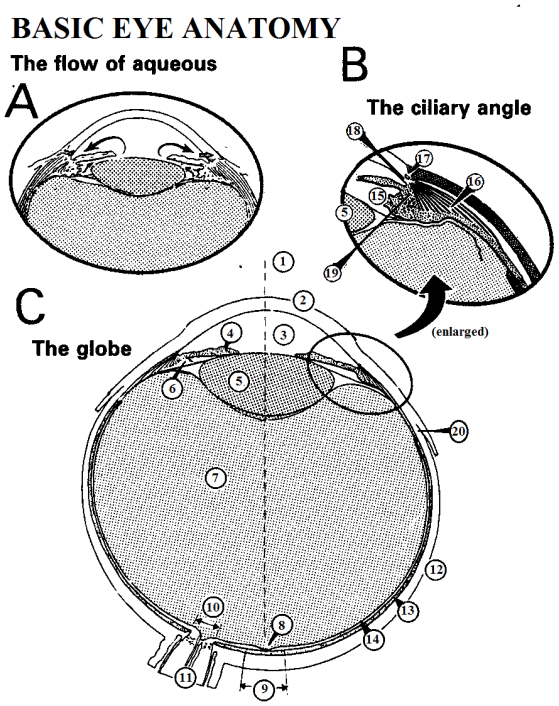


Fig. 28-1 BASIC EYE ANATOMY.
A,B, flow of aqueous from the ciliary body (15) into the posterior chamber (6), through the pupil into the anterior chamber (3), then through the trabecular meshwork (19) via Schlemm's canal (18) into the scleral sinus (17). C, The visual axis passes through the middle of the pupil (made by the iris (4)) and through the centre of the lens (6), and the vitreous (7) to the fovea (8) which is at the centre of the macula (9). The optic nerve (11) enters the globe at the 'blind spot' and makes the optic disc (10). It is contiguous with the light-sensitive retina (14), bounded by the choroid (13), and sclera (12), which joins the cornea (2) anteriorly at the limbus (20), where the conjunctiva makes a groove known as the fornix. The globe rotates within a fascial layer, Tenon's capsule, which covers the sclera and forms the sheaths of the extraocular muscles, the outer layer joining the conjunctiva at the limbus.

(b) History

Always take a careful history; it may be critically important. Focus on how vision has changed and whether there is discomfort in the eye.

Vision can be divided into distance, near, peripheral, stereo double or single, colour, or night vision. Ask which type has been most affected.

Has vision been lost rapidly (specifically ask about trauma, resulting in retinal detachment, haemorrhage, or optic nerve damage), or slowly (cataract 28.4, presbyopia 28.8, diabetic retinopathy 28.9)?

Is central vision lost (macular disease from diabetes, macular degeneration, cataract) or peripheral vision (glaucoma, retinal detachment, inherited eye disease)?

Remember 'double vision' may actually be blurred vision. Ask about 'floaters' and 'flashes' in the vision.

Ask about ocular discomfort: conjunctival pain tends to feel like sand or hair in the eye, while very high pressure or inflammation of the eye can feel more like a deep ache or throbbing pain. Note any watering of the eye.

Optic nerve disease can present with pain on eye movements and loss of vision. Light sensitivity can be due to inflammation of the eye, or sometimes cataract and post-trauma or surgery problems.

Ask about a family history of eye disease such as glaucoma, cataract, or night blindness.

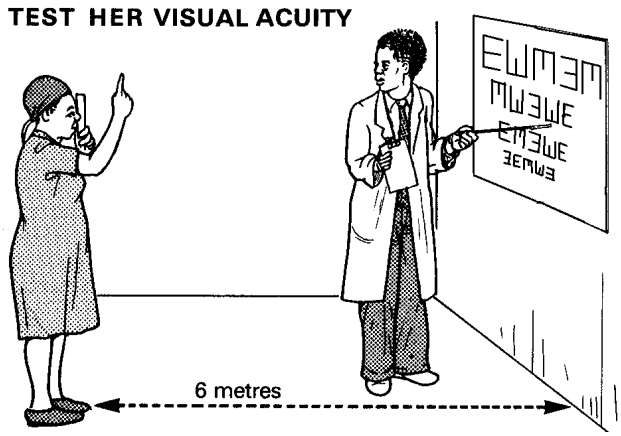


Fig. 28-2 TEST THE VISUAL ACUITY before you do anything else. Stand the patient 6m from the test chart and ask her to tell you if the 'three legs go up, down, right, or left'.
After: www.talcuk.org.

(c) Examining an eye. The standard examination of an eye is time-consuming to do well, so train a nurse or medical assistant to test the visual acuity and examine the eyes. Your consulting room must be at least 6m long and you should be able to darken it. You *must* have a good pen light. Most examinations can be done while a patient sits in front of you.

ALWAYS test the visual acuity. Explain that you want to test the eyes. Begin by testing them separately (with distance glasses if worn); test them again on each subsequent visit; and record your results, so that you will know if vision is deteriorating or not.

If the patient can read, test each eye separately either with Snellen's or LogMAR charts. Stand him 6m from the well-lit chart (28-2), and close the left eye with a piece of paper or your left hand. Ask him to start at the top until he cannot read anymore. If he is a young child or cannot recognize letters, use the tumbling E chart. It may be helpful to get him to point with fingers in the direction of the letters on the chart. Values are written with the top figure as the distance in metres to the test chart, the bottom one the distance at which a person with normal vision can read that line. The standard chart is calibrated: 6/6, 6/9, 6/12, 6/18, 6/60, and 3/60: these represent steadily deteriorating vision measurements. A value of 6/12 is normally required for driving a car. Counting fingers (CF) at 3m is equivalent to 3/60. If CF<1m, try hand movements (HM), and then test for the perception of light (PL). Get children to point at pictures of objects.
Visual acuity can be usefully divided into four groups:
(1) good vision 6/6-6/18,
(2) poor vision 6/24-6/60,
(3) partially blind CF5m to PL,
(4) totally blind to NPL (no perception of light).

The LogMAR charts have 5 standard shape letters in each row, varying logarithmically in size and spacing. The score is based on the number of smallest letters read.

If you shine a torch into each of the 4 quadrants of the visual field, can he tell you where it is coming from?
Blindness is 'a loss of vision which results in the patient being unable to continue with normal life, and to walk unassisted'. It is usually equivalent to binocular vision of <3/60, which is the same as CF<3m. Before you decide that there is complete blindness, test with a very strong light. If an eye cannot see any light, and its pupil does not react to light, it is sure to be beyond help, so there is no point advising otherwise. If the vision is normal and remains normal and the eye is not inflamed, pathology is unlikely.

Here is some basic eye equipment: it does not include equipment for operating inside the eye. Many of the instruments are very fine: look after them with the greatest care:

CHARTS, visual acuity, (a) Snellen and (b) illiterate E charts, both for use at 6m. These are essential, and can usually be produced locally. They have patterns of 'E's of different sizes in different positions, and can be used by patients who cannot read.
TEST TYPE, reading pattern. Use this for examining older patients with presbyopia (28.8) who need glasses. If necessary, you can also use a book or newspaper.
TORCH, for focal illumination, local pattern, preferably pen type, with 'lens bulb'. A locally available torch is adequate: it can be easily replaced, as can its bulb and batteries.
LOUPE (x2-8 magnifying spectacles), binocular, surgical, headband type. Some simple form of magnification is useful for examining the front of the eye, for removing superficial foreign bodies, and for other kinds of fine work, such as suturing nerves.
TONOMETER, Schiötz (28-3). You must be able to measure the intraocular pressure (IOP) if you are going to diagnose glaucoma. Digital measurement is simple but unreliable. This instrument is not much seen nowadays, but is still very useful.
OPHTHALMOSCOPE, simple pattern, Keeler type, battery handle. An ophthalmoscope is very useful, but you can do much good eye work without one.
SLIT LAMP MICROSCOPE, on stand, simple pattern. You will find a slit lamp useful, although you can diagnose uveitis without one. If you need to do a lot of eye work, this is very useful: spend some time, though, with an experienced operator before you purchase one of these delicate instruments.
SPECULUM, ophthalmic lid, solid blades, hinged with screw adjustment.
SCISSORS, ophthalmic lid, blunt points. If necessary, you can use any fine scissors.
FORCEPS (clamp), tarsal cyst (chalazion, 28.12*), 8mm ring, Lambert pattern*. This has two blades, one with a ring and the other with a plate. Use it to hold an eyelid while you incise a tarsal cyst.
CURETTE, chalazion (tarsal cyst).
CAUTERY, simple type, ball pattern. Heat this on a spirit lamp, or get a battery-operated type.
CLAMP, eyelid, entropion, Desmarre's or Snellen's, (a) medium and (b) large. Use this to hold the eyelid when you operate for entropion.
SCISSORS, ophthalmic, spring pattern, Westcott's or Castroviejo's. These are very delicate instruments which need treating with special care.
BLADE, crescent type.
FORCEPS, fine, toothed, St Martin's.
RETRACTOR, eye, Desmarre's. Use this for examining children.
NEEDLE HOLDER, ophthalmic, curved with lock, Castroviejo pattern.
LENS LOOP for cataract extraction.
INTRAOCULAR LENS: standard PMMMA 21 dioptre are sufficient.
CHEAP SPECTACLES can be made from malleable wire and polycarbonate instead of glass for the lens (www.onedollarglasses.org). Otherwise there are self-adjustable fluid-filled glasses available (28.8).
GLASSES, simple frames, second-hand if necessary, spherical lenses +1 to +3.50 – the most commonly needed glasses are +2 and +2·50. Collect unwanted glasses and allow patients to try for themselves: this way you can deal simply and effectively with the reading difficulties of many.
Don't operate with the large instruments of a basic set. Use special fine instruments listed above. For operations on the globe, an operating loupe and a bright focal beam are almost essential, using preferably a 12V spotlight or LED source.

THE PIN-HOLE TEST is a useful way of screening for refractive errors. If there is poor vision, place a card with several 1mm holes (punched 5mm apart) in front of the eye. For repeated use, attach the card to the insides of each lens of a pair of spectacles. Cover each eye in turn for testing. If there is an uncorrected refractive error, the vision will be significantly improved to 6/9 or 6/12.

(d) Check the visual fields, in all 4 quadrants, while sitting face-to-face with the patient, covering the right eye and your left eye and *vice versa*, comparing them.

(e) Examining the outer eye

Start by looking at the whole face. Note any asymmetry of the position of the eyes, or any protrusion (best judged from above and behind the patient). Palpate for a lump, and for high intraocular pressure.

Ask the patient to look down and keep looking down, but not to actively close the eyes. Put the tips of both your index fingers on one of the globes, so as to feel the sclera through the upper lid above the upper border of the tarsal plate.

Gently press with alternate fingertips towards the centre of the globe:

(1) Gently fluctuate it from one finger to another.

(2) Indent it with one finger and estimate the sense of fluctuation imparted to your stationary finger.

(3) Estimate the indentation of the sclera as you relax your indenting finger. You can judge the eye to be 'soft' (<10mm Hg), 'normal' (10-40mm Hg), or 'hard' (>40mm Hg). This is a crude test, and there must be a significant rise of pressure (>40mm Hg) before you can detect a raised intraocular pressure.

Test the movements of both the eyes together, and then test each eye separately, in all directions, including convergence. Note any squint (28.9).

If there is much pain, and the eyelids are in spasm, a drop of sterile LA will make examination tolerable. This will also allow you to insert a speculum to examine the eye more easily. Whilst the patient is looking, grasp the top lid with your finger, and slip the top blade of the speculum under it. Then ask the patient to look up, grasp the bottom lid, and slip the lower blade of the speculum under that. Adjust the arm of the speculum until the eye is exposed, and then tighten the locking nut.

CAUTION! Beware that the speculum does not press on the eye or damage the cornea.

Note abnormalities of the lids, lacrimal apparatus, puncta and canaliculi, the lacrimal glands and sacs, and also any epiphora (tears running down the cheeks).

Do the eyelids open and close normally? You can see this best on blinking. Check the lids for swellings, and check that the lashes are in their normal position.

Look at the conjunctiva. Note particularly the distribution of any redness. If it is maximal near the corneoscleral junction, this occurs in iritis and corneal ulcer. If it is maximal at the periphery but often extending all over, it is likely to be conjunctivitis.

To examine the conjunctiva of the upper lid, evert it (28-8H-K). This is necessary to exclude a foreign body.

Look for pus or mucopus in the inferior fornix. This is present in all cases of bacterial conjunctivitis, and in some cases of viral conjunctivitis. Look also for signs of vitamin A deficiency: dry-looking conjunctivae, or Bitot's spots (white patches on the temporal side of the conjunctiva, produced by keratin mixed with gas-forming bacteria).

Look at the cornea of each eye. Is it shining and clear, reflecting the light of a torch, or its surface irregular (corneal ulcer)? Is there clouding superiorly (trachoma, 28.13), or a general haziness (oedema from trauma, keratitis, or glaucoma)? A bright light and a loupe can detect keratic precipitates and adhesions (synechiae, 28-9B, of iritis).

If you suspect the surface is injured or ulcerated, instil 1 drop of 2% fluorescein, or dip the end of a fluorescein-impregnated filter paper inside the lower lid for a few seconds. Mop out the excess fluorescein with tissue paper. Shine a light on the eye at an angle. Gaps in the corneal epithelium (ulcers, abrasions) stain bright yellow-green.

Look at the anterior chamber and note its depth. Is there any blood (hyphaema) or pus (hypopyon, 28-9C) at the bottom of the anterior chamber?

Look at the pupils. Do they look black and do they react to bright light? Are the pupils grey or white (opacities in the lens, cataract)? Note their size, shape, and if their outline is irregular (synechiae, due to iritis, 28.5). If a pupil constricts incompletely when light is shone into that eye, and then constricts further when it is shone into the good eye, and when the light is shone back into the abnormal eye, both pupils enlarge, this means there is optic nerve damage, commonly caused by glaucoma, but you should exclude a stroke or brain injury. This is known as a relative afferent pupillary defect.

(f) Fundoscopy (ophthalmoscopy) examines the fundus and media of the eye. You must either dilate the pupils with a mydriatic such as cyclopentolate 1%, or do your examination in a dark room. *This is however ineffective where the vitreous or cornea is opaque or very unevenly curved (extreme astigmatism).*

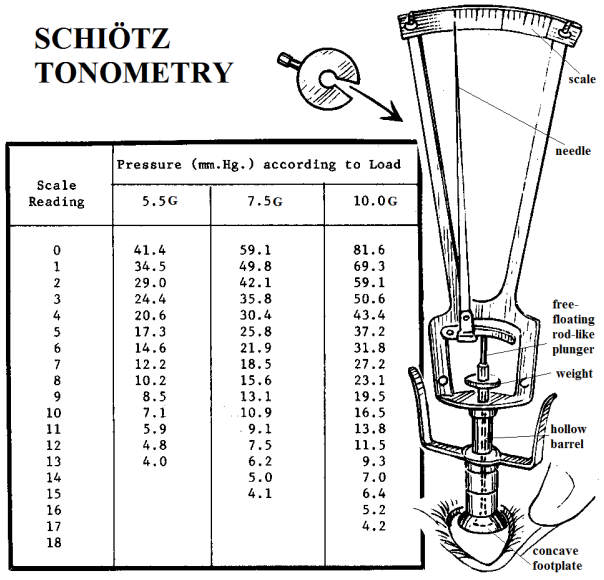
(1) Get the patient to keep both eyes open and look straight ahead.

(2) Start with the '0' lens in the ophthalmoscope (unless you have a refractive error and are not wearing glasses; if so, select the appropriate correcting lens and remember this as your starting point).

(3) Use your right hand and your right eye for the patient's right eye and your left hand and your left eye for the patient's left eye.

(4) Hold the sight hole of the ophthalmoscope close to your eye, resting it against your nose and orbit, and move it with you as if it was attached to your head. To find this position, look through the sight hole at some distant object.

(5) With your thumb on the patient's forehead, gently raise the upper lid clear of the pupil.
(6) Start with the ophthalmoscope 20cm from the eye, and shine the light into the pupil; it should glow uniformly red (the red reflex). This indicates the absence of a cataract.
(7) Move closer and watch for any opacities in the media silhouetted against the red reflex. Corneal opacities appear to move in the opposite direction to the ophthalmoscope; vitreous and posterior lens opacities appear to move with the ophthalmoscope. If you see a shadow, use the + lenses (+5 to +12) to see it more clearly.
(8) Ask the patient to look straight ahead, and move as close as you can to the eye without touching the eyelashes or cornea.
(9) Find and look at the optic disc: it is 15º to the nasal side of the optical axis of the eye.
(10) Turn the lens wheel in the ophthalmoscope with your forefinger from +6 down to zero to get the best view of the disc. Examine:
(a) the vertical cup/disc ratio (a ratio of >0·7 suggests glaucoma, 28.6, 28-11),
(b) the disc margins; if these are blurred all round (360º), it suggests papilloedema,
(c) the blood vessels; look for nasal displacement of the central retinal vessels and for haemorrhages and exudates suggestive of diabetic retinopathy,
(d) the macula (28-1C), by asking the patient to look directly into the light source, for black and white pigmentation which may suggest choroiditis (28.5) involving the macula (maculopathy).



Scale Reading	Pressure (mm.Hg.) according to Load		
	5.5G	7.5G	10.0G
0	41.4	59.1	81.6
1	34.5	49.8	69.3
2	29.0	42.1	59.1
3	24.4	35.8	50.6
4	20.6	30.4	43.4
5	17.3	25.8	37.2
6	14.6	21.9	31.8
7	12.2	18.5	27.2
8	10.2	15.6	23.1
9	8.5	13.1	19.5
10	7.1	10.9	16.5
11	5.9	9.1	13.8
12	4.8	7.5	11.5
13	4.0	6.2	9.3
14		5.0	7.0
15		4.1	6.4
16			5.2
17			4.2
18			

Fig. 28-3 SCHIÖTZ TONOMETRY.
The scale is merely an example; use the scale which is supplied with your instrument. 3 weights (5·5, 7·5, 10g) are usually supplied with each instrument.

SCHIÖTZ TONOMETRY
You may well never see this old-fashioned, but simple, instrument. If you happen to find one, clean the instrument with a pipe cleaner and ether. You'll find it quite useful to diagnose glaucoma. It is, however, a delicate instrument, so keep it carefully!
Using the standard 5·5g weight and the metal footpad, make sure the instrument is calibrated to zero.
Explain what you are going to do, lay the patient flat and instil LA into the conjunctiva. Ask him to open both eyes, and look straight up at a target placed on the ceiling.
With the 5·5g weight in place, put the tonometer plunger gently on the centre of the cornea with the eye open, and read the scale. If in doubt, repeat the reading 3 times. Use the tables provided with the instrument to calculate the IOP from the scale reading.
The normal IOP is 7-25mm Hg. In practice, using the 5·5g weight, a scale reading of ≤2 (>28mm Hg) indicates a raised IOP. A reading of ≥3 (<25mm Hg) is 'normal'. If the IOP is >40mm Hg, the cornea is likely to become oedematous (the characteristic 'hazy cornea' of glaucoma), and you can see this with a torch. This is usually a late sign of glaucoma.

BINOCULAR INDIRECT OPHTHALMOSCOPE

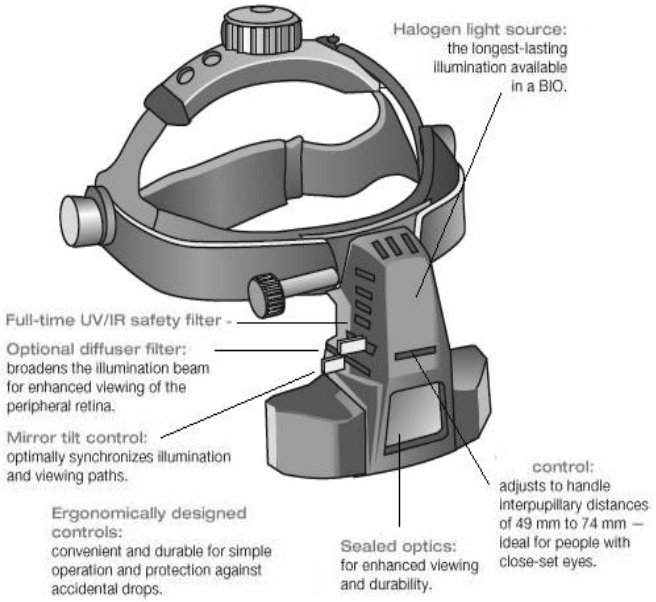

Halogen light source: the longest-lasting illumination available in a BIO.
Full-time UV/IR safety filter
Optional diffuser filter: broadens the illumination beam for enhanced viewing of the peripheral retina.
Mirror tilt control: optimally synchronizes illumination and viewing paths.
control: adjusts to handle interpupillary distances of 49 mm to 74 mm — ideal for people with close-set eyes.
Ergonomically designed controls: convenient and durable for simple operation and protection against accidental drops.
Sealed optics: for enhanced viewing and durability.

Fig. 28-4 BINOCULAR INDIRECT OPHTHALMOSCOPE.
This allows good examination of the anterior and posterior segments at much less cost than a slit lamp. It is also portable (28-5).
After Chaudhury A, Chugh A, Role of Indirect Ophthalmoscopy in Rural Settings, Rural Surgery 2009;5(3):19-20.
(www.mercoframes.net/product/binocular-indirect-ophthalmoscope)

(g) Indirect ophthalmoscopy

A binocular indirect ophthalmoscope (28-4) provides stereoscopic, wide-angle, high-resolution views of the entire ocular fundus. It is not hindered, as is the standard ophthalmoscope, by a hazy media or scleral or central opacification. With the addition of a +20 dioptre condensing lens, by varying the illumination and viewing angle, you can readily look at both the anterior and posterior segments.

N.B. Examine layer by layer: lid margin → conjunctiva → cornea → anterior chamber → lens → vitreous.

Lid margin: Plugged orifices, lice, erosions?
Conjunctiva: Foreign body?
Cornea: Foreign body embedded in the cornea? Ulcer? Note its size and shape after instilling fluorescein and using the blue light. On the back of the cornea look for keratic precipitates (KP, these are clumps of white cells), indicating uveitis.
Anterior chamber: Look for cells and flare, pus and blood; estimate its depth.
Lens: Diffuse opacity, discolouration? Posterior synechiae from the iris? Focal opacities?
Vitreous: Particles from a recent posterior uveitis, or bleeding?

BINOCULAR INDIRECT OPHTHALMOSCOPY

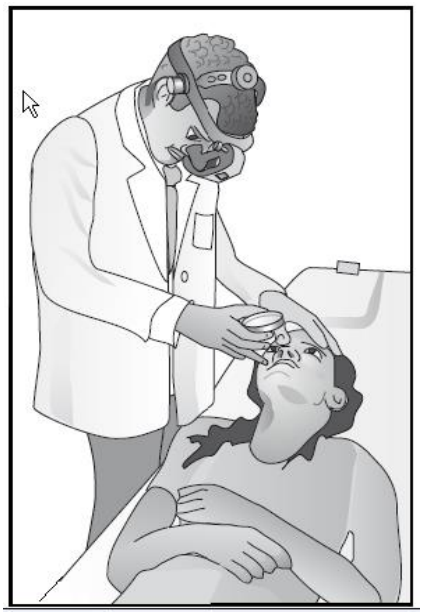


Fig. 28-5 PRACTICE OF INDIRECT OPHTHALMOSCOPY. Use a +20D magnifying lens held close to the patient as shown.

SLIT LAMP MICROSCOPY

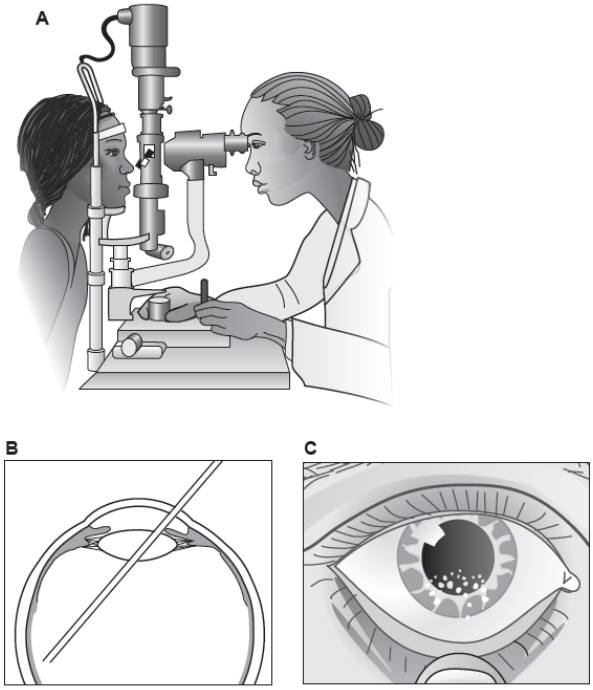


Fig. 28-6 SLIT LAMP MICROSCOPY. A, shines a narrow pencil of light illuminating the eye from an angle while it is examined with a low-power microscope. B, layers of the cornea and lens demonstrated. Particles in the aqueous and vitreous reflect light, like dust particles illuminated by a sunbeam in a darkened room. C, you may be able to see keratic particles directly with bright light.
A-B, After Parr J. Introduction to Ophthalmology, OUP 2nd ed 1982, with kind permission. C, After Trevor Roper PD. Lecture Notes on Ophthalmology, Blackwell 6th ed 1980, p.8.

EXAMINING A BABY'S EYES

Fig. 28-7 EXAMINING A BABY'S EYES.
Sit the baby on the mother's lap and hold the head between your knees.

(h) Slit lamp microscopy

Use this for accurate visualization of the anterior part of the eye and its contents (28-6). Position the head by placing the forehead and chin on the rest. Vary the angle of the light as convenient.

(i) Ultrasound

Using a hand-held 10mHz probe, you can (with a little practice) distinguish much pathology, especially on an otherwise uncooperative child.
You should be able to detect:
(1) Corneal opacities
(2) Hyphaema
(3) Cataract
(4) Vitreous haemorrhage
(5) Choroidal masses
(6) Foreign body
(7) Retinal or choroidal detachment
(8) Orbital lesions
(9) Optic nerve cupping
(10) Optic nerve sheath diameter – this as an indirect measure of raised intracranial pressure.

(j) Basic eye medication

Drugs for use on the eye differ from other preparations: for topical use they come as either ointments or drops. The former are for longer-lasting effect, the latter for immediate and, usually, short-lasting effect.

Some drugs are toxic to the eye through systemic use: these include chloroquine and ethambutol. Others are locally toxic, such as penicillin, or dangerous if used for the wrong condition, such as steroids if used when there is a herpetic corneal ulcer present.

N.B. Many antibiotic eye preparations also contain steroids: avoid these!

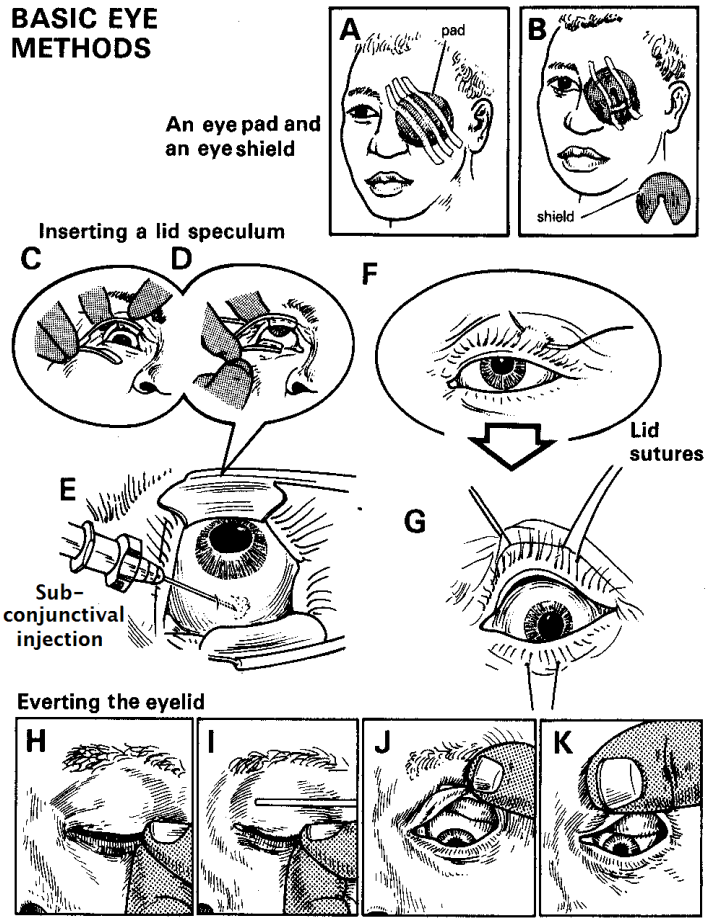


Fig. 28-8 SOME BASIC EYE METHODS.
A, eyepad. B, eye shield. C, insert the upper blade of a lid speculum while the patient is looking down. D, insert the lower blade while he is looking up. E, subconjunctival injection is an effective way of getting a high concentration of an antibiotic inside the eye. F, insert the first lid suture. G, stay sutures in place: 2 for the upper and 1 for the lower lid. H-K, steps in everting the upper lid.

Certain drugs are specifically used to help examination: cyclopentolate 1%, or phenylephrine 10%, will dilate the pupil for some hours only, so use these when you want a temporary effect for example when using an ophthalmoscope. Useful LA agents are lidocaine hydrochloride 4% or amethocaine hydrochloride 1%. Remember that an anaesthetized eye is in great danger if an unnoticed foreign body gets into it, or an abrasion injury is not felt; so shield it (28-8B) after appropriate examination.

To diagnose corneal injury, fluorescein papers are better than fluorescein drops, because you can more readily keep them sterile. To use eye drops, pull the lower eyelid down so that you can see the conjunctiva. Ask the patient to look up. Put drops or ointment into the outer third of the conjunctiva.

Close the eye for 2mins to allow **the drug to enter the eye.** ***Don't let the dropper touch the eye, and don't put the dropper down on a surface***, as it may become contaminated. If possible, patients should keep their own drops, because of the danger of cross-infection.

TO MAKE YOUR OWN CHLORAMPHENICOL EYE DROPS, dissolve two 250mg capsules in 100ml of water. Filter the solution into sterile 10ml dropper bottles. Screw the caps on loosely, and sterilize them in a hot water bath or autoclave at 100ºC for 30mins, without letting water splash over the necks of the bottles. Refrigerate them; their shelf-life is 2 months at 2-8ºC. The shelf-life of commercial drops is only 4 months, so this is a useful method.

Subconjunctival antibiotics are indicated if there is a severe corneal infection or ulceration, especially with a hypopyon.

N.B. If you use 0·2ml of 2% lidocaine, the injection will be almost painless, but *be careful mixing antibiotics and LA in the same syringe to maintain sterility*.

Otherwise anaesthetize the eye with a few drops of LA solution. Ask the patient to look up. Pull down the lower lid, with your finger on the cheek. Use a sharp 0·4mm needle on a 2ml syringe (28-8E).

Rest the needle flat on the conjunctival surface of the globe, with the bevel facing away from it. Push the needle under the conjunctiva, parallel to the surface of the globe, rotating it gently as you do so. If it is in the right layer, you will see its point under the conjunctiva. Then inject 0·5ml (max. 1ml).

If the infection is getting worse, repeat the injection the next day. A severely infected eye is likely to improve, or be lost within 6h, so use a subconjunctival injection usually only once. If you fear the development of an endophthalmitis, use IV antibiotics.

28.2 Operating on the eye

Try to get special training, especially for cataracts. Learn how to do the more important procedures: entropion (28.13), tarsal cysts (chalazion: 28.12), tarsorrhaphy (28.10), and evisceration, enucleation, and perhaps exenteration of the eye (28.14).

(a) Preparation
Prepare the face from the hairline to the chin and from ear to ear, using iodine 10% in a non-alcoholic lotion which will not harm the eyes if it spills on them accidentally. Make a special drape with a slit from the middle of one end to the centre. Place this under the chin, and up each side of the face. Fold it over the head and keep it there with a towel clip. Place another drape across the forehead over the eyebrows, and clip this to the first one. If the patient is intubated, place a third drape over the nose and the mount which connects the patient's endotracheal tube.

(b) Anaesthesia. You can usually use LA, using a retrobulbar block with a 15mm 27G needle; otherwise use ketamine or GA for a perforation (if LA is complicated by retrobulbar haemorrhage, it may aggravate loss of eye contents).

If you are using LA, *don't cover the nose or mouth*.

(c) Position the table so that you can sit comfortably with your knees underneath it. If necessary, put the head at the foot end, or rest it on a board, or sheet of wood, pushed under the mattress, and projecting beyond the table.

Sit your assistant on your left for a right eye, and on your right for a left eye. *Keep your own eyes on the patient.* This means you need to have trained your assistant to hand you the right instruments properly.

Use a speculum, or lid stay sutures, to hold an eye open while you operate on it (28-8G). These sutures serve simply to hold the lids away from the eye while you operate on it. They avoid the risk of a speculum which may press on the eye, and perhaps scratch the cornea.

In the upper lid insert two 3/0 silk or monofilament sutures, just above the lash line and down to the tarsal plate. In the lower lid insert one suture just below the lash line. *Don't penetrate the conjunctiva of either lid*. Hold these sutures with haemostats.

(d) Bleeding. The cornea is avascular and cannot bleed. If the conjunctiva or sclera bleed, apply a pad and very gentle pressure. Or irrigate the wound with saline from a syringe and an irrigating needle. The blood will stream in the clear saline, so that you can see the exact point where it is coming from, and control it with cautery. Heat a squint hook or a small cautery in the flame of a spirit lamp, until it is hot, but not red-hot. Touch the bleeding point with this, through the stream of saline. This will cool its tip enough to prevent burning, but will leave it hot enough to seal the bleeding vessel. *Don't use diathermy*.

(e) Pad the eye if there is a minor injury with no suspicion of perforation (28-8A). An eyepad, with gentle firm pressure, will reduce discomfort, and promote healing by preventing the lids moving over the injured area.

Close the eye, put a pad of gauze over it; place 2 pieces of adhesive strapping diagonally across the pad, from the forehead to the cheek, to hold the pad in place. Change the pad daily, and look for signs of ulceration or infection.

CAUTION! The great danger of an eyepad is that it may rub against an anaesthetized eye, and cause an abrasion. A layer of petroleum jelly gauze on the pad will help to avoid this. *So shut the eye when you apply the pad*.

(f) Shield the eye:
(1) after any severe injury, especially if there is a perforation.
(2) after any eye operation.
Shielding it (28-8B) allows it to open and close, without anything extraneous touching the cornea and perhaps scratching it. A shield is the safest way to protect an anaesthetized eye, and is very helpful for a painful inflamed eye with photophobia.

Cut an 8cm diameter circle from cardboard, or an old X-ray film. Cut a radius in this, fold it into a cone, and maintain the cone with a piece of strapping. Hold the cone in place with two pieces of adhesive strapping, or plastic tape from the forehead to the cheek.

CAUTION! Never occlude the eye of a child <7yrs for >7 days, because this may cause amblyopia (28.9).

28.3 The painful red eye

Acute red painful eyes are due to:
(1) conjunctivitis (much the most common cause at any age). In the newborn this is often due to gonococcus, in children between 6 months and 6 years secondary to measles, and in adults in endemic areas, chlamydia.
(2) a corneal ulcer.
(3) acute iritis.
(4) acute glaucoma.
(5) trauma.

The problem in a busy clinic is that conjunctivitis is so much more common that these other causes are easily missed. So your first task in managing red eyes is to make sure that these rarer causes are recognized. The history, the visual acuity, and the examination of the eye with a torch should enable you to distinguish between conjunctivitis and something more serious.

(a) Diagnosis
If there is conjunctivitis, the discomfort is of a gritty nature caused by rubbing of the conjunctivae on the cornea; pain varies from mild to severe:
(1) Both the eyes are usually involved.
(2) The visual acuity is normally good.
(3) There usually is a purulent discharge.
(4) Red conjunctivae, especially in the fornices (28-1C).
(5) The cornea is clear and does not stain with fluorescein (unless the conjunctivitis has produced a corneal ulcer).
(6) The pupils are normal.
(7) The tension in the globe is normal.

(b) Differential diagnosis
Distinguish particularly between the redness of conjunctivitis, which is typically *bilateral* and maximal at the periphery, but is often uniform everywhere (very common), and redness which is most marked at the corneoscleral junction (less common).

CAUTION! Look for mucopus in the inferior fornix (28-1C): it is *always* present in bacterial conjunctivitis; *hesitate to diagnose conjunctivitis if you don't find any*.

(c) Conjunctivitis can be infectious, allergic, or chemical. Bacterial conjunctivitis is common (especially from *neisseria*, *listeria* and *corynebacterium*) in the developing world, and may be mild, or so severe that the conjunctiva extrudes pus, and the lids swell so much that the eyes remain closed.

Bacterial conjunctivitis needs an antibiotic. Viral conjunctivitis usually resolves spontaneously without, if the cornea is not involved. Allergic conjunctivitis rarely needs steroid treatment.

Besides infecting the conjunctiva, bacteria can infect the lids (blepharitis), or the cornea, where they can cause changes in the stroma (keratitis and sometimes a corneal abscess), which may result in corneal ulceration, through which infection may spread inside the eye as an endophthalmitis, which may end in blindness.

TREATMENT. Clean the eyes with a cotton swab and saline. Instil chloramphenicol or ciprofloxacin ointment hrly in severe infections, and 3hrly if less severe. Continue for 2 days after symptoms have resolved. Allow the exudate to escape, clean the eyes with a clean cloth and water, add an ointment at night to prevent the eyelids sticking together, and *don't put a pad on the eyes*.

If the conjunctivitis is severe, use subconjunctival antibiotics. Watch carefully for a corneal ulcer, and if necessary examine the cornea repeatedly with fluorescein. *If the conjunctivitis is very severe, and especially if there is a corneal ulcer*, instil chloramphenicol eye drops every min for 1h, every hour for 1 day, and then 3hrly.

If the cornea is not clear and the visual acuity is poor, there is a corneal ulcer and the eyesight is in danger.

If a neonate has severe conjunctivitis after birth (ophthalmia neonatorum), this may be *gonococcal* or *chlamydial*. *This is an acute emergency which may cause blindness.* Treat with chloramphenicol drops as above, and add either ceftriaxone or gentamicin IV, and oral erythromycin.

If you are treating a child between 6 months and 6 years, check for a combination of malnutrition, vitamin A deficiency, and recent measles. Look for:
(1) Night blindness (inability to see in dim light).
(2) Bitot's spots (white foamy spots on the lateral conjunctiva).
(3) Xerosis (dryness of the conjunctiva with inability to produce tears, or a dry hazy cornea).
(4) Keratomalacia (corneal ulceration, softening of the cornea).
Treat with vitamin A 200,000IU by mouth immediately, again after 24h, and again after 1wk. Also, use a topical antibiotic such as ciprofloxacin. Improve the nutrition especially with plenty of dark green leafy vegetables.

(d) Chronic low-grade conjunctivitis, characterized by yellow-grey dots (follicles) under the upper eyelid, in someone from an endemic area, is almost certainly TRACHOMA caused by *Chlamydia trachomatis*.

N.B. Different strains of this bacteria cause 3 distinct diseases: urethritis and PID; lymphogranuloma venereum; and trachoma.

Trachoma passes through 4 stages (28.13). During the acute stage, make sure the patient actually puts tetracycline eye ointment 1% into the eyes bd for 6wks. Add a single dose of 1g azithromycin orally.

Advise thorough washing of the face and hands several times daily, avoiding rubbing of the eyes. Explain that the disease is due to the entry of dirt, often from flies, but also from sharing face towels with an infected person.

(e) Allergic conjunctivitis
Suspect this if large gelatinous vegetations have formed on the upper tarsal conjunctiva and look like cobblestones, or on the bulbar conjunctiva surrounding the limbus. It is common in children and young adults. Their eyes may or may not be itchy but typically there is extreme watering. Suppress the inflammation with antihistamine drops or a very weak steroid. Beware of steroid glaucoma (28.6, 28-12D), because steroids, once started, may be needed for many years. Inject triamcinolone 1ml (40mg) IM into the upper eyelid for severe symptoms.

(f) Endophthalmitis (panophthalmitis) may be the result of:
(1) a corneal ulcer, especially bacterial.
(2) a perforating injury of the cornea or sclera, especially if a foreign body has been left *in situ*, or if a wound is neglected, or after recent eye surgery.

Once bacteria have entered the eye, the chance of total blindness is high. If presenting early, when the infection is fairly localized, some useful vision may remain. If you cannot control the infection, an evisceration is necessary (28.14). The anterior chamber is full of pus.

If the endophthalmitis is early, with some hope of vision, try to control infection and minimize pain. Use subconjunctival chloramphenicol and IV gentamicin for 7 days. The infection may settle.

If the endophthalmitis is due to a foreign body in the eye, remove it. It is usually superficial, so that it is possible to remove it through the wound by which it entered, which is usually in the cornea, even if this has to be enlarged. Remove any prolapsing iris, and leave the cornea unsutured. Use subconjunctival chloramphenicol and IV gentamicin.

If presentation is late, with no hope of vision and an anterior chamber full of pus, and the corneal ulcer has weakened, softened, and distorted the globe (phthisis bulbi, 28.4), especially with no improvement after 48h antibiotic treatment, eviscerate the eye (28.14). Be sure that the patient understands the necessity of removing the eye because of the mortal danger of orbital cellulitis and meningitis.

Suggesting acute iritis (28.5): one (sometimes two) moderately painful red eye(s) with no discharge. Pain is often only mild. Reduction in visual acuity is usually mild. A clear cornea is surrounded by redness at the corneoscleral junction. A small constricted pupil which becomes irregular on dilation, due to posterior synechiae (adhesions), is typical. An inflammatory exudate in the anterior chamber is visible most easily with a slit lamp: the aqueous is not as clear as it should be. The beam from the lamp shows a flare, like a beam of light shining across a dusty room. You may also see little lumps of cells (keratic precipitates) sticking to the back of the cornea, and

posterior synechiae between the iris and the front of the lens. The inflammatory cells in the anterior chamber may form a sterile hypopyon. The intraocular pressure may be increased due to secondary glaucoma (28.6).

Suggesting acute angle closure glaucoma (28.6): one (seldom 2) very painful red eye(s) with severe unilateral headache, and slight watering. There is severely impaired visual acuity, often down to hand movements or perception of light only, with haloes, and sometimes even blindness. Circumcorneal hyperaemia is mild in the early stages. A hazy cornea (due to raised IOP) without its normal shine is associated with a shallow anterior chamber; this is best seen by shining a torch from the side. A vertically oval dilated pupil which does not react to light is classical. IOP is raised (28.1).

(g) A corneal ulcer may be due to:
(1) Bacteria.
(2) Herpes simplex virus.
(3) Fungi.
(4) Other conditions such as leprosy, causing incomplete eyelid closure (lagophthalmos) and exposing the cornea to trauma.
Demonstrate a corneal ulcer with fluorescein.

One eye is severely painful with reduced visual acuity (if the ulcer is central); scleral redness is most marked round where the ulcer is situated, with photophobia, swollen eyelids, and watering.

Look for a grey-white spot (the ulcer) on the cornea, which stains with fluorescein. If it is not obvious, look for a defect in the smooth surface of the cornea in the reflection from a focused light.

If the infection is severe, pus cells sediment at the bottom of the anterior chamber, with a fluid level (hypopyon, 28-9C). The pupil is usually regular.

This is an *emergency needing admission*. Start aggressive treatment with antibiotics urgently. A shield (28.2, 28-8B) or sunglasses will make life more comfortable. *Don't use an eyepad or patch.*

If the ulcer is severe, and particularly if there is a hypopyon, inject subconjunctival (28.1) gentamicin 20mg, or chloramphenicol 100mg and apply hourly chloramphenicol 1%, or ciprofloxacin 0·3% eye ointment.

If the ulcer is not so severe, and there is no hypopyon, treat as conjunctivitis.

Also, with any corneal ulcer, provided it has not already perforated, use atropine eye ointment bd or tds to keep the pupil dilated. This will prevent adhesions forming between the iris and the lens (posterior synechiae, 28-9A).

Advise warm soaks: their use is effective for soothing a painful eye. Wrap a cloth round a spoon, dip this into very hot water, and let it cool till you can hold it as close to the eye as is bearable. Soaks are also useful for a stye (infected eyelash follicle, hordeolum).

Use vitamin A supplements if there is any suspicion that it may be deficient.

(h) Complications of corneal ulceration include:
(1) Diffuse scarring of the cornea (28.4).
(2) A dense white scar (leucoma: 28.4).
(3) Perforation of the cornea, with adherence of the iris, and perhaps staphyloma (an opaque protrusion of the cornea, *not related to staphylococci*).
(4) Endophthalmitis.

If there is pain and watering without a history of a foreign body, look for a DENDRITIC ULCER (28-9E). Stain the cornea with fluorescein and look for a branching irregular pattern. This is due to infection by herpes simplex. Dendritic ulcers occur especially after fevers, particularly measles, malaria, and meningitis. If possible, use an antiviral agent: idoxuridine ointment (x5 daily), trifluorothymidine drops (hourly), or aciclovir ointment (x5 daily).

If the lesion is severe, combine this with mechanical removal of the epithelium containing the virus. Apply a topical anaesthetic, and stain the cornea with fluorescein. Using a loupe, a good light, and a ball of cotton wool on the end of an applicator, gently scrub the surface of the cornea in the region of the ulcer to remove its epithelium. A chronic stromal keratitis with corneal scarring and blindness can complicate herpetic eye disease.

CAUTION! Never apply steroids, because these may spread the infection to the stroma of the cornea, and make the condition worse.

Suggesting a foreign body: The signs of an abrasion, and a foreign body, are similar to those of a corneal ulcer: unilateral pain, photophobia, a watery discharge, sometimes impaired vision, and hyperaemia, which is marked near the lesion. Ask if there is a history suggesting trauma, and *don't forget that contact lenses are foreign bodies and easily become infected if not kept scrupulously clean. Check underneath the upper eyelid!*

(i) Difficulties with red painful eyes
If a chemical has got into the eye, the conjunctiva is intensely red (more so than in infective conjunctivitis), the cornea may be opaque (from keratitis or an ulcer), and the vision impaired. Unlike in infective conjunctivitis, mucopus is absent. Traditional medicine is sometimes inserted for a painful eye, which makes it worse.

If the chemical is still present, wash it out with much water, making sure it does not spill over the other eye. Remember that if it is acid or alkali, a 1ml cavity needs 1L water to alter the pH by a level of 3. Use an analgesic, and shield the eye. Instil an antibiotic ointment; its paraffin base will be soothing, and the antibiotic may prevent secondary infection.

If there is an acutely inflamed and oedematous lid or face, with a black slough, and surrounding thick oedema, this may be ANTHRAX, especially if there has been contact with animal carcasses or hides.

The eyelid may be completely destroyed, but the eye is normal. Use high doses of IV penicillin and sulphonamides. Anthrax responds rapidly to penicillin. Later, if necessary, toilet the slough and graft the raw area. If you leave raw lids ungrafted, *severe scarring and a scar-induced ectropion (lid eversion) will follow*.

THE IRIS & CORNEA

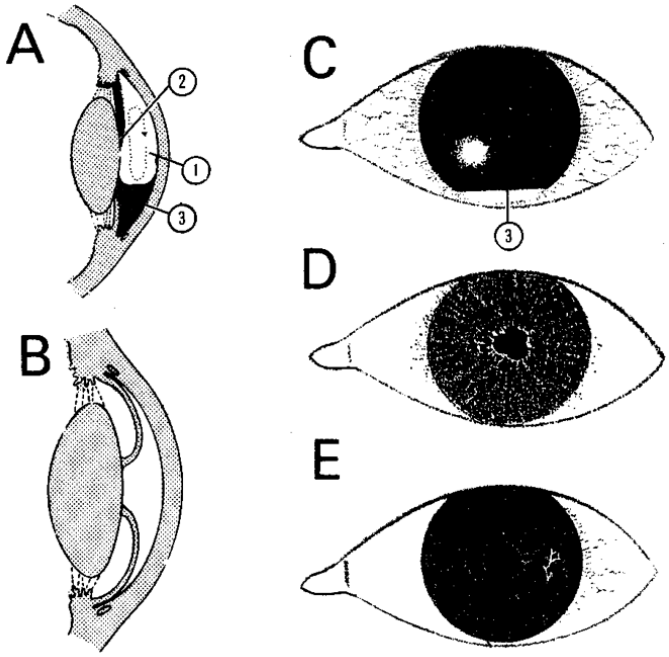


Fig. 28-9 THE IRIS AND THE CORNEA. A, vertical section of the eye showing keratic precipitates floating in the aqueous (1), posterior synechiae (2), and a hypopyon (3). B, iris bombé: the iris is adherent to the lens all round and is bulging forwards. C, acute bacterial corneal ulcer with a hypopyon. D, acute iridocyclitis. The pupil is small and irregular, because posterior synechiae have formed. E, dendritic ulcer of the cornea, the result of herpes simplex infection. *After Parr J. Introduction to Ophthalmology, OUP 2nd ed 1982 with kind permission.*

28.4 Loss of vision in a white eye

This is one of the common presentations of eye disease. Loss of vision in a white eye can be slow or fast.

If there is slow loss of vision over months or years, there may be:
(1) A corneal scar.
(2) Cataracts.
(3) Glaucoma.
(4) A refractive error.
(5) Disease of the retina due to:
  (a) Senile macular degeneration.
  (b) Retinitis pigmentosa (congenital photoreceptor deficiency).
  (c) Chloroquine maculopathy.
  (d) Old macular scars.
(6) Optic atrophy.

If there is sudden loss of vision over minutes or days, the cause is usually inflammatory or vascular.

If the complaint is simply that reading is difficult, especially in poor light, this is usually presbyopia (28.8).

(a) Corneal scars cause 70% of blindness in children and 25% in adults in the developing world. They can be:
(1) Diffuse.
(2) A circumscribed white patch (leucoma).
(3) A staphyloma, which is a bulging of the cornea forwards between the lids, due to its thinning, caused by previous ulceration *(not staphylococci)*.
(4) Phthisis bulbi, which is disorganization of the entire eye, leaving it small and shrunken.
Bilateral scarring follows neonatal conjunctivitis (ophthalmia neonatorum), vitamin A deficiency, traditional eye medicine and trachoma (28.3,13). Unilateral scars are more likely to be caused by corneal ulceration due to bacteria, herpes simplex, fungi or trauma.

If the cause of the scar is still present, and it is getting worse, remove the cause. This may include scratching of the cornea by the inwardly turned eyelashes of trachoma (*trichiasis*, 28.13). Vitamin A deficiency causes an acute ulcer in young children, and does not cause progressive scarring.

If there is still adequate vision in the other eye and disability is not severe, no treatment is indicated.

If there is no light visible at all, explain that nothing can be done.

If there is blindness, and a central leucoma which obscures the pupil, with an area of clear peripheral cornea, a peripheral iridectomy is necessary. This will provide an artificial pupil peripherally, behind the area of clear cornea, and should give enough vision for independent mobility. It is contraindicated if there is already enough vision for mobility, or if the peripheral cornea is opaque.

If there is blindness due to diffuse corneal scarring which has not made the eye perforate, a corneal graft is the only solution.

If the eye is blind and painful, consider evisceration or enucleation (28.14).

(b) Cataracts cause about 50% of blindness in the world, where the incidence is c.1:200. A large majority (85%) of cataracts occur in the elderly, and the rest are either congenital or familial, or due to trauma, iritis, or diabetes. Vitamin D deficiency causes lamellar (flaky) cataracts in infants. Cataract presents with gradual loss of vision, in one of or both the eyes. The corneas are clear, and there is an opacity in the pupil(s). A cataract can be immature (making the pupil grey), or mature, or hypermature (making it white). Sometimes a cataract swells, pushes the iris forwards, occludes the angle of the eye, and causes secondary glaucoma.

Removing cataracts is a standardized and repetitive task; it is also a skilled one but *is rarely urgent*. To learn this, it is best to apprentice yourself to an expert for several months, and try to remove at least 50 under supervision. Or, better, send a motivated assistant to learn this skill. Cataracts can often be removed on a mass scale in special 'eye camps'. In good hands the chance of success is >90%. If you operate on a patient for the right indications, even moderate success in one eye only will provide much-sought-for independence.

Aim to insert an intraocular lens (IOL), which provides much better vision. The IOL can now be obtained for a reasonable price and is made in Eritrea and Nepal for example, so this is no longer an impossible dream in the developing world. It should be standard, as refractive errors are better corrected and waiting for maturity of the cataract is no longer necessary.

Manual small incision cataract surgery (MSICS) has become the preferred extracapsular method for removal of cataracts in low-income settings. It does not require sutures, can be done inexpensively, and produces high-quality results. *High-cost cataract removal alternatives are not necessary*.

An operating microscope is especially valuable for reducing the incidence of complications; the less experience the surgeon has, the more important the microscope quality becomes. If you are more experienced, you can use loupes, but if you are less experienced, you may cause problems which you may not even see. A cheaper special MSICS microscope is currently being developed.

Bad outcomes are related to:
(1) poor case selection, *i.e.* operating on patients who actually have a corneal scar or glaucoma,
(2) complications such as vitreous loss or infection,
(3) uncorrected refractive error,
(4) post-operative posterior capsule opacification.

(1) Measure the visual acuity accurately in both eyes. The pupils should react briskly to light. If they don't, suspect that there is also some other condition, such as optic nerve disease.
(2) Measure the IOP to make sure that the loss of vision is not due to glaucoma (28.6).
(3) Dilate the pupil and examine the red reflex with an ophthalmoscope to assess how dense the cataract is, especially if it is immature. If you can easily see the optic discs, the cataract may not yet be dense enough to be worth extracting.

CATARACT EXTRACTION (GRADE 2.5)
INDICATIONS
(1) To improve sight.
(2) To treat complications, especially secondary glaucoma.

If there are bilateral cataracts, operate when the acuity in both eyes has fallen to worse than 6/60 (CF at 6m).

If there is a unilateral cataract, surgery is only indicated to treat or prevent secondary glaucoma, or uveitis. It will not improve sight significantly.

If there is already loss of sight in the other eye for any reason, and there is now a cataract in the remaining eye (cataract in an only eye), delay surgery until there is difficulty getting around independently and near blindness (CF<3m), because any complication will cause total blindness.

If one cataract has already been successfully removed, you can schedule the second cataract extraction at any time. But, this case now will be a lower priority.

If the cataract extraction is not possible, atropine ointment weekly, or minus (concave) glasses may improve eyesight.

CONTRAINDICATIONS
(1) Unilateral cataracts with adequate sight in the other eye.
(2) Bilateral small immature cataracts with acuity above 6/60 in both eyes together; review the progress in 3-6 months.
(3) Active uveitis: *don't perform a cataract extraction at the same time as an iridectomy*.

METHOD
The principle is to make a self-sealing tunnel to extract the cataract and insert the new lens. It may be combined with a trabeculectomy (28.6).

Dilate the pupil with cyclopentolate 1%.

Insert the eyelid retractor, or place stay sutures on the eyelids. Grasp the *superior rectus* at its insertion, about 7·5mm behind the limbus (28-1C) and rotate the eye inferiorly; insert a 4/0 or 5/0 stay suture through the conjunctiva and beneath the muscle (28-10A). Raise a flap by picking up the conjunctiva at the superior limbus (junction of sclera and conjunctiva) and buttonhole the conjunctiva with fine scissors. Extend this in the sub-Tenon's space and lift the conjunctiva and Tenon's capsule (28-1C) off the sclera for 1cm (28-10B). Ensure haemostasis with cautery and that the field is dry before you proceed to the next step.

Make a 6-7·5mm long curvilinear scleral partial thickness (0·3mm deep) shelved incision 3mm posterior to the limbus on the superior side of the eye (28-10C). Deepen this incision by advancing the crescent blade into the sclera and slowly cutting on either side, thus making further room for the blade. Judge the correct depth by making sure the crescent remains visible through the sclera (28-10D).

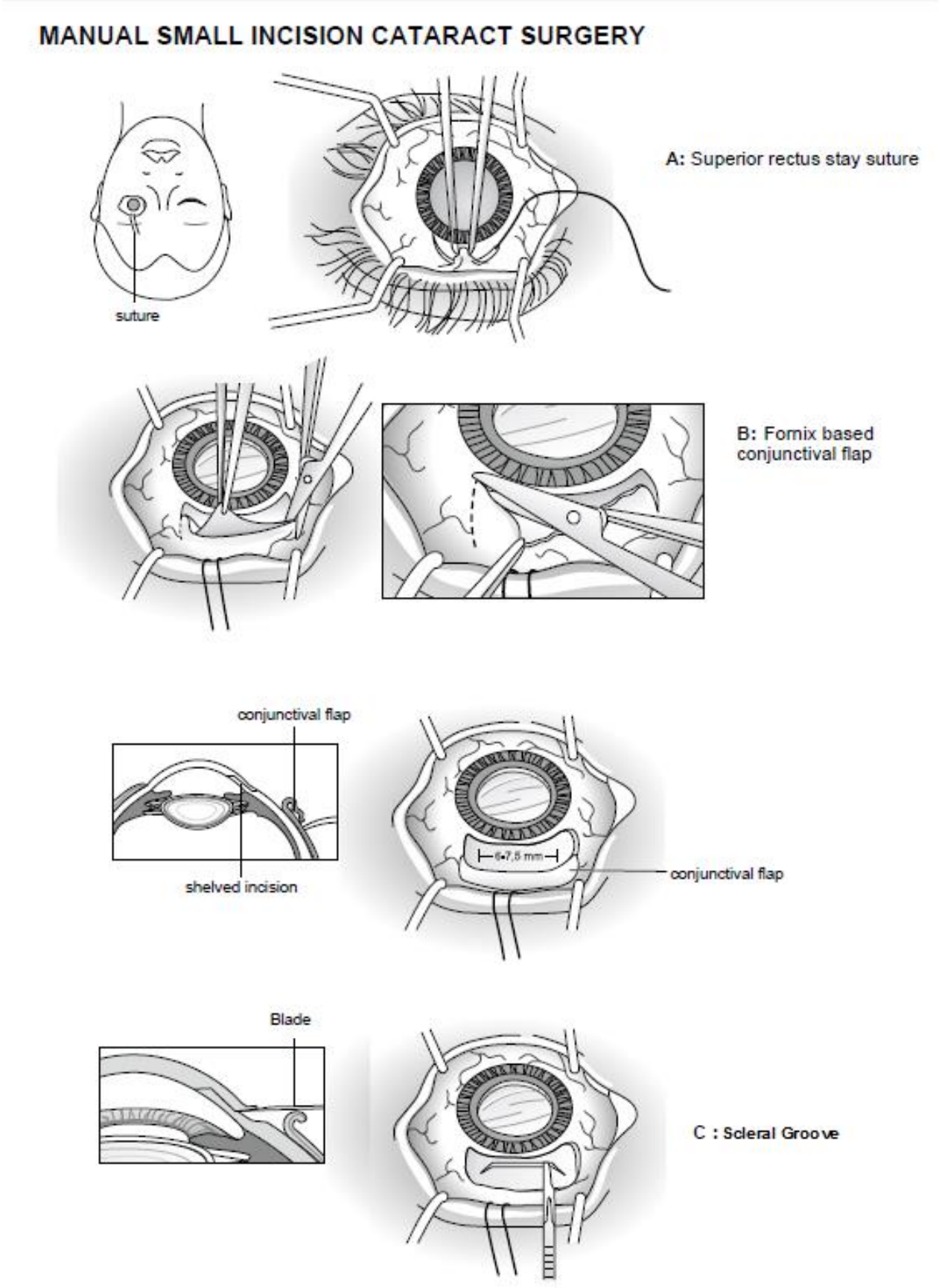
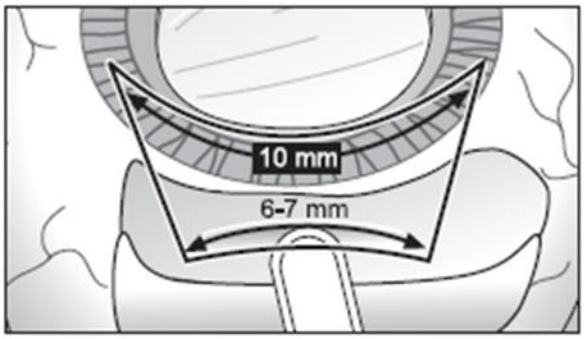
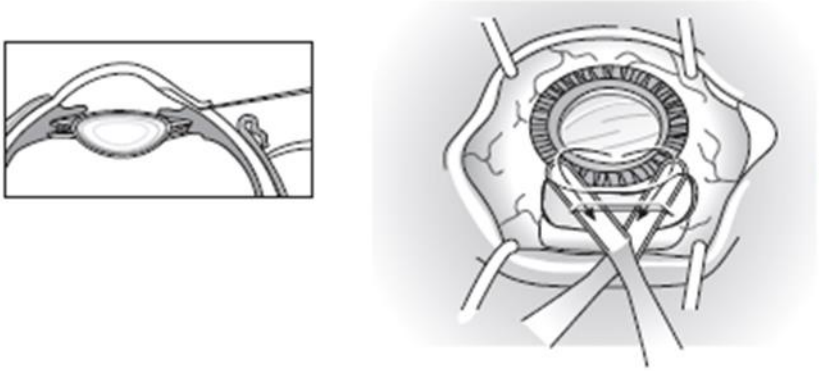


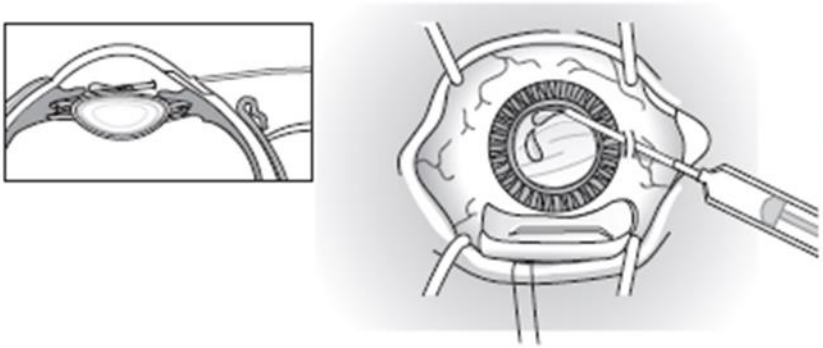
Fig. 28-10 MANUAL SMALL INCISION CATARACT SURGERY. A, *superior rectus* stay suture. B, fornix-based conjunctival flap. C, scleral groove: a 6-7·5mm shelved incision posterior to the limbus on the superior side of the eye.



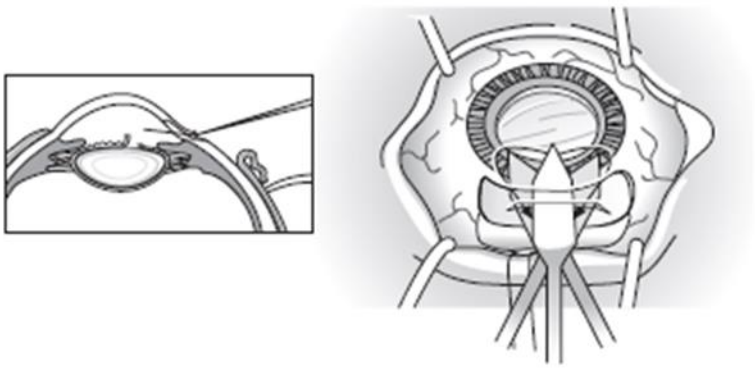
D : Making the tunnel trapezoid shaped (internal 10 mm, external 6-7 mm)



E : Preparing the scleral tunnel

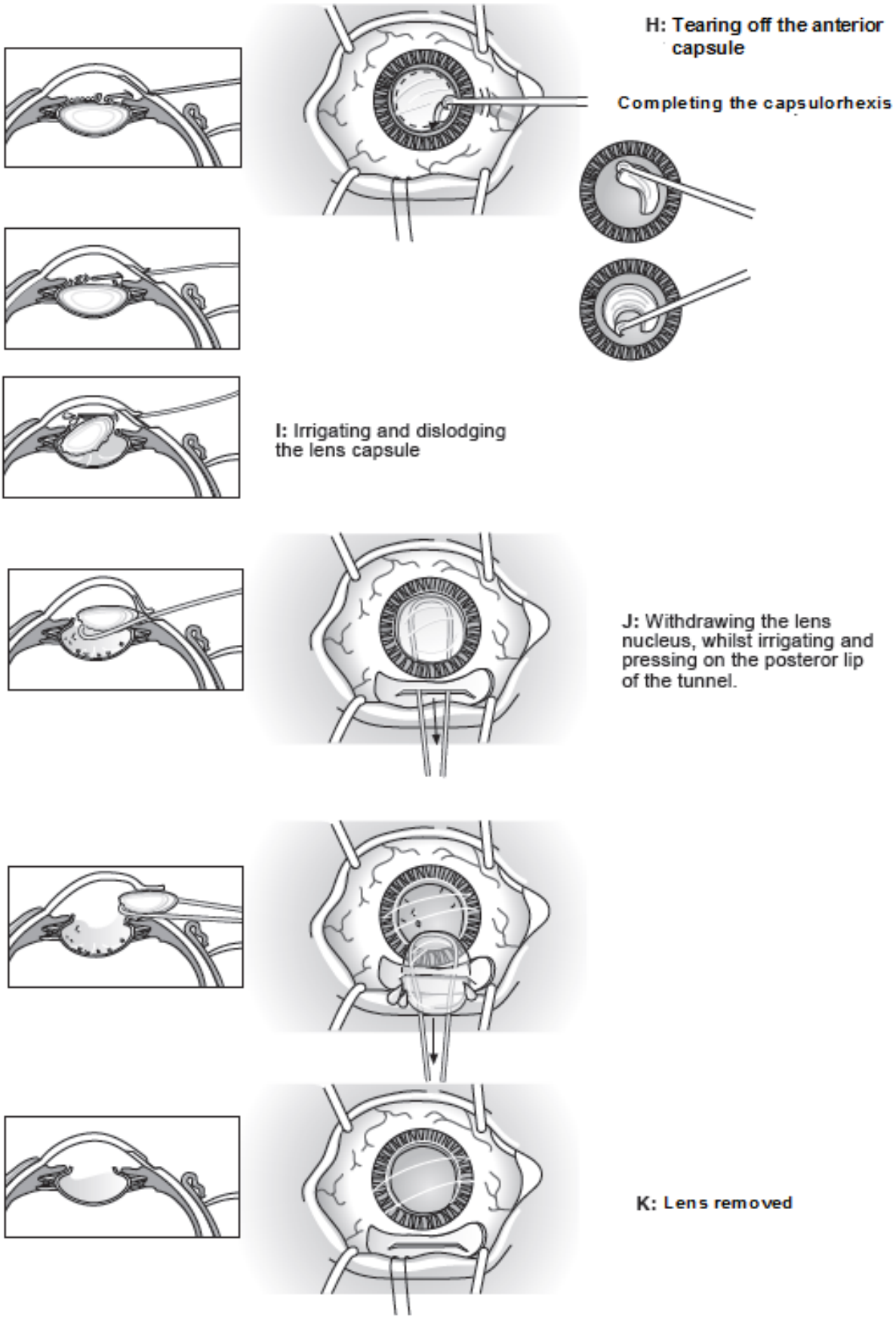


F Paracentesis: injecting dye to stain the lens capsule and air to protect the underside of the cornea

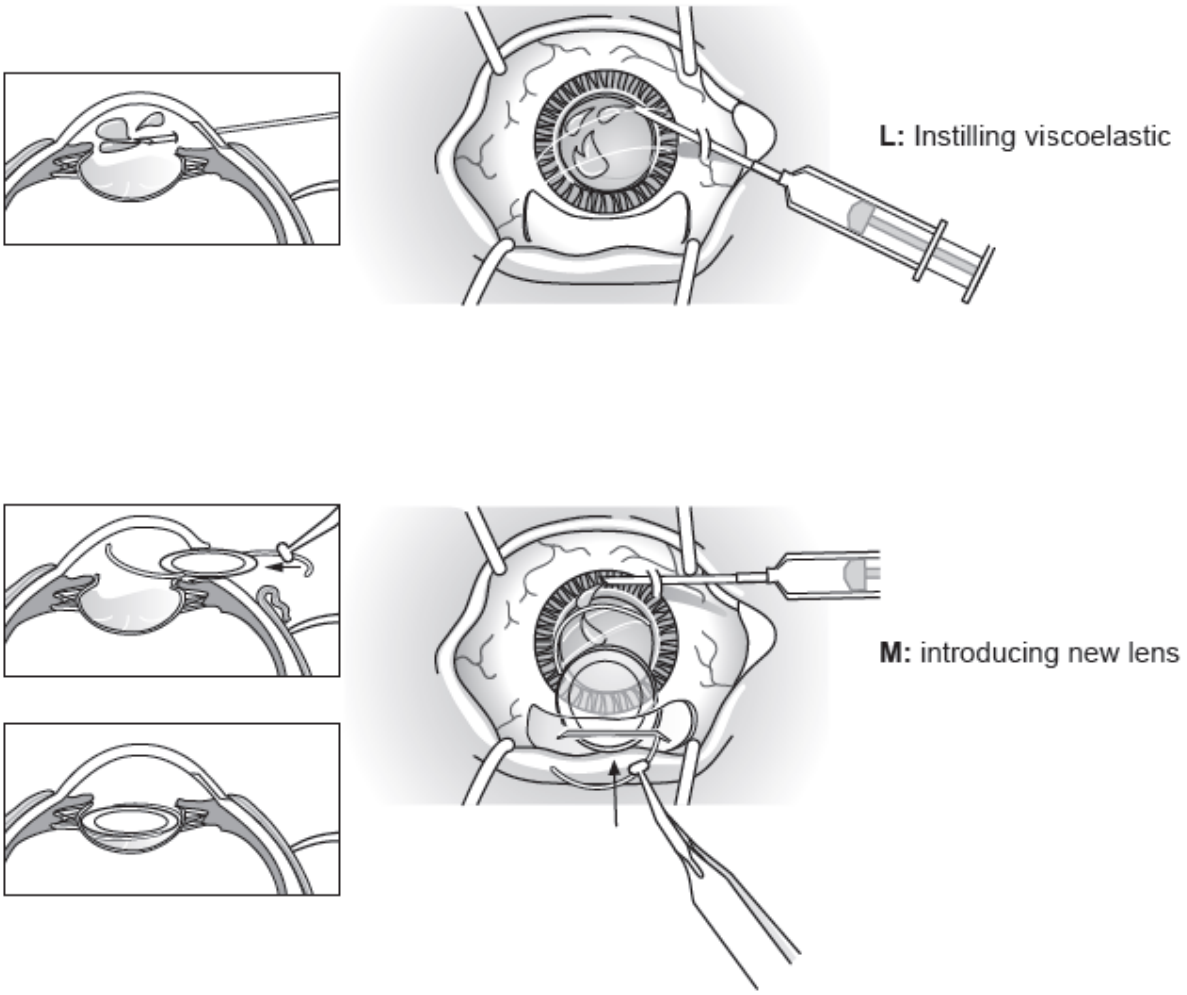


G: Piercing the anterior capsule in a complete ring

D, make the scleral tunnel trapezoid-shaped with a crescent blade. E, enlarge the tunnel. F, paracentesis: make a sideport and inject dye to stain the lens capsule and air to protect the underside of the cornea. G, capsulotomy: pierce the anterior capsule in a complete ring c.7·5mm in diameter with a keratome.


H: Tearing off the anterior capsule
Completing the capsulorhexis
I: Irrigating and dislodging the lens capsule
J: Withdrawing the lens nucleus, whilst irrigating and pressing on the posteror lip of the tunnel.
K: Lens removed

H, capsulorhexis: tear off the anterior capsule with a special hook or one made from a 27gauge needle. I, irrigate and dislodge the lens capsule. *Avoid touching the inner surface of the cornea!* J, introduce the lens loop within the capsular bag under the lens nucleus, and slowly ease both loop and lens out, whilst irrigating and pressing on the posterior lip of the tunnel. K, lens removed. *Don't irrigate >30secs. Don't aspirate the posterior capsular surface!*



L, instil 0·3ml viscoelastic (a cohesive substance usually of sodium hyaluronate 1·4%) and air to re-create the anterior chamber and seal the incision. M, introduce the intraocular lens into the capsular bag, holding onto the trailing haptic, which should curve to the right. *Make sure the lens is the correct way up! Don't hold onto the lens itself!*

Keep the crescent flat on the globe during dissection, so that the tunnel depth remains uniform. Once you reach the limbus, extend the tunnel by forward and backward motion, cutting as you come out; this way, you create scleral pockets on either side of the tunnel which becomes trapezoid in shape with its inner margin 7-8·5mm, *i.e.* larger than the outer margin 6-7·5mm long, adapted to the size of the nucleus (28-10E).

Pierce the cornea just above the limbus to enter the anterior chamber with the keratome. At this point you can make the lens capsule more visible, by injecting 0·2ml trypan blue dye through a sideport, using a 25gauge cannula and adding a little air which protects the underside of the cornea. Wait a full 30secs *but no more*, otherwise you will dye all the tissues blue. Then wash out the dye with balanced saline (0·9% saline made up to pH 7·3 with bicarbonate). Then deepen the chamber by injecting 0·3-0·5ml air (28-10F).

Now open the tunnel into the anterior chamber by advancing a keratome through the tunnel, tilting it downwards, and advancing into the anterior chamber (28-10G). Move the keratome medially and laterally the full length of the tunnel while keeping the tip of the blade in the anterior chamber. Insert a 27G needle with the tip bent slightly downwards like a hook.

Press the hook into the anterior capsular lens surface to create a circular 360° opening about 7·5-8.0mm cutting parallel to the limbus (28-10H).

Irrigate this space to help free the nucleus (28-10I); rocking it side to side or turning it round may help free it. Then introduce a lens loop into the tunnel, and pass this under the cataract in the capsular bag and slowly ease it and the nucleus out of the anterior chamber (28-10J), at the same time pressing gently on the posterior lip of the tunnel to help expel it. *Avoid touching the inner corneal surface*.

Continue irrigating as you manipulate the lens nucleus out, and once it is out, pick up the sclera edge and aspirate any cataract fragments, leaving a clear anterior chamber. Keep pressing gently on the posterior lip of the tunnel to allow debris to flow easily out. *Don't irrigate for longer than 30secs. Don't aspirate the posterior capsule.*

Inject 0.3ml viscoelastic (28-10L) and insert a 21 dioptre polymethylmethacrylate (PMMA) intraocular lens into the capsular bag (28-10M). *This is the standard size and is suitable for 80% or more of patients. Make sure the lens is the correct way (flat or concave surface) up!* Hold the lens with long smooth (*e.g.* McPherson long-angled) forceps with the leading haptic (curved hook attached to the lens) sweeping to your left and the trailing haptic curving to the right. Advance the lens by holding the haptic with the forceps, *but don't hold the lens itself with the forceps*.

Re-create the anterior chamber by injecting 0·3-0·5ml air through the sideport without applying any pressure on the tunnel (28-10M). No sutures are required since the properly formed tunnel acts as a one-way valve to prevent leaks. The conjunctival flap becomes covered by the eyelid, and needs no suture. Apply topical chloramphenicol or ciprofloxacin, and dexamethasone 0·1% 3hrly for 1wk and then 6hrly for 2wks, with or without LA.

N.B. Don't forget to remove the stay suture in the superior rectus *muscle!*

POST-OPERATIVELY, watch for a leaking wound (with or without iris prolapse), infection, bleeding, and a raised IOP. Gently open the lids, and examine the eye with a torch.

If the patient is restless and expels the air bubble in the anterior chamber causing it to flatten, take him back to theatre and re-inject air through the wound. If this keeps leaking out, suture the tunnel with 10/0 nylon.

If the remaining lens matter is swollen and fluffy, keep the pupil dilated with atropine drops 1% bd.

If there is any iris prolapse, return to theatre, reduce the prolapse, make sure the anterior chamber is filled with balanced saline, and suture the wound.

If the cornea is hazy with a striate pattern (striate keratitis), it will probably settle.

If there is blood in the anterior chamber (hyphaema), pad the eye and insist on bed rest.

If the anterior chamber is shallow and the pupil not round, the wound may be leaking. (You may prove this with a fluorescin test.) Return to theatre and wipe the wound with cellulose swabs, fill the anterior chamber with 0·3-0·5ml air, and close the wound properly.

If there is pus in the anterior chamber (hypopyon), there is infection (endophthalmitis). The eye is likely to be painful and the visual acuity very low. Use subconjunctival gentamicin or cefuroxime (28.1), and topical chloramphenicol or ciprofloxacin hourly. The eye may be lost in any case.

If the red reflex is absent after several months, there is some opacity in the media. This may be from re-growth of new fibres in the posterior capsule; perform a CAPSULOTOMY by holding the *medial rectus* tendon in forceps and passing a keratome through the cornea laterally backwards to cut the posterior capsule. Then keep the pupil dilated with 2 drops atropine 1% bd and add 2 drops chloramphenicol 0·5% 4hrly for 3 days.

If there is much pain and the cornea is hazy, the IOP is probably raised (aphakic glaucoma), so measure it. The vitreous jelly may be blocking the pupil. Immediately dilate the pupil with cyclopentolate and phenylephrine drops, followed by atropine ointment for 6wks.

If visual acuity is not improved and there is no evidence of endophthalmitis, increase topical steroid to 2hrly and check for improvement in 1wk.

(c) Rarer causes of gradual loss of vision in a white eye

Examine the macula and the optic cup with particular care. For many of the following, *there is no remedy*.

If an old person has gradual loss of central vision, atrophy, and irregular pigment at the maculae, suspect senile macular degeneration.

If there are pale, white, flat optic discs (distinguish these from the pale cupped discs of glaucoma, 28-11C), **and normal maculae**, there is optic atrophy. Try to find the cause (there are many, including a space-occupying lesion around the optic chiasma).

If there is gradual loss of vision at any age, often starting with night blindness, a family history, and dark pigmentation which follows the retinal vessels and takes the form of 'bone spicules', suspect retinitis pigmentosa, a congenital disease of photoreceptor loss.

If there is gradual loss of central vision from excessive doses of chloroquine (>1·5g weekly for >1yr), **or ethambutol**, suspect maculopathy. The macula has a typical 'bull's eye' pattern with a dark centre and a paler surrounding ring. Stop the drugs.

If there are old macular scars (large white areas with black edges, often around the optic disc and the macula), they may be due to previous *toxoplasmosis* (treat with pyrimethamine 25mg od and cotrimoxazole 40mg/kg od for 3wks) or *toxocariasis* (treat with albendazole 5mg/kg bd for 5 days).

(d) Sudden loss of vision in a white eye

Loss of vision can occur over minutes, hours or days, in one or both eyes, which are white.

If at any age there is steady loss of vision over 24h, in one eye or occasionally both eyes, suspect posterior choroiditis (28.5) due to *toxoplasmosis* or other causes. The important sign is inability to see the retinal vessels due to hazy vitreous caused by inflammatory cells. Treat as above.

If symptoms started with a flash of light followed by black objects floating in the field of vision, and then a curtain or cobweb across it, suspect retinal detachment. Part of the retina may look grey-green. Dilate the pupil and examine the fundus. You will see an abnormal red reflex in one part of the fundus, with elevation of part of the retina, and tortuosity of its vessels, which are difficult to focus on. Expert surgery may save eyesight.

If there is instantaneous loss of vision, suspect occlusion of the central retinal vein (a swollen disc with many haemorrhages all over the retina) or artery (a swollen disc, oedema of the retina, and often a cherry-red spot at the macula). Or, suspect a stroke (cerebrovascular accident). Check the blood pressure. If it is not elevated, and visual loss is less than 6h old, start anticoagulants. There is otherwise no definitive treatment. If there is central retinal vein thrombosis, follow up to check for secondary glaucoma, which needs treatment.

If there is loss of central vision with an abnormal pupil response to light, suspect optic neuritis (any age, usually in the 3rd and 4th decades, and usually unilateral). The vitreous and optic disc are usually normal. This will usually improve over about 8wks. Bilateral optic neuritis following methyl alcohol or quinine is permanent. There is no specific treatment.

28.5 Anterior uveitis (iritis and iridocyclitis) and posterior uveitis (choroiditis)

Any part of the uveal tract can become inflamed: the iris (iritis), the ciliary body (cyclitis), or the choroid (choroiditis). More than one part may be involved at the same time (iridocyclitis).

Although iridocyclitis may be caused by bacteria invading the eye through a corneal ulcer (28.3), it and other forms of uveitis are more often due to a sterile inflammation, usually from an unknown cause. Uveitis of several kinds is common.

Iritis (more strictly iridocyclitis) has several consequences:
(1) The inflamed iris may stick to the lens by posterior synechiae (adhesions) or, less often, to the back of the cornea by anterior synechiae.
(2) If the entire margin of the pupil sticks to the lens, the iris balloons forwards (iris bombé: 28-9B), and causes secondary glaucoma (28.6).
(3) Abnormal proteins enter the aqueous, and cause an aqueous flare, which you can see with a slit lamp. You can also see leucocytes as tiny particles floating in the aqueous.
(4) These particles may stick to the back of the cornea as keratic precipitates, and they may be numerous enough to gather at the bottom of the anterior chamber similarly to a hypopyon.

Unlike the hypopyon that results from entry of bacteria through a corneal ulcer, the fluid in iridocyclitis is usually sterile. Untreated iridocyclitis eventually subsides spontaneously, typically in c.6wks, leaving the eye severely damaged. It may relapse, or it may be insidious and chronic, with few symptoms except progressive loss of vision.

Uveitis presents in 2 ways (or when in both ways together as panuveitis): as anterior uveitis (iritis) or posterior uveitis (choroiditis). The former presents as an 'acute red eye', so being one of the important differential diagnoses of conjunctivitis (28.3), whilst the latter presents as progressive loss of vision in a white eye (28.4).

Iritis is usually a sterile reaction to one of the infections listed below. If *onchocerciasis* (28.7) is endemic, it will certainly be the most common cause. Usually, no cause is found, and iritis is presumed to be due to an autoimmune disease. Atropine will keep the pupils well dilated, and help break down synechiae. Steroid use is controversial: it probably hastens resolution, *but don't use it if there is any sign of infection, especially a corneal ulcer*.

Remember also that steroids:
(1) will make a red eye white, regardless of the cause, without necessarily curing it;
(2) will suppress the normal inflammatory response, without killing the causative agent;
(3) may raise intraocular pressure, and may rarely cause a secondary glaucoma that could produce blindness;
(4) may cause a cataract if used long-term, *but this will not happen in the short time needed to treat acute iritis*.

(a) Diagnosis
Uveitis may be unilateral or bilateral, and presents in various ways.

(b) Acute anterior uveitis (iritis, iridocyclitis: 28.3) presents as a red, painful eye, with photophobia, lacrimation and often blurred vision. There is circumcorneal redness, and often general hyperaemia also. The pupil is constricted.

(c) Posterior uveitis (28.4) mainly involves the choroid, and presents as fairly sudden loss of vision over 24-48h in a white and usually painless eye, due to damage to the retina and an exudate of cells and pigment into the vitreous. After dilation, you can see these as a vitreous haze with an indistinct retina. At a later stage, when the haze has cleared, you may see foci of white depigmentation, surrounded by heaped up black pigment which results in impaired vision, especially if it involves the macula.

(d) Panuveitis (quite common) is a combination of anterior and posterior uveitis, and causes loss of vision in a red, painful eye.

(1) CAUSES
You will probably find no cause, but if any of these are present elsewhere, they may be responsible: syphilis, tuberculosis, leptospirosis, leprosy, *herpes*, *toxoplasmosis*, *toxocariasis*, *onchocerciasis* (28.7), HIV disease, Ebola (in survivors), trauma, or leakage of lens protein from a hypermature cataract.

(2) TREATMENT
Dilate the pupil with short-acting mydriatics (cyclopentolate and phenylephrine). When the pupil is dilated, maintain atropine ointment 1% tds, until the uveitis is no longer active, as shown by the absence of KP bodies and redness. This will prevent posterior synechiae, which would lead to the complication of secondary glaucoma and cataract, and so blindness. So, keep the pupil dilated until all the inflammation has subsided.

If the disease is unilateral, cover the eye with a shield (28.1) if it is severe, and a shade if it is not.

If the disease is bilateral, use eye shades. You can make these from exposed X-ray film.

STEROIDS
Iritis will subside spontaneously, but topical steroids will hasten its resolution.
CAUTION! NEVER use steroids if:
(1) there are signs of infection.
(2) there is a corneal ulcer.

If there is anterior uveitis (iridocyclitis), instil hydrocortisone drops 1% into the conjunctival sac 3hrly.

If there is posterior uveitis, use oral prednisolone 20-30mg/day for 3-6wks. *Don't continue beyond 6wks*. Tail this off over 1wk at the end of the course.

If the IOP is raised, add oral acetazolamide 250mg qds, until the inflammation is under control. Double the dose if the IOP remains raised. If possible, monitor the IOP weekly by tonometry (28.1).

(3) DIFFICULTIES WITH UVEITIS
If posterior synechiae develop, a cataract may follow. The adhesions may occlude the pupil and cause pupil block glaucoma (iris bombé) with an increased IOP. Be sure to dilate the iris vigorously with atropine, so that it does not stick to the lens.

If there is secondary glaucoma, the pupil will not dilate. Try to arrange an iridectomy, but it is often too late even to recommend this.

28.6 Glaucoma

Glaucoma is a group of blinding diseases in which the intraocular pressure is usually raised, causing damage to the optic nerve, and resulting in loss of vision.

There are 4 kinds:
(1) Primary (chronic) open-angle glaucoma (POAG).
(2) Primary angle closure glaucoma (ACG).
(3) Secondary glaucoma, as a complication of trauma, swollen cataract, iritis, etc.
(4) Congenital glaucoma (buphthalmos).

(a) Open-angle glaucoma (POAG, chronic glaucoma) occurs in eyes in which the angle between the iris and the cornea is normal, and is probably due to a block in the drainage of intraocular fluid at the trabeculum (28.1). POAG causes most cases of glaucomatous blindness in Africans. Up to 1% of those >40yrs may be affected.

POAG is bilateral, but is often asymmetrical; it is insidious and progressive, and causes no symptoms until much eyesight is already lost. Glaucoma cannot be prevented, and even early treatment cannot restore lost vision. The best that can be done is to recognize it early, and to prevent vision getting worse.

For this to be possible, all health workers must be aware of the possibility of glaucoma in any patient who complains of loss of vision. The key to early diagnosis is to pick up early changes in the optic discs and a raised IOP, both of which can be recognized by eye assistants. The aim of medical and surgical treatment is to lower the IOP to a level which will stop further damage to the optic nerve, and therefore preserve vision at its present level.

Trabeculectomy is a relatively simple operation, with a reasonable chance of preserving what vision still exists; however, you may need to repeat the operation within 2yrs because recurrence is common. Learn it from an expert at the same time that you learn cataract extraction.

The symptoms of POAG are non-specific. There is slow loss of vision in one or both of the eyes over months or years (28.4). Sometimes, there is marked loss of vision in one eye while the other eye is normal or nearly so. Occasionally, there is pain and headache, but this is late. Glaucoma is often familial.

DIAGNOSIS. Measure the visual acuity of any patient who presents with loss of vision. A hazy cornea or a pupil which does not respond normally to light should make you suspect glaucoma. If the IOP is >28, or the cup/disc ratio is >½, there may be glaucoma (28-12G,H,I). The end stage of glaucoma is a patient with a blind or nearly blind eye, with a large pupil that does not react to light. Loss of visual field is an early sign, but is not easy to test with simple equipment.

CAUTION!
(1) The level of IOP is useful in confirming glaucoma, but is not absolute, and is not always related to optic nerve loss.
(2) Glaucoma can occur with a normal IOP (30% of patients with glaucoma have an IOP <22mm Hg).
(3) The IOP fluctuates, so if you are in doubt, repeat the measurements over a few days.
(4) *LOSS OF VISUAL ACUITY is not an early sign in POAG*.

Aim to diagnose glaucoma long before blindness with the following three tests.

(i) CUPPING OF THE OPTIC DISCS is the most important sign. Chronic glaucoma causes the discs to become deeper and wider, and the remaining rims of disc tissue to atrophy (28-11). One eye is commonly affected more than the other, so that a definite difference between the eyes is probably abnormal.

Enlargement starts at the upper or lower margins, so that a vertically ovoid cup with a cup/disc diameter ratio of >½ is probably abnormal. Eventually, the margin of the cup approaches the margin of the disc, so that only a narrow rim of tissue remains. Its wall becomes steep, so that vessels bend abruptly as they reach the level of the surrounding disc. If the edge of the disc overhangs the cup, you may lose sight of the vessels until they appear over the edge of the cup.

A large physiological cup can, however, be difficult to distinguish from that in early glaucoma. With practice, eye assistants should be able to distinguish 'normal discs', 'suspicious discs' and 'advanced glaucomatous cupping'. This is the most important sign to look out for in glaucoma: you usually need to dilate the pupils.

NORMAL DISCS:
(1) The discs are the same in both eyes.
(2) The ratio of optic cup to optic disc is ≤½ (28-12G).
(3) The cup is circular and the periphery of the disc (the optic nerve rim) is pink.
(4) The appearance of the disc remains constant over time.

SIGNS SUGGESTIVE OF GLAUCOMA:
(1) A cup/disc ratio >½.
(2) A vertically oval cup, perhaps with notching at the upper or lower poles.
(3) An area of pallor >30% of the disc area.
(4) Asymmetry of the cup/disc ratio of the 2 eyes.

(ii) ABNORMAL PUPIL RESPONSES are a useful way of testing for glaucoma, and only a torch is needed. Initially, one pupil does not react as briskly as the other. Finally, there is no response at all.

If you shine a light into a normal eye in a semi-dark room, its pupil will constrict (direct response), and so will the other pupil automatically (consensual response).

If the optic nerve is completely destroyed, there will be no direct or consensual response (total afferent pupil defect).

If the optic nerve is partly destroyed *(for example, 90%)*, the pupil will constrict slowly when the light shines in it (partial afferent pupil defect), and the consensual response will be present.

(iii) THE SWINGING TORCH TEST is a useful test for early asymmetrical optic nerve damage, and does not need an ophthalmoscope. It is theoretically difficult, but is easy in practice. In a semi-dark room shine a light into the good eye, and then swing it across into the bad eye (the eye with reduced vision).

As the light shines in the good eye, the pupil of the bad eye will constrict. As you swing the light quickly across to the bad eye, its pupil, which was previously constricted, will now dilate. This indicates a relative afferent pupil defect, early optic nerve damage, and a difference in function between the 2 optic nerves.

VIEWS OF OPTIC DISCS (LEFT EYE)

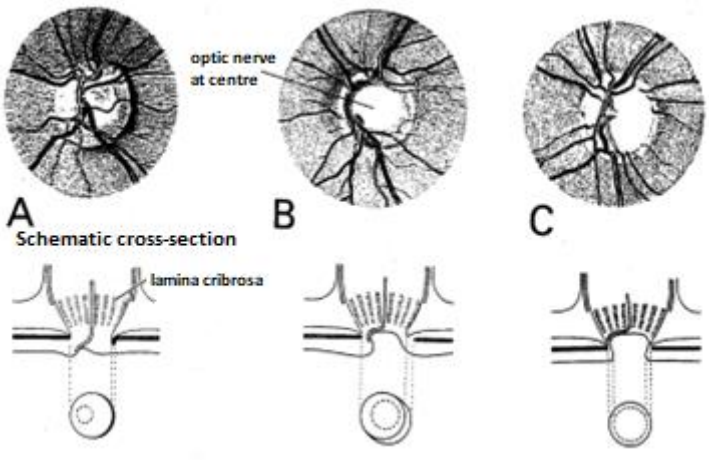


Fig. 28-11 SOME OPTIC DISCS OF THE LEFT EYE.
A, normal optic disc with a moderately sized cup. The *lamina cribrosa* is a mesh-like structure of collagen in the sclera wall which maintains the pressure gradient. B, another normal optic disc. This has a large physiological cup and a temporal scleral crescent. C, optic disc of a patient with gross 'chronic' open-angle glaucomatous cupping.
After Parr J. Introduction to Ophthalmology. OUP 2nd ed 1982, with kind permission.

The practical test is to swing the torch from one pupil to the other and back again in a semi-dark room. If one pupil consistently dilates as light shines on it, that eye has a reduced pupil response, relative to the better eye. You should investigate this for optic nerve disease, perhaps POAG.

RISK FACTORS FOR POAG:
(1) Age >40yrs.
(2) A +ve family history in first-degree relatives.
(3) A vertical cup/disc ratio >½.
(4) An IOP of >28mm Hg (<2 with a 5·5g weight).

MANAGEMENT will preserve what sight exists, but will not improve it.

If there is any sight left, perform a trabeculectomy. Even when sight is as poor as CF1m (28.1), there may still be some benefit from treatment.

Medical treatment can lower the IOP, but it has to be constant, consistent and continue for life, which is usually impracticable, so that immediate surgery is better. Start with pilocarpine 4% qds or timolol 0·5% bd (expensive), and if this fails to maintain the IOP <20mm Hg, add acetazolamide 250mg qds orally for short periods.
N.B. Several side-effects may occur with continued use.

To treat glaucoma effectively, measure the IOP and monitor the visual fields regularly. Do this in a specialist clinic.

Surgical treatment is a trabeculectomy which removes a piece of the filter (the trabecular meshwork), and so allows the intraocular fluid to drain under the conjunctiva; this increases drainage and reduces the IOP. The operation has an immediate success rate of >80%, and is the recommended treatment for most patients with POAG, but it does not have a permanent effect. You should follow up these patients long-term; they may need repeat surgery.

(b) Angle closure glaucoma (ACG, acute glaucoma) usually occurs >35yrs, in women more often than men, with an abnormally narrow angle between the iris and the cornea. If this angle should happen to close a little more than usual, it causes an abrupt rise in the IOP with resulting unilateral episodic attacks of pain, misty vision, and rainbow-coloured haloes round lights. Between attacks the eye is normal. Sooner or later, an episode of raised IOP does not resolve, causing classical acute congestive glaucoma (28.3). Acute glaucoma is relatively uncommon, and is rare in Africa. Its incidence is highest in Inuits and Mongolian peoples, and in Southeast Asia. Most acute glaucoma is usually this type, but secondary glaucoma (see below) may occasionally present acutely.

IF AN OLDER PERSON COMPLAINS OF POOR VISION, CHECK FOR GLAUCOMA

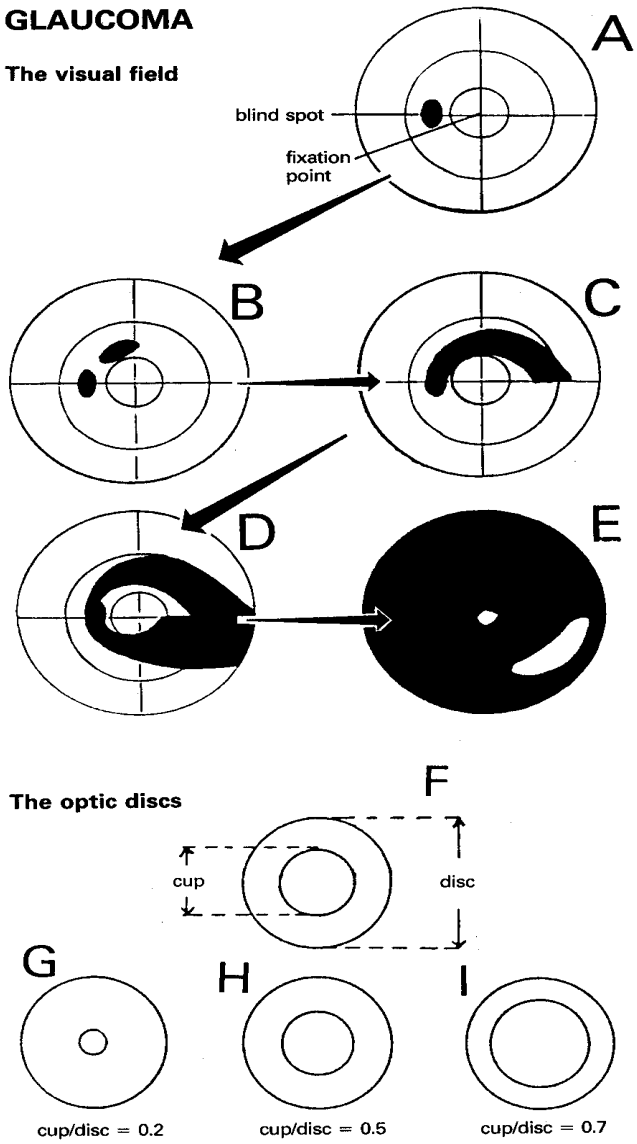


Fig. 28-12 GLAUCOMA.
A, normal visual field with its normal blind spot (28-1C). B, paracentral scotoma (blind area) in early glaucoma. C, arcuate scotoma in early glaucoma. D, ring scotoma in early glaucoma. E, complete loss of visual field apart from a small central island, and a larger temporal island, in advanced glaucoma. F, schematic representation of the cup/disc ratio. G, normal optic disc with its physiological cup. H, cup/disc ratio of 0·5, the borderline of normality. I, pathological cupping of the optic disc due to glaucoma with a cup/disc ratio of 0·7.
Kindly contributed by Allen Foster.

DIAGNOSIS
Presentation is with an 'acute red eye' at any age (one of its rarer causes, 28.3), severe unilateral headache in and around the eye, and sudden profound loss of vision. Vision is reduced, the eye is red, the cornea is hazy from oedema, the anterior chamber is shallow, the pupil is usually dilated, and IOP usually >40mm Hg.
CAUTION! Blindness is inevitable unless you treat this quickly.

TREATMENT
Admit the patient as an emergency. If referral is delayed, start treatment before operating. Use analgesic to ease the pain, and treat both the eyes. Aim to:

(1) Lower the IOP by increasing the drainage of aqueous. Use acetazolamide 500mg orally followed by 250mg qds, as soon as the nausea has subsided. Apply timolol 0·5% immediately and repeat this twice.
(2) Keep the pupils constricted. This will keep the periphery of the iris away from the angle of the eye, where the aqueous flows out, and so help it to drain. Treat both eyes with drops of pilocarpine 1% every 15mins, for 2h. If this makes the pupil constrict, the angle will be opened.
(3) You can also lower the IOP by increasing the tonicity of the blood. Use 50ml of flavoured glycerine by mouth if nausea has settled. Alternatively, use 200ml of 20% mannitol IV over 20mins.
When medical treatment has reduced the IOP to normal, arrange a peripheral iridectomy soon, to prevent a future attack; recommend a prophylactic iridectomy on the other side also.

CAUTION!
(1) Blindness can occur in 12h, so treatment is urgent.
(2) Rainbow-coloured haloes round lights, and misty vision, are important prodromal signs, and need urgent investigation and treatment.
(3) *Atropine can precipitate an attack in a patient with a shallow angle!*

The dangers of atropine in glaucoma result from its effect in dilating the pupil:
(1) The iris is kept away from the lens, which prevents adhesions (synechiae) forming between them, which is valuable in iritis.
(2) The iris is crowded into the angle of the anterior chamber, where it impedes the drainage of aqueous. This is never a desirable effect, but it does not matter in a normal eye or in iritis; *it can however turn an eye blind if drainage is already impaired by glaucoma!*
So, use atropine in iritis, but *not in glaucoma!*

TRABECULECTOMY & PERIPHERAL IRIDECTOMY (GRADE 2.4)
It helps to have a microscope to do this operation.

Insert the eyelid retractor. Secure the eye by means of a traction suture of 4/0 silk through the belly of the *superior rectus* muscle. Select the site of the incision cranially (usually at the 10-11o'clock position) by injecting 0·2ml saline subconjunctivally. Incise the conjunctiva at the limbus (28-19B) for 5mm and undermine it to free it from Tenon's capsule (28-1C). Control bleeding with a fine cautery. Mark a rectangular sclera flap 5x5mm of *half the scleral thickness* hinged at the limbus, dissecting this forward till you see the transparent cornea.

Make an incision through the cornea into the anterior chamber behind the hinge of the sclera flap and cut out a 4x2mm block of tissue containing the trabeculum and Schlemm's canal (28-1B). Then grasp the peripheral iris with forceps, prolapse it and excise it to make a peripheral iridectomy.
N.B. If you incise too much sclera, you may reach the ciliary body and cause a choroidal detachment.

Return the sclera flap on its hinge to its bed, and close the wound, and then close the conjunctiva at each corner both with 9/0 or 10/0 nylon.

(c) Secondary glaucoma
This complicates:
(1) trauma, including hyphaema (blood in the anterior chamber).
(2) swollen cataract (see below).
(3) some cases of iritis.
Treat the primary condition, and try to arrange referral: in the meantime, treat with acetazolamide, mannitol, or glycerol as above.

(d) Difficulties with glaucoma
If there is an acute red painful eye, a fixed dilated white pupil, a shallow anterior chamber and a hazy cornea, this is a SWOLLEN CATARACT causing secondary glaucoma. Use acetazolamide 500mg immediately followed by 250mg qds, and arrange cataract extraction.

If there is loss of vision from chronic use of steroid drops, suspect STEROID GLAUCOMA. Topical steroids cause a genetically determined rise in IOP in 30% of people. This is sometimes severe enough to cause glaucoma, exactly like POAG. Stop the steroids.

If a child has big eyes, which may be associated with photophobia, blepharospasm, and tears, suspect CONGENITAL GLAUCOMA (buphthalmos, ox-eye), due to maldevelopment of the angle of the anterior chamber. The sclera of a child are soft, so that the eyes enlarge when the IOP rises.

Other signs are: an increased IOP, a corneal haze (variable), sluggish reaction of the pupils to light, and enlargement of the cornea (>12mm), or of the whole globe. Try to arrange an urgent incision of the trabecular meshwork to allow flow of aqueous.

28.7 Onchocerciasis (River blindness)

Onchocerciasis is a parasitic infection of the skin and eyes caused by *Onchocerca volvulus*, which used to be endemic in parts of West Africa, with foci in East Africa and Latin America. WHO-led programmes have virtually eliminated this disease except in West and Central Africa.

In endemic areas 20% of the population are infected by a worm transmitted by the blackfly. Microfilariae invade all parts of the eye: the cornea (keratitis), the anterior chamber (iritis), the retina (chorioretinitis), and the optic nerve (optic neuritis). Blindness and irreversible eye lesions are most often found in people ≥30yrs.

A patient from an endemic area complains of itching, with or without a rash. There may be skin nodules on the hip or shoulder (in Africa) or scalp (in S. America), night blindness (enquire about this), gradual loss of vision in both the eyes, and sometimes tears and photophobia. Look

for microfilariae in snips from the skin. There may also be huge adenolymphocoeles in the groin (18-5).

(a) Cornea. There is a sclerosing keratitis with opacification of the lower third of the cornea. Tongues of opacification invade the cornea from the 3 and 9o'clock positions, or from anywhere in the lower ½, where they may form an apron across the cornea. If this is not treated, opacification slowly advances upwards over the pupil, until all that may be left is a clear area at 12o'clock. Slit lamp microscopy shows 0·5mm linear and fluffy opacities at all levels in the stroma, and minute wriggling microfilariae in the anterior chamber.

(b) Iris. The final stage is a small, non-reactive, down-drawn, pear-shaped pupil. Earlier stages are a loss of pigment in the margin of the pupil, exudation in front of, across, or behind it, posterior adhesions which turn it inwards, and small keratic precipitates. A gelatinous exudate sometimes drags the lower margin of the pupil down and everts it. Posterior synechiae and peripheral anterior synechiae lead to secondary glaucoma.

(c) Retina and optic discs
Look for:
(1) Diffuse white areas and black pigmented ones most marked temporal to the macula (the Ridley fundus).
(2) Optic atrophy, with sheathing of the vessels close to the nerve.
(3) Microfilariae wriggling in the anterior chamber (after putting the head between the knees for at least 1min).

(d) Treatment. Use ivermectin once yearly as a single dose according to weight, for 15yrs (the lifespan of an adult worm, which is not killed by the drug):

15-25kg	3mg
26-44kg	6mg
45-64kg	9mg
65-84kg	12mg

If there is also *loiasis* (28.18) with high quantities of microfilariae, add prednisolone 20mg od 3 days before starting treatment and till 3 days afterwards to prevent severe encephalopathy.

If there is an onchocercal iritis, dilate the pupil with atropine and use topical steroids (28.5). If secondary glaucoma (28.6) develops, arrange surgery.

28.8 Refractive errors: difficulty reading and presbyopia

In terms of comfort, increased efficiency and the number of people who benefit, the prescription of glasses is among the most valuable procedures in medicine. So do what you can to supply them. You may be able to get cheap self-tuning glasses (which contain an adjustable amount of fluid between the clear plastic making the lenses). However, these are difficult to maintain and require skill to use.

The refractive errors are:
(1) Myopia (short sight).
(2) Hypermetropia (long sight).
(3) Astigmatism (the refractive mechanism is aspherical).
(4) Presbyopia (common long-sightedness of old age).
(5) Aphakia ('no lens') after old-style simple cataract extraction.

Presbyopia is part of the normal process of ageing: it appears earlier in the tropics than in higher latitudes, and is easily diagnosed and treated. It is responsible for 85% of the need for glasses.

Whereas a myopic child has to be specially fitted, because the eyes may not be the same, a presbyope can, if necessary, be left to choose the glasses which best suit him from a pile of second-hand ones. Astigmatism is more difficult to correct, but it is usually so mild that it needs no correction: its correction is often overemphasized.

N.B. The visual acuity of all patients with refractive errors improves when they look through a pin-hole, which uses only their central vision. This is the basis of the pin-hole test (28.1).

If there is astigmatism, due to irregularly curved lenses, the vision is blurred.

If there is hypermetropia (long-sightedness), a patient has difficulty reading or seeing things close at hand, *e.g.* threading a needle.

If there is myopia (short-sightedness), a patient cannot see clearly at a distance, but near objects are clear.

Unless you can perform retinoscopy, test each eye alone by trial and error with spherical lenses. Use the charts (28-2).

For long-sightedness, start with +1 dioptre, the usual requirement at 45yrs, with +0·5 dioptre increments for each 5yrs. The smallest number that gives the best acuity is the prescription for the glasses needed.

For short-sightedness, start at -0·5 dioptre and continue adding -0·5 increments. Again the smallest number that gives the best acuity is the prescription needed.

Aphakia after old-style cataract extraction requires +10, 11 or 12 dioptre lenses for distance, and +13, 14 or 15 lenses for reading. These are easily broken, rendering the patient blind again!

Check that the eye is not red, and that there are no other serious eye problems.

A child with reduced visual acuity due to refractive errors needs glasses, whereas an adult probably does not. A child <7yrs with a squint needs surgery (28.9).

Remember, glasses for reading and other close work are not useful for seeing at a distance.

28.9 Neuromuscular eye disease: squints, amblyopia, and diplopia (double vision)

If the eyes don't look in the same direction, this is known as a squint (strabismus). Squints are common, and are usually accepted with resignation. Although treating a squint needs relatively simple technology, it is time-consuming and requires skill.

Two images are generated. This causes confusion, and to avoid this, the brain suppresses one image. If this suppression continues for long enough, vision disappears in that eye. This is called amblyopia, which is a reduction in vision due to lack of use of an apparently normal eye. If amblyopia is uncorrected in a child by 7yrs, it becomes permanent. So try to diagnose a squint and arrange surgery before this age.

CAUTION! Never occlude the eye of a child <7yrs for several days, because this may cause amblyopia.

DIAGNOSIS

The corneal light reflex: Shine a pen torch directly in front of you, and ask the patient to look at it. If each of the eyes is properly fixing the torch, its reflection from the corneal mirrors will be the same, and more or less central on each cornea. Are the reflections from your torch equally centred on the pupils?

The cover test: Ask the patient to look straight ahead at some target in the distance. Cover the left eye with a piece of paper. If the right eye moves, in or out, to fix on the distant target, it was previously squinting. If it does not move, it was looking straight at the target.

Now put the paper in front of the right eye. If the left eye moves as you remove the paper, it was previously squinting in or out. If it does not move, there is no manifest squint, and both eyes look straight.

MANAGEMENT is limited.

If an adult has a squint and double vision, this suggests a serious recent disease of the extraocular muscles or their nerves, such as diabetic neuropathy, myasthenia gravis, or raised intracranial pressure. He needs a full medical and neurological examination.

If an adult has a squint and no double vision, it may be the cause of reduced vision in the squinting eye (amblyopia). There is no treatment at this age.

If a child <7yrs presents with a squint:

(1) Dilate both the pupils, and use an ophthalmoscope to make sure that the squint is not due to a retinoblastoma in one of the eyes (an uncommon cause, 28.16: look for a yellowish mass on the retina).

(2) If the retinae are normal, try to assess the visual acuity in both eyes. This is relatively easy at >3yrs, but is difficult at younger ages (28.1).

If there is reduced vision in either eye, or a definite squint at >7yrs, there may be need for glasses, and perhaps surgery on the extraocular muscles.

Correct any refractive error and occlude the eye that is most used. Occlude it for ½-2h/day during close work, reading, or drawing. The duration of treatment depends on the duration of the amblyopia. If treatment is prompt, 6-8wks of intermittent occlusion treatment may be enough. If it is delayed, you may need to continue for 1yr.

AFTER THE AGE OF 6 MONTHS
DON'T IGNORE A SQUINT!

28.10 Eyelid & nasolacrimal disease

Diseases of the eyelids and nasolacrimal system include tumours, deformities of the lids, and watering (epiphora). Globally, the most important disease of the lids is trachoma, which scars the lids, and causes them to turn inwards (entropion, 28.13).

The commonest and usually the most harmless disease of the lid is a stye. The lid is involved in *herpes zoster* ophthalmicus, where the virus affects the VIIth cranial nerve, often associated with HIV infection. Cutaneous *leishmaniasis* causes the lid to scar and produce an everted eyelid (ectropion). *Loiasis* is found in equatorial rainforests, where the vigorously mobile *loa loa* worm is found under the conjunctiva, causing an acute inflammatory swelling.

(a) The lids

If a patient has a red swelling on the lid margin, with an eyelash coming out of it, this is a stye (hordeolum). It is a staphylococcal infection of an eyelash follicle. Pull the eyelash out of the swelling, using an analgesic. Only use antibiotics in recurrent styes, or if infection is spreading beyond the lid (cavernous sinus thrombosis is a rare complication, 6.6). Warm soaks (28.3) are useful.

If there is a swelling in either lid, some distance from its margin, pointing towards its conjunctival surface, it is probably a tarsal cyst (chalazion, Meibomian cyst). Avoid an external scar by incising the conjunctiva wherever the cyst is about to burst (28.12).

If a few lashes turn in on the eye (trichiasis), remove them with electrolysis.

If most of the lashes or the margin of the lid are turned in (entropion), perform surgery (28.13).

If the upper eyelid, eyebrow and forehead are involved in a vesicular eruption with an abrupt demarcation in the midline, this is *herpes zoster*. Acyclovir given early will help. Later complications can result in ectropion, keratitis, uveitis and secondary glaucoma.

If there is an acute attack of localized oedema of the orbit in an endemic area, consider *loiasis*. If you can see the worm under the conjunctiva, instil lidocaine drops, make a small incision and remove the worm with forceps. Ivermectin annually is useful as a prophylactic.

If the lids don't close properly, as a result of a VIIth cranial nerve palsy (from herpes, iatrogenic injury in parotid surgery, leprosy, or parotid malignancy), the cornea will remain exposed, especially at night, and exposure keratitis will result. To avoid this, do a tarsorrhaphy unless you can supply artificial tears.

If the margin of either lid is everted (ectropion), usually as the result of scarring, or longstanding palsy, release of lid contracture with skin grafting of the defect is necessary.

TARSORRHAPHY

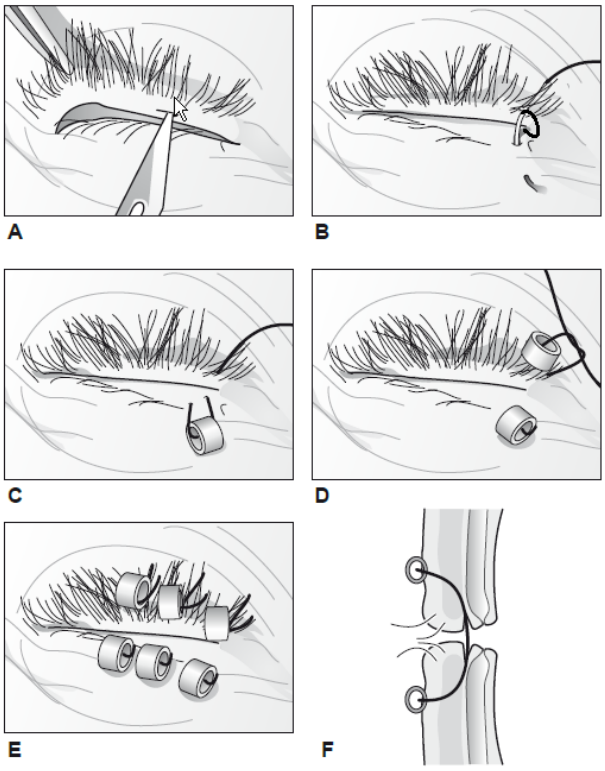


Fig. 28-13 TARSORRHAPHY. A, lid margin incision on upper lid. Make the same incision on the lower lid. B, the suture passes from above the eyelash through the two incisions and out below the lower eyelash. C, pass the suture through a rubber ring, and then back through the incision, *not on the undersurface*. D, lock the suture on 2 protecting rings. E, completed tarsorrhaphy. F, cross-section of position of suture.
After Galbraith JEK. Basic Eye Surgery Churchill Livingstone 1979 p.44 Figs 5.29-33.

(b) Tarsorrhaphy (GRADE 2.2)

INDICATIONS
Eyelids which permanently fail to cover the cornea properly, especially when the cornea is anaesthetic.
N.B. This is usually a temporary measure.

METHOD
Stretch the lid by grasping one end of the lid margin and make an incision 2mm deep along the middle of the upper and lower margins, just beyond the eyelash follicles (28-13A). Then pass a 4/0 silk suture through the skin above the upper eyelash line and out through the incision, and similarly in through the lower lid incision and out below the lower eyelash line (28-13B).

Turn the suture back, through 3mm rubber rings to take the tension, through the skin (28-13C) and tie it so that the lids are well approximated (28-13D).

You need as many sutures as required to get adequate eyelid closure; this may only be necessary on the lateral aspect, but *don't make the tarsorrhaphy too small*. Put chloramphenicol eye ointment or drops qds and make sure no eyelashes project back inside the suture line.

CONTRACTURES OF THE LIDS

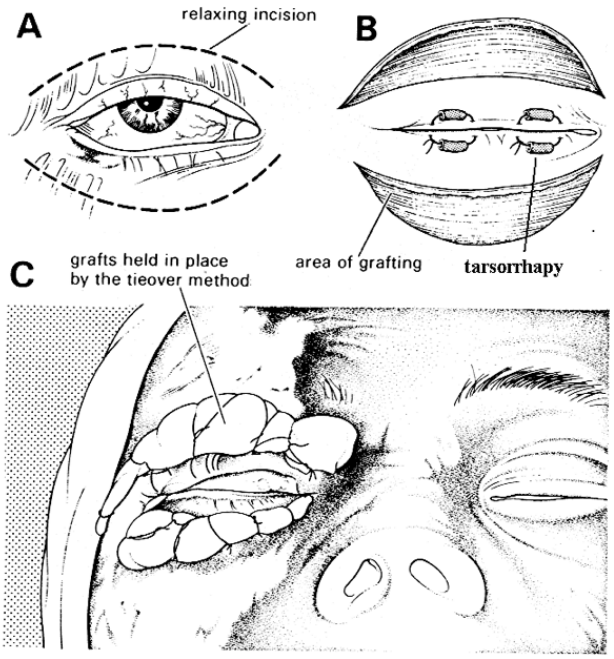


Fig. 28-14 CONTRACTURES OF THE EYELIDS.
A, retraction of scar tissue everting the eyelid and exposing the cornea. B, make relaxing incisions and get ready for grafting. C, hold grafts in place by the tie-over method.
Kindly contributed by Randolph Whitfield II.

(c) Release of lid contractures (GRADE 2.2)

INDICATIONS
Contractures starting to expose the eyelids and expose the cornea. *You should have done a tarsorrhaphy already.*

METHOD
Put stay sutures through the lids (28-8F), so that you can move them up or down as necessary. Make the relaxation incisions (28-14A).

When you have prepared a satisfactory bed for the graft, and controlled bleeding, stretch it, apply a piece of split

skin graft, and hold it in place with tie-over sutures using 4/0 monofilament (28-14C). If the graft contracts after 12wks, repeat the release and apply another graft.

Don't worry too much about what the patient's eye looks like at this stage. What matters is that the cornea should not be exposed.

DON'T DELAY GRAFTING THE LIDS

CAUTION! Stretch the lid first so that there will be some slack tissue when it contracts later. The thinner the graft, the more the shrinkage. If you are skilled, apply a full thickness graft. Primary skin grafting will not prevent ectropion, and you may need 2 or 3 operations to insert enough skin.

(d) The nasolacrimal apparatus

If something interferes with the drainage of an adult's tears, the eye waters (epiphora) even if there is no local irritation. Epiphora can occasionally be so severe that it needs surgery (a dacryocysto-rhinostomy).

If a mother brings you her young child saying that he has had a watering eye since birth, this is congenital atresia of the nasolacrimal duct. It will probably resolve spontaneously by the age of 18 months. Reassure her, and use a topical antibiotic if conjunctivitis develops.

If the eye is still watering at 2yrs, the nasolacrimal duct needs probing and syringing.

If there is a tender swelling between the eye and the side of the nose, this is probably acute dacryocystitis (an abscess in the tear sac). Use IV gentamicin, an analgesic, warm soaks, and incise the skin of the lower eyelid over the lacrimal sac (6.6).

28.11 Proptosis (Exophthalmos)

If there is a space-occupying lesion in the orbit, it pushes the eye forwards. Proptosis is always serious, and it can be difficult to diagnose, but is uncommon. Some of its causes need medication (orbital cellulitis: IV antibiotics and Burkitt's lymphoma: cytotoxics).

Most patients who need surgery are either going to die from malignant tumours anyway, no matter what is done, or they have slow-growing benign tumours, which you have time to try to refer. So your ability to help a patient with proptosis is limited; but you should try to make a diagnosis.

Proptosis can occur slowly over years, or rapidly over days. Its causes vary geographically, and with the age of the patient. The more common causes are listed first below; the later ones are mostly very rare.

N.B. Thyrotoxicosis may cause unilateral proptosis, so check for weight loss, and tachycardia.

An *adult* may have:
(1) A retrobulbar haematoma following an injury (common). This is only an incident in a head injury, and the diagnosis is obvious.
(2) A mucocoele of the frontal sinus (the commonest cause), due to an infection followed by an obstruction, which prevents the sinus draining into the nose.
(3) Orbital cellulitis, or an orbital abscess, usually following frontal or ethmoid sinusitis (6.6), or occasionally trauma.
(4) A pseudotumour of the orbit due to a granuloma of unknown cause.
(5) An epidermoid or dermoid cyst, which may be of the 'dumb-bell' type, and extend into the anterior cranial fossa. *Don't operate on these*, unless you are skilled enough to dissect widely, and have made an accurate diagnosis.
(6) A lacrimal pleomorphic adenoma, palpable at the inferior orbital rim.
(7) A haemangioma; you may be able to empty a haemangioma temporarily by pressing it back into the orbit.
(8) A hydatid cyst, if this disease is endemic (15.10).
(9) A cavernous sinus thrombosis (6.6)
(10) A carotico-cavernous fistula, where there is an audible noise in the head and a bruit heard over the eye; this may follow trauma but is usually spontaneous.
(11) A metastasis.
(12) A malignant melanoma (34.6).
(13) A conjunctival carcinoma.
(14) The hyperophthalmopathic form of thyrotoxicosis (Graves's disease, 25.2).
(15) A meningioma of the sphenoid.

A *child* may have:
(1) A retinoblastoma in the first 5 years of life (28.16).
(2) Acute ethmoiditis, commonly around 2yrs.
(3) Burkitt's lymphoma (17.6), usually associated with a jaw tumour.
(4) A rhabdomyosarcoma (34.15).
(5) Some other kind of lymphoma (17.6).
(6) A neuroblastoma.
(7) A metastasis.

(a) History. Long history? (benign lesion). Short history? (malignant lesion or acute infection). Acute onset with pain? (infection).

(b) Examination. Sit the patient down, stand behind him, look down on the eyes from above, and observe the relative positions of the globes. This will help to distinguish pseudoproptosis, due to the relative widening of one palpebral fissure.

(c) Radiographs may demonstrate:
(1) Erosion of orbital bones.
(2) Sclerosis of orbital bones (typical of a meningioma).
(3) Calcification (sometimes in a retinoblastoma).

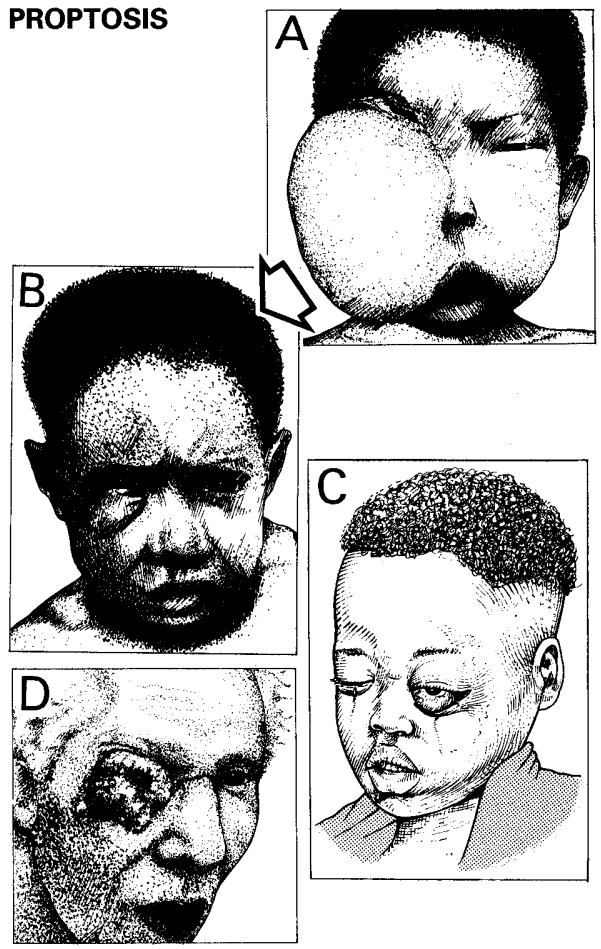


Fig. 28-15 PROPTOSIS.
A,B, Burkitt's lymphoma, before and after treatment. C, proptosis, cause not yet established. D, carcinoma of the maxillary antrum extending into the orbit.
A,B,D, Parsons GA, Berg D, Proptosis and orbitotomy in Papua New Guinea, Tropical Doctor 1977;7(2):129-33. C, kindly contributed by Edward Kasili.

Hold a ruler horizontally, and measure the position of each cornea from the midline. If there are 2 protruding globes, and they are both equidistant from the midline, this is probably thyrotoxicosis (the most likely cause of bilateral proptosis). If they are not equidistant, one globe has probably been pushed out of place by an orbital mass. Examine the fundi for papilloedema and optic atrophy. Search for signs of a primary malignant tumour.

CAUTION! Don't confuse proptosis with a staphyloma due to a neglected corneal ulcer (28.3). The normal intraocular pressure has caused the previously weakened cornea to bulge, in a manner which you can mistake for a tumour. The globe however remains in its normal position.

(d) Biopsy may be practical.
If a tumour is palpable externally, take a biopsy, but if there is a swelling of the upper lateral quadrant of the orbit, pushing the eye downwards and inwards, which has grown slowly over many months or years, this is probably a lacrimal pleomorphic adenoma, *which you should not biopsy for fear of spreading the tumour tissue.*
Don't be deceived by the small mass of tumour palpable externally: most of it will be inside the orbit behind the eye. It needs removing through a lateral orbitotomy.

(e) Diagnosis and management
If the proptosis arose acutely, and the lids are red and swollen, perhaps with a fever and tachycardia, this is orbital cellulitis, or an orbital abscess. Use IV antibiotics. If there is an abscess, drain it (6.6).

If a child c.2yrs has sudden unilateral proptosis, with swollen lids and conjunctiva, fever and tachycardia, suspect acute ethmoiditis. Use IV antibiotics.

If there is an acute pulsating proptosis, which may be unilateral initially, but soon becomes bilateral, with engorgement of the veins, and total inability to move the eye, with severe prostration or loss of consciousness, suspect cavernous sinus thrombosis (6.6).

If there is a swelling which has enlarged slowly (weeks or months) in the superior nasal quadrant of the orbit, pushing the eye downwards and outwards, this is probably a mucocoele of the frontal sinus (common). By an approach between the periosteum and the frontal bone, keeping outside the orbit, enter the sinus and drain the mucopus. Place a drain from the sinus into the nose. Suture the skin in layers. Remove the nasal drain at 6wks.

If the patient is between 15-35yrs, and the proptosis occurred over several weeks or months, suspect idiopathic orbital inflammation. The diagnosis is largely made by excluding other causes. It will respond well to prednisolone 60mg od for 1wk, reducing slowly to 5mg od by the 4th wk. Maintain 5mg od for several months, or it will recur.

If the proptosis of thyrotoxicosis does not respond to medical or surgical treatment (25.2), try high-dose systemic steroids. If this fails, try to arrange surgical orbital decompression by removal of fat or bone.

(f) Treatment for the exposed cornea
Examine the cornea to make sure that it is not ulcerated. Apply antibiotic eye ointment qds, and especially at night. If necessary, protect it by tarsorrhaphy (28-13). *Padding can be dangerous*, because the pad may abrade and ulcerate the cornea.

28.12 **Tarsal** (Meibomian) **cysts** (Chalazions)

Meibomian glands secrete oil at each blinking of the eyelid; this prevents evaporation of the fluid film on the eye. Cysts may form in these glands on the conjunctival side of the tarsus (or eyelid). They present as a swelling in either lid, which may become chronically or acutely infected. Small asymptomatic ones need no treatment, and may resolve spontaneously. Incise an acute infection and curette a chronic one.

These cysts are common everywhere, so that treating them is a common outpatient eye operation. Sometimes, they present as granulomas.

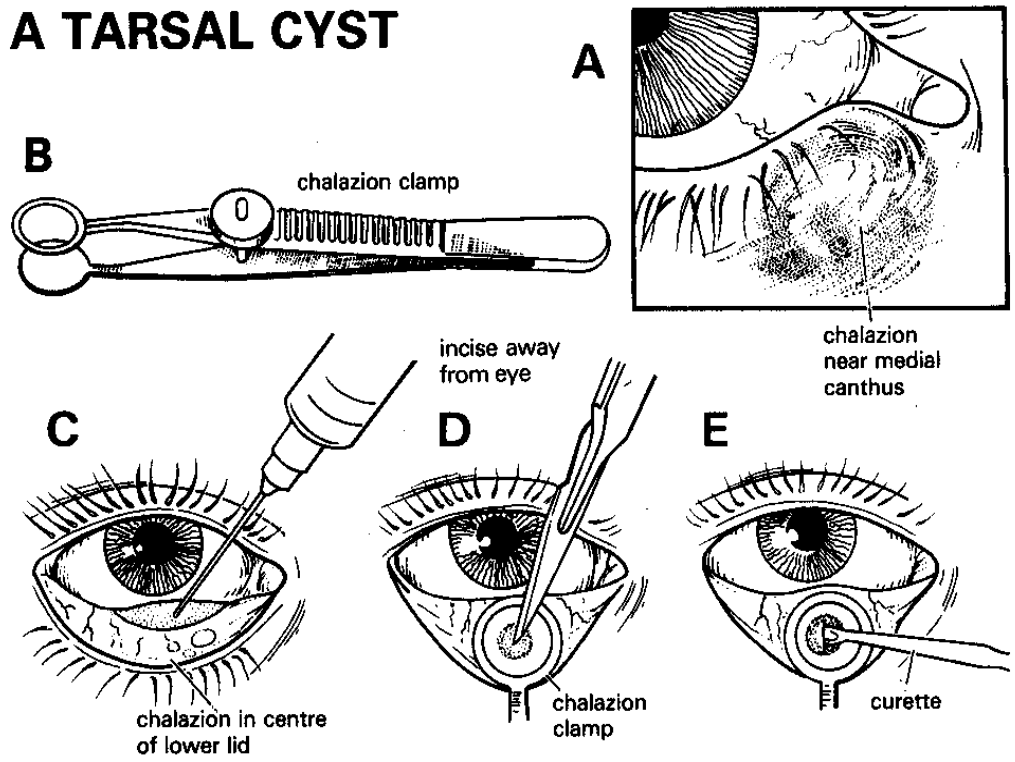


Fig. 28-16 CURETTING A TARSAL CYST.
A, chalazion close to the medial canthus. B, chalazion forceps. C, introducing LA. D, chalazion clamp in place ready to incise a chalazion in the centre of the lower lid. E, curetting the chalazion.

(a) Anaesthesia
Anaesthetize the conjunctiva with drops of lidocaine 4%, or amethocaine hydrochloride 1%. Infiltrate the lid with lidocaine and adrenaline around the chalazion. Insert the needle at the upper margin of the upper tarsus, and the lower margin of the lower tarsus. Carry it forwards to the lid margin, on either side of the chalazion.

EQUIPMENT. *Chalazion clamp, # 11 scalpel blade and curette.*

(b) Curettage (GRADE 1.2)
Evert the lid slightly. Put the chalazion clamp over the cyst, so that the solid blade lies on the skin of the eyelid, and the ring lies on the conjunctiva over the cyst.

Close it so that it holds the lid and the cyst. Insert the tip of a #11 blade, so that it cuts away from the eye. Always make the incision perpendicular to the lid margin so as to avoid cutting the *levator* muscle. Swab its contents clean.

CAUTION! Take care to curette away any pockets of granulation tissue, which may be hidden by a flap of conjunctiva, or have herniated themselves through the tarsal plate into the *orbicularis* muscle. *If you don't do this, it may recur*.

Remove the clamp and pinch the lid until it stops bleeding. If this is troublesome, wash it with warm saline. Place chloramphenicol ointment in the conjunctival sac tds for 1wk.

If the material you incise is hard, and not gelatinous, suspect a carcinoma. Send it for histological examination.

28.13 Entropion

(a) Trachoma is the commonest eye infection in the tropics and also amongst Aborigines in Australia, and in its blinding hyperendemic form is worldwide the 2nd commonest single cause of blindness and impaired vision (cataract is the first). It is a *chlamydial* infection, which spreads from the eyes of one person to another, especially among children, in the poorest and most disadvantaged communities, particularly those in the Middle East and Africa.

Trachoma is a chronic follicular conjunctivitis; it scars the conjunctiva of the eyelids and the cornea, and goes through 4 stages (28.3). A single dose of 20mg/kg azithromycin (or 500mg bd erythromycin for 1wk in pregnancy) orally is effective in arresting the disease.

(1) STAGE I. There is a mildly red, watery eye due to bilateral conjunctivitis, especially of the upper lids, but without any distinguishing features.

(2) STAGE II. Under the upper lid there are dilated blood vessels and hyperaemic, oedematous epithelial tissue (papillae). There are also yellow-grey swellings (follicles). Look at the corneoscleral junction with a loupe.

If the edge of the cornea looks mildly grey, owing to an arcuate (crescent-shaped) grey infiltration, and blood vessels go beyond the grey area into the cornea, there is pannus (meaning a curtain). This starts at the 12o'clock position, and extends to 9 and 3o'clock.

Follicles and pannus indicate Stage II trachoma. Follicles are not diagnostic, but pannus is.

(3) STAGE III. The follicles in the lids become coarser and pannus spreads, sometimes across the pupillary area of the cornea. Scarring makes the margin of the upper lid irregular, and turns the upper tarsus inwards (entropion), taking the lashes with it, so that they scratch the cornea during every blink (trichiasis, 28-18C). This causes recurrent attacks of keratitis, which eventually results with a corneal opacity causing blindness.

(4) STAGE IV. Fibrous tissue replaces the follicles in the lids. This is the stage of scarred, or healed trachoma. The cornea is grey and scarred, the vision severely impaired, and the lids are deformed.

If you work in an endemic area, you are likely to have many patients with entropion, so learn how to correct their eyelids yourself, and if necessary train an assistant to do this. The operation is always worth doing, even if the lids are severely scarred: sight may recover surprisingly.

Several operations are possible:
(1) Splitting the eyelid margin
(2) Radical eyelash excision
(3) Tarsal eversion.

If only a few lashes are turning in, try removing these with an electrolysis needle under LA. Epilation (pulling out the lashes) is ineffective as they will grow back. *Don't cut the eyelashes short* because this makes them sharp and even more irritant.

(b) Entropion correction

INDICATIONS
Trachoma which has distorted the upper tarsus, so that it has curled inwards and made the lashes scratch the globe. Operate as soon as possible after entropion occurs.

You can operate on both the eyes at the same time, but this will require admission for 3 days, to allow the oedema of the eyelids to subside. Absorbable sutures allow discharge without the patient needing to return.

EQUIPMENT. *An eye set, a scalpel with #15 blade, 4/0 absorbable suture. A cautery will help to control bleeding.*

ANAESTHESIA
Anaesthetize the upper lids through the skin with 1ml of 2% lidocaine with adrenaline. Anaesthetize the conjunctiva with 2 drops of amethocaine, or lidocaine.

EYELID MARGIN SPLITTING FOR TRICHIASIS (GRADE 1.3)

Split the eyelid into two parts along a 'grey' line defined by the *orbicularis oculi* muscle: just inside are the openings of the Meibomian glands and just outside are the eyelash roots.

The divided eyelid will then have an inner part containing the conjunctiva and tarsal plate, and an outer part containing the *orbicularis oculi* muscle, the inverted eyelashes and their roots. Rotate this outer part outwards and fix it by an everting suture over a gauze swab.

RADICAL EYELASH EXCISION

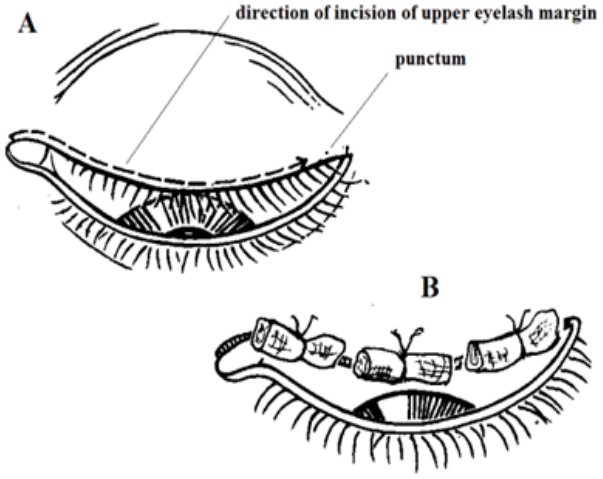


Fig. 28-17 RADICAL EYELASH EXCISION.
A, direction of incision. B, excision complete.

RADICAL EYELASH EXCISION FOR ENTROPION (Malcolm Phillips) (GRADE 2.3)

Removing the eyelashes completely will mean they cannot re-grow and scratch the cornea. This results in little cosmetic disability, especially with darker skin, and the relief that follows is dramatic.

If you are a right-handed operator, start with the right eye. Use a scalpel to incise the margin of the upper lid, at the lateral end of the lashes, to a depth of 3mm (28-17A). Using small sharp scissors, remove the margin of the lid bearing the roots of the lashes. Cut towards the medial end and preserve the punctum. Evert the lid as you do this, by pressing it with a swab. Then repeat the procedure on the left eye.

Control the considerable bleeding that will result by suturing the conjunctiva to the skin of the eyelid with 3/0 absorbable sutures on a cutting needle. Insert about 5 sutures, 5mm apart, knotting them, and use the same suture to hold little rolls of gauze. Apply an eyepad for 24h. Remove the part of the sutures holding the gauze rolls after 3 days.

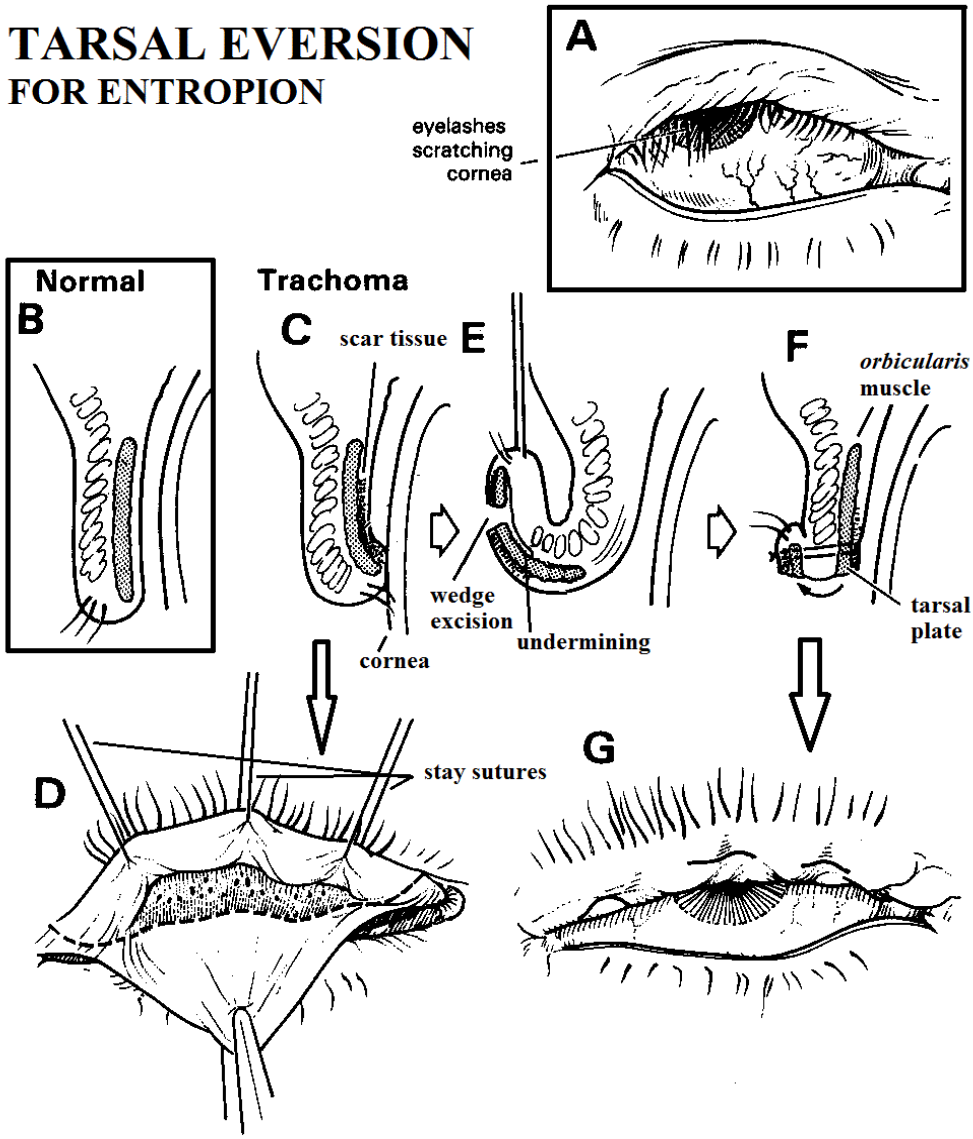


Fig. 28-18 EVERTING THE TARSUS FOR LATE TRACHOMA. A, inwardly turned upper lid, with the lashes scratching the cornea. B, normal eyelid. C, lid scarred with trachoma, with its lashes rubbing against the cornea. D, the incision, extended through the lid margin at both ends (*arrows*). E, evert the lid with tension sutures. Make the incision, and undermine the superficial surface of the tarsus gently in both directions. F, rotate the margin of the lid and suture it in place. G, completed lid eversion.
Kindly contributed by Roy Pfaltzgraff.

TARSAL EVERSION FOR ENTROPION (GRADE 2.4)

For this method, the tarsal plate must be stiff enough to take sutures. Place 3 stay sutures of black braided silk in the upper lid, evert it over a roll of gauze, and clamp the sutures to a drape.

Using a #15 scalpel, make an incision about 3mm from the inner margin of the lid, and parallel to it. Cut through the conjunctiva and the full length of the tarsal plate, at 90º to its surface, so as to free a strip from its edge (28-18E). Curve each end of the incision towards the free edge of the lid, so that you can evert the strip of lid that bears the lashes.

Use skin hooks to retract the free edge of the lid. Use the tip of your scalpel to free the tissues from the anterior surface of the strip of tarsal plate for about 2mm, until you see the follicles of the lashes in the base of the wound.

Now undermine the anterior surface of the main part of the plate to a depth of about 4mm. A little undermining like this will help you to mobilize the free edge of the tarsus. Do this in the plane between the *orbicularis* muscle and the insertion of the *levator palpebrae superioris* tendon.

CAUTION! Take care not to buttonhole the skin.

You will now be able to rotate the distal fragment through 180º (28-18F). If you cannot, undermine the conjunctiva more widely. Insert 3 small mattress sutures of 4/0 absorbable, so that the knots are buried (28-18G). Put tetracycline eye drops into the conjunctiva tds for 1wk.

This operation gives the upper lid a new edge, and makes it c.3mm shallower, but it will still meet the lower lid on shutting the eye.

Alternatively, if the tarsal plate is shrunken and degenerated, it will not take sutures, so remove it entirely.

A more complicated procedure is a tarsal plate rotation with a mucosal graft from the mouth, but this is for an expert.

If the eye is painful and blind, it may be better removed. This is one of the occasions on which the indications are more critical than the operation.

28.14 Destructive methods for the eye

Before you start any destructive operation:
(a) Get signed informed consent from a child's parent or guardian or from an adult himself.
(b) Make sure you operate on the correct eye!

Don't rush in to perform these procedures:

(a) The main indication for enucleation is persistent severe pain in a blind eye.

(b) If it has been injured, always try to repair it first, no matter how hopelessly injured it seems. In some cases the eye may scar but remain stable for years.

(c) Prostheses may be difficult to find, and the best one is the natural eye, even if it is blind.

CAUTION!

(1) Unless you are operating for malignancy or acute infection to save life, the eye must be totally blind. Test this with a strong light.

(2) When pain is the main indication, it must be considerable. Pain is subjective, so make sure, if you can, that it is genuine. Review this on several occasions.

If there is any sensation of light, *don't perform a destructive procedure*. That little eyesight may be useful later.

(a) Evisceration of an eye (GRADE 3.2) is the least radical procedure; scrape out the contents of the globe and leave the sclera intact. This is the only safe procedure if the eye is infected, because a sleeve of dura containing CSF surrounds the optic nerve. Other operations require that you cut it, and so open up a potential path of infection to the meninges.

You may need to eviscerate the eye:

(1) When antibiotic therapy fails to control a severe infection causing suppurative endophthalmitis, leading to orbital cellulitis, and oedema of the lids. If you don't eviscerate the eye and drain the pus from it, the infection may spread and cause cavernous sinus thrombosis and meningitis, and even death.

(2) When there is a chronic, less urgent infection in a blind, painful, useless eye.

Don't take the eye out unless you are left with no other option. Try to find a prosthesis to insert when the sepsis has settled: it vastly improves the patient's appearance.

ANAESTHESIA. If there is no significant infection, you can use the combination of a facial and a retrobulbar block. Otherwise use ketamine or GA.

METHOD

Incise the conjunctiva all round 360^0 at its junction with the cornea, using fine-toothed forceps and fine scissors (28-19A). Separate Tenon's capsule (28-1C) bluntly from the underlying sclera in 4 quadrants. Cut through the corneoscleral junctional at the limbus with scissors (28-19B). Excise the entire cornea (28-19C).

Scoop out all uveal tissue contents of the eye with an evisceration spoon, curette or periosteal elevator (28-19D).

Pack the sclera for a few minutes to control bleeding. Excise a 5mm triangle of sclera from each side (28-19E), to help make the globe collapse. Rinse the inside of the globe with hydrogen peroxide.

EVISCERATION OF THE EYE

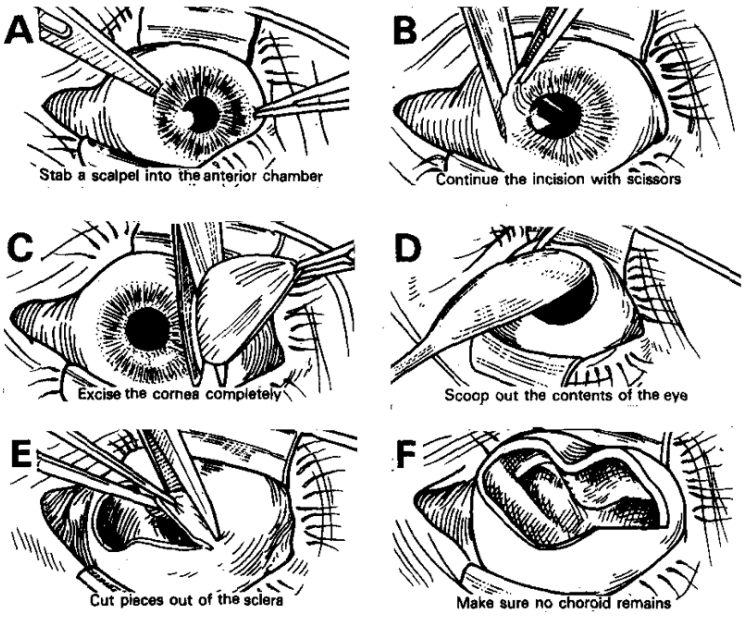


Fig. 28-19 EVISCERATING AN EYE. A, conjunctiva already incised and undermined. Stab a #11 scalpel blade through the cornea into the anterior chamber. B, continue the corneal incision with scissors. C, excise the cornea completely. D, scoop out the contents of the eye with a curette. E, cut a triangular section out of each side of the hole in the sclera. F, inspect the inside of the eye to make sure that no choroid remains.

After Galbraith JEK. Basic Eye Surgery, Churchill Livingstone 1979 p.84 Figs 9.17-24 with kind permission.

Make sure that no black choroid remains to avoid the risk of sympathetic ophthalmitis. Close Tenon's fascia and conjunctiva with 6/0 nylon.

If you are operating for acute infection, leave it open to drain.

If there is a chance of getting an artificial eye, insert a plastic conformer shell.

POST-OPERATIVELY, control bleeding by bandaging 2 eyepads firmly over the socket. Leave the dressing on for 2 days. Clean the lids and lashes bd, and put 2 drops of chloramphenicol 0·5% into the socket. You can insert an artificial eye after 4-6wks.

(b) Enucleation of an eye (GRADE 3.3) removes the globe by dividing the conjunctiva, the extrinsic muscles of the eye, and the optic nerve. *Do this only where there is no active infection; it is contraindicated when there is*. Indications are:

(1) A malignant intraocular tumour (retinoblastoma or melanoma) is an absolute indication.

(2) A penetrating wound, especially in the circumcorneal region, complicated by iridocyclitis, and entanglement of the iris, lens capsule, and vitreous. *If you leave an eye like this, sympathetic ophthalmitis may follow in the other eye*.

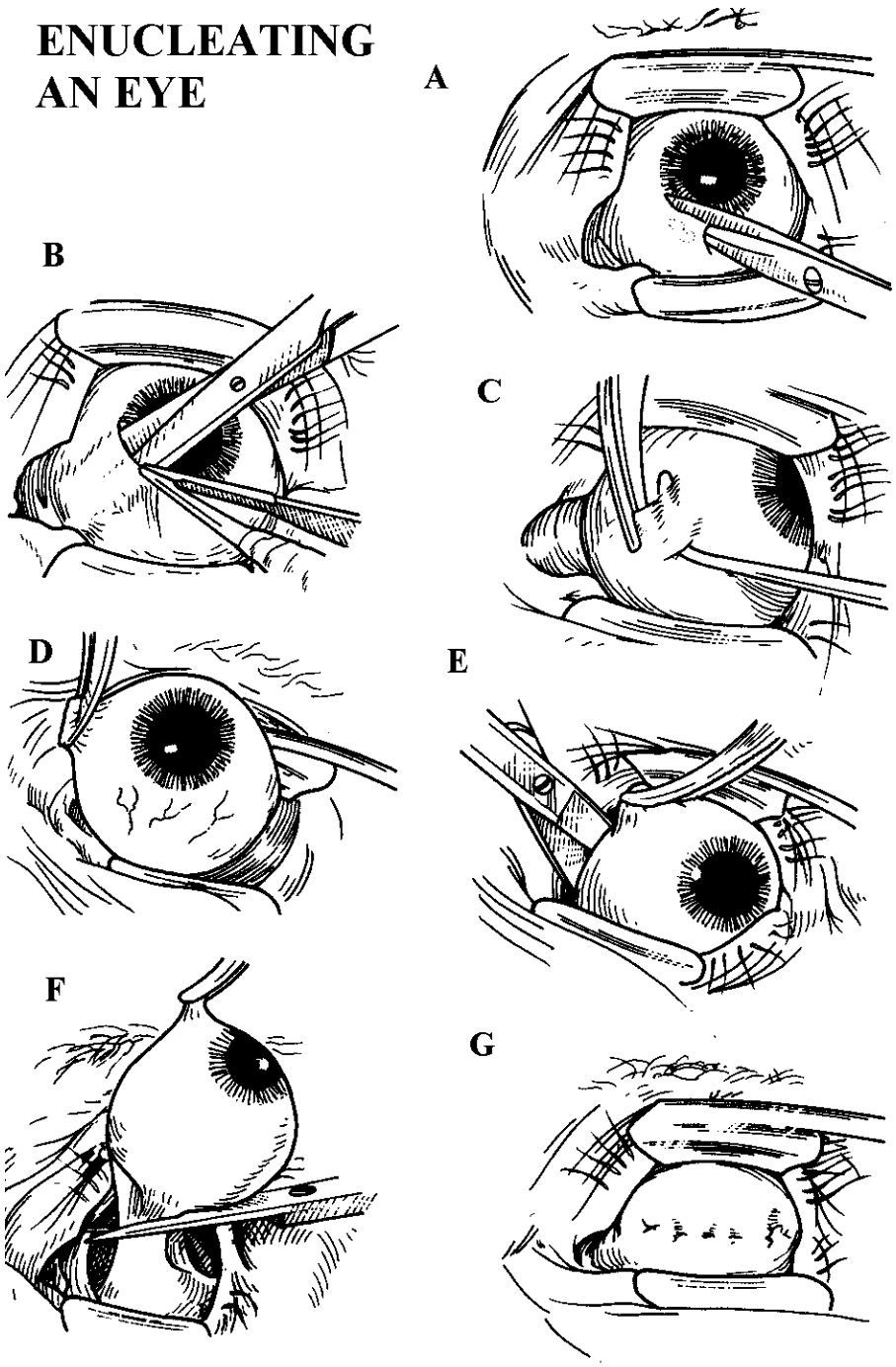


Fig. 28-20 ENUCLEATING AN EYE.
A, incise the conjunctiva. B, undermine the conjunctiva for about 8mm. C, slip a muscle hook under each *rectus* muscle, bring it forwards into the wound, and cut it. D, draw the eye forwards by pulling on the insertion of the *medial rectus* muscle. E, cut the optic nerve from the medial side. F, cut any remaining adherent tissue. G, suture the conjunctiva with absorbable suture.
After Galbraith JEK Basic Eye Surgery, Churchill Livingstone 1979 p.81-2 Figs. 9.9-15 with kind permission.

ANAESTHESIA
A retrobulbar block using ≥6ml of lidocaine, combined with a VIIth cranial nerve block. Otherwise use ketamine or GA.

METHOD
Mobilize the globe from its conjunctival attachments. Cut around it, and define the insertion of the 4 extraocular *rectus* muscles, about 8mm from the edge of the cornea (28-20B). Slip a squint hook under the *medial rectus* muscle (28-20C), and pull it into view.

In order to put in an implant, lift the muscle and insert a mattress absorbable suture through its belly, about 3mm from its insertion. Clamp its insertion, remove the squint hook, and cut the muscle with scissors.

Separate each *rectus* muscle in the same way. Leave the stump of the *medial rectus* tendon a little longer, so that you have something with which to hold the globe.

Make the globe prolapse forwards out of the orbit by closing the arms of the speculum behind it, and pushing them backwards. If the eye is so enlarged that it will not fit between the blades of the speculum, pull it forwards by holding the stump of the *medial rectus* muscle with a haemostat (28-20D). Pass an angled forceps down the medial side of the orbit, feel for the optic nerve and arterial supply behind the eye, open it and clamp the vessels, and divide them and the nerve (28-20E,F).

If you are enucleating the eye for a malignant tumour, cut as far posteriorly and laterally as you can, to keep clear of tumour. Pull the eye forwards, and cut any tissue that remains attached. Put a hot wet pack into the orbit, and press on it until bleeding stops.

If you wish to insert a simple glass globe implant, place it in the muscle cone and Tenon's capsule, and suture the conjunctiva over it.

If there is no possibility of an implant, close the conjunctiva and Tenon's capsule separately with 5/0 absorbable (28-20G). Irrigate the socket with 0·5% chloramphenicol.

(c) Exenteration of the orbit (GRADE 3.4) is a bloody, mutilating operation. It removes the entire contents of the orbit, together with its periosteum, the globe, and all its extrinsic muscles.

Consider doing this when there is an advanced conjunctival carcinoma (28.15) or a retinoblastoma which has penetrated the globe and caused proptosis.

It will not prolong life, but the last days might at least be more comfortable. An empty orbital cavity will remain, which you can line with split skin, or allow to granulate.

ANAESTHESIA
Use ketamine or GA. Have blood for transfusion available.

METHOD
If the lids have been involved by tumour, suture their margins together with 3/0 silk. Use a #15 scalpel blade to cut round the margins of the orbit. If the lids are not involved, incise closer to the scleral margins, so as to save all, or some, of the skin from the lids to line the empty orbit. Control bleeding from the upper inner margin of the orbit with diathermy, adrenaline solution or hydrogen peroxide. Incise the periosteum round the margin of the orbit, and reflect it as far posteriorly as you can. It is firmly adherent at the bony suture lines of the skull.

CAUTION!
(1) The bone on the medial wall of the orbit is very thin, so elevate the periosteum here with special care.
(2) The tumour may have eaten through the wall of the orbit, into the brain. If so, you may find it difficult to be sure of the anatomy.
The periosteum should strip easily until you reach the orbital fissures, and the nasolacrimal duct (6-4). Cut this.

Separate the palpebral ligaments, the trochlea (the pulley structure for the tendon of the *superior oblique*), and the *inferior oblique* muscles from the bone with the periosteum. Use curved scissors to cut the structures entering through the orbital fissures. Pull the contents of the orbit forwards, and cut the tissues at its apex with strong scissors as far back as you can.

Bleeding may be profuse. Remove the contents of the orbit quickly, and then control bleeding.

Turn the skin at the edges of the orbit back into it. Graft its raw surfaces with split skin, either now or as a secondary procedure in 10-14 days. Gently pack the orbit. If you are not grafting it, dress it with disinfectant such as acriflavine wool, and apply a firm bandage.

(d) Difficulties with destructive methods for the eye

If the patient refuses to have a painful eye enucleated or eviscerated, consider injecting absolute alcohol behind it to destroy its sensory nerves. You can use any strength of alcohol, provided it is more than 50%, but you may need to repeat the injection if pain returns. Permanent relief is uncertain.

Retrobulbar alcohol is very painful for about 30secs, so use a retrobulbar block of lidocaine 1ml. Remove the syringe and needle. When the block is effective, put another syringe on the needle and inject 2ml of alcohol. The orbit will become severely oedematous for 10 days. Add chloramphenicol eye drops qds for 1wk.

28.15 Conjunctival carcinoma

The conjunctiva is a thin transparent mucous membrane which covers the inner aspects of the eyelids and is reflected over the anterior part of the eyeball and ends at the limbus. Conjunctival epithelium is non-keratinized. It is continuous with the cornea at the limbus and the skin at the lid margins.

Squamous carcinoma of the conjunctiva used to occur mostly in the elderly but now is commoner among young HIV+ve patients, in whom it acts much more aggressively. It may arise on its own or from a pre-existing lesion like a pterygium (28.18). When small, it appears as a whitish lesion surrounded by a hyperaemic area, whose surface is irregular with small nodular parts. Initially it is mobile but as it invades, it becomes fixed.

Advanced cases are easy to diagnose, and may be associated with metastases to pre-auricular and submandibular lymph nodes.

(a) Small lesions you can completely excise; this is advisable as it can then provide histological confirmation as well as information on the invasiveness of the tumour.

(b) Large lesions need an exenteration (28.14) with removal of regional lymph nodes. Radiotherapy is useful, but chemotherapy needs the help of an expert, especially if HIV disease is present.

28.16 Retinoblastoma

This uncommon, malignant, radiosensitive tumour of the embryonic cells of the retina usually presents in children <2yrs. It is bilateral in 25% of cases, and occurs in 2 forms, familial and sporadic. At first, the tumour enlarges within the eye; later it grows through the sclera, or chambers of the eye, to perforate the cornea. It can also spread through the optic nerve (where it may cause glaucoma) to the subarachnoid space. Familial cases are inherited as autosomal dominants. Healthy patients with one affected child have only a 5% risk of producing a second one. If a child survives, there is a high risk of transmitting the disease to the offspring.

The earliest sign is a squint or a fixed dilated pupil with a white reflection ('cat's eye'), and greyish white tumour visible with an ophthalmoscope. Presentation is often late, with a large globe, and tumour penetrating the sclera or cornea.

(a) Differential diagnosis varies with the stage of presentation, and is easy after glaucoma and intraocular extension have occurred. Before this, the diagnosis can be difficult, when all you can see is a white mass in an infant's vitreous. Dilate the pupils, and examine both the fundi under LA. Enquire for a family history.

Early:
(1) Traumatic perforation of the globe.
(2) Traditional medicine in the eye.
(3) Retrolental fibroplasia (history of prematurity at birth).

Late:
(1) A corneal ulcer leading to perforation, an anterior staphyloma (due to bulging of a weak cornea, 28.3).
(2) Panophthalmitis.
(3) Congenital glaucoma (28.6).

(b) Management depends on the stage of presentation.
If early with the tumour confined to the globe, try to refer for radiotherapy, which cures 85% of cases. Otherwise, enucleate the globe (28.14).

If the tumour has extended through the globe, but not through the wall of the orbit, you should remove the globe and refer for radiotherapy, but the prognosis is so poor that a long journey to a referral hospital is not worthwhile. Consider chemotherapy.

If there is proptosis and a fungating mass, exenteration of the orbit (28.14) will remove the mass, but is unlikely to prolong life.

CHEMOTHERAPY
In 35% of cases the following regime causes a partial response, and in 65% it prevents progression for a while. Use vincristine 1·4mg/m², doxorubicin 50mg/m² and cyclophosphamide 1g/m². Administer all drugs IV once every 21 days.

28.17 The eyes in leprosy

Leprosy causes:
(1) Paralysis of the facial nerve, affecting the *orbicularis* muscle so that the eye does not close (lagophthalmos).
(2) Loss of sensation in the ophthalmic division of the Vth cranial nerve, which makes the cornea anaesthetic.
(3) An acute iritis (uncommon), which is usually associated with a type II reaction and is less common if the patient is taking clofazimine.
(4) A chronic iritis (common) causing atrophy of the *dilator pupillae* and a small unreactive pupil.

The combination of these lesions can have a devastating effect on the sight. An anaesthetic cornea prevents a patient noticing that there is something in the eye, or that it is dry. The blink reflex disappears, so that, even if there is still enough power in the VIIth cranial nerve to blink, it does not wash and wet the conjunctiva automatically. As a result, the cornea may be unprotected, especially during sleep, so that it may develop exposure keratitis, and ulcerate. If the centre of the cornea becomes opaque, sight is damaged. So, warn of the danger of an anaesthetic cornea, and examine the eyes regularly.

To decide if the cornea has been damaged, look for superficial scars, and use fluorescein drops, or papers, to search for central staining. If the cornea is anaesthetic, the eye is at great risk. If there is lagophthalmos, but the cornea is not anaesthetic, he may have enough sensation to complain of discomfort or burning.

To find out if there is significant lagophthalmos, examine the closed eyes. If the cornea is completely covered, all is well. But if any part of it remains exposed, you should do something to protect eyesight. Several operations are possible. Tarsorrhaphy aims to reduce, or remove entirely, the gap between the closed eyelids. It has cosmetic disadvantages, but it does save sight, and it is not difficult, so you should be able to do it if you care for leprosy patients. A more effective procedure is transfer of the *temporalis* muscle.

28.18 Other eye problems

Here are some of the other eye problems you may meet.

If opening the eye is impossible, this is PTOSIS. This may be congenital, traumatic, acute as the result of an oculomotor palsy, HIV disease, or the result of myasthenia gravis.

If there is a small yellowish-white lump adjacent to the cornea in the region exposed by the palpebral opening, this is a PINGUECULA. It is harmless so give reassurance, though it may develop into a pterygium.

If there is a wing-shaped vascular thickening of the conjunctiva which grows on to the cornea, usually from the medial side, this is a PTERYGIUM. It is a wedge-shaped benign fleshy growth on the scleral conjunctiva that may grow onto the cornea; this is more frequent in increased UV exposure from outdoor activities. It is seldom serious. If vision is good, leave it. If it is advancing over the centre of the cornea and impairing vision, excise the pterygium carefully under LA off the conjunctiva, and dissect it off the cornea. Leave the sclera bare, and gently cauterize it. Up to 50% recur.
Scarring can result in a squint.

If there is a worm under the conjunctiva (*e.g.* in the rainforest belt of West Africa), this is *loiasis*. Use albendazole 5mg/kg bd for 3wks.

If an old person has a complete or incomplete white ring encircling the cornea about 1mm within the limbus, this is ARCUS SENILIS. It is a sign of high blood cholesterol levels.

If the sclera are deep yellow, this is jaundice. There may be surgical correction possible if the cause is cholestasis (15.9).

29 The ear, nose & throat

29.1 Introduction

Ear, nose and throat (ENT) disease is common in primary care. It is said that 1:3 of all patients coming to primary care do so because of an ENT problem. Patients come with a sore throat, poor hearing, noises in their ears, bleeding from the nose, blocked nose and many other symptoms. In some ways, ENT symptoms are often neglected in primary care because they are often thought not to be so important: they don't usually kill people although they do make life unpleasant and uncomfortable, and these areas of the body can be difficult to examine. Sometimes, however, there is indeed something serious, such as a cancer or a blocked airway, so it is important to be able to perform an ENT examination and to recognize common and serious conditions.

To see the eardrum or the larynx or the inside of the nose, it is necessary to have equipment. This equipment does not have to be sophisticated and expensive. A torch, even a simple one, can allow you to look at the throat, up the first part of the nose and into the ear canal. However, to see the whole of the inside of the nasal cavities, to see the eardrums and to see the back of the tongue and voice box, you need to have some equipment such as an otoscope, a headlight and a mirror. You also need forceps and a suction machine for treatment.

This chapter will describe common surgical ENT conditions, and how to manage them with limited resources. In primary care, your healthcare worker should be able to:
(1) do a basic ENT examination,
(2) remove most foreign bodies of the ear and nose, and some of those in the throat,
(3) diagnose common infective conditions of the ear,
(4) clean the ear safely of wax or pus,
(5) estimate hearing loss and understand if the hearing loss is conductive or sensori-neural,
(6) drain pus,
(7) recognize an obstructed airway and carry out emergency surgery to help the patient breathe,
(8) deal with bleeding noses,
(9) recognize cancers of the throat,
(10) treat sinusitis.

This is the equipment you will need:
LIGHT SOURCE, either a headlight (better) or torch.
OTOSCOPE, of which there are many types, and which can be used either with rechargeable batteries or with a charger.
TUNING FORK, Hartmann's, 512Hz, or musical tuning fork of 440Hz for tuning musical instruments (much cheaper).
WAX HOOK (a hair pin or long lumbar puncture needle with the sharp end broken off and the end 2mm bent through 90^0).
PROBE, such as the Jobson-Horne.
COTTON WOOL wound around thin tooth pick to mop out an ear (normal cotton buds are usually too large).
AURAL SYRINGE, Bacon's, for one-hand use. This has a rubber bulb, a tube, and a valve. It delivers a steadier stream of fluid than a metal syringe, and can be used with one hand. If you don't have an ear syringe, use an ordinary 20ml syringe with an eccentric nozzle. If you wish, you can fix a small plastic cannula to its tip, and cut it short to prevent it being pushed in too far.
FORCEPS of different sizes for use in the ear canal, nasal cavity and mouth.
NASAL SPECULUM such as the Thudichum's, Mark Hovell modification, size 7. Use this for examining the nose. Dangle one on the distal IP joint of your flexed index finger, and control it by holding the limbs together between your middle and ring fingers.
SUCTION motor or foot-driven and suction nozzles.
MIRRORS for examining the throat and larynx: laryngeal mirrors.
N.B. Dental mirrors are usually concave (not flat like laryngeal mirrors) and therefore impossible to focus on the larynx.
SNARE for removing nasal polyps.
TONGUE DEPRESSORS, wooden (preferably) or metal; the handle of an ordinary tablespoon will do (sterilized after each use).
ELECTRO-CAUTERY.
RIBBON GAUZE, 1cm width for packing the nose; 0·5cm width for packing the ear.
FOLEY CATHETER, Ch12, 14 or 16 for posterior epistaxis.
PASTES FOR DRESSINGS: (1) BIPP (bismuth iodoform paraffin paste) gauze, for packing the ear. (2) Simple petroleum jelly for lubricating ribbon gauze for the nose.
SILVER NITRATE CAUTERY STICKS.
SALINE FOR IRRIGATING THE NOSE to keep it clean of pus and crusts.
OLIVE OIL is useful for softening wax.

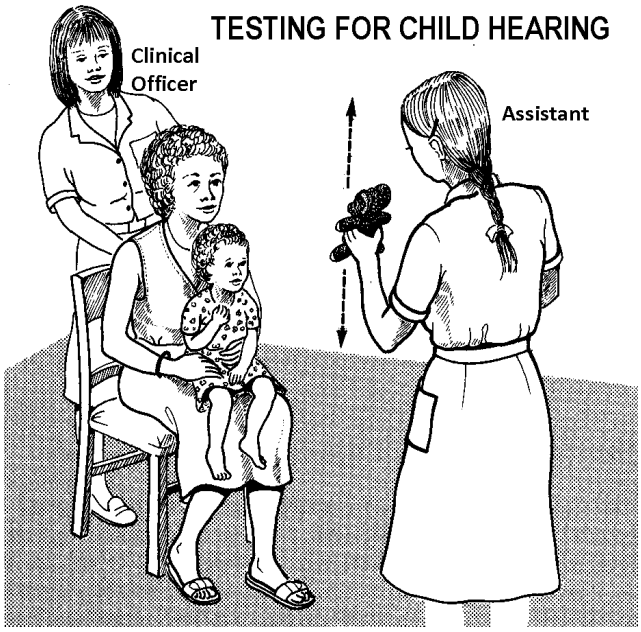


Fig. 29-1 TESTING FOR CHILD HEARING.
Sit the child on the mother's knee facing your assistant. Meanwhile remain out of sight behind the mother. The distraction test is described later (29.2)**.**

29.2 Deafness

Severe deafness cripples the mind by preventing communication with other people. It is thus a serious handicap, and, alas, a neglected one. Some 360 million (1:20) worldwide have moderate to profound hearing loss, of whom 80% live in LMICs; 50% of these cases would have been preventable. Current production of hearing aids meets only 10% of the world need, and <1:40 people who need them have them.

Moderate hearing loss – difficulty hearing normal conversation.
Profound hearing loss – unable to hear shouted speech when standing just 1m away.
Try to find out the incidence of deafness in your district, and the common causes for it. *Remember diabetics lose their hearing just as they lose peripheral sensation!*

Chronic ear infection leads to hearing loss, as can meningitis, measles and mumps. Other common causes include exposure to excessive noise, head and ear injury, ageing and the use of ototoxic drugs (especially gentamicin, streptomycin and some cytotoxic drugs). Congenital hearing loss can occur with maternal rubella, syphilis, and HIV disease; low birth weight and birth asphyxia, and neonatal jaundice as well as drugs are other common causes.

The ear is made up of the:
(1) outer ear: external auditory meatus, pinna, ear canal,
(2) middle ear: tympanic membrane (eardrum),
three small bones (ossicles: *malleus*, *incus* and *stapes*),
(3) inner ear: labyrinth (3 semicircular canals, vestibule and cochlea) with the VIIIth cranial (auditory, acoustic *or* vestibulo-cochlear) nerve.

Hearing involves sound coming to the ear and being directed down the ear canal, with the sound vibrations being transmitted to the eardrum and then through the ossicles to the cochlea. In the cochlea, the mechanical sound vibrations are transformed into electrical impulses which are transmitted along the auditory nerve to the brain stem and then to the cerebral cortex and consciousness.

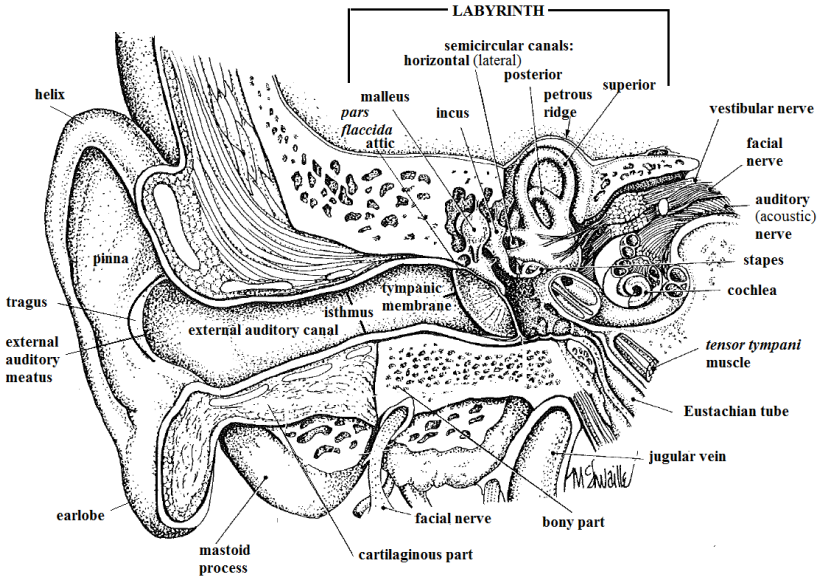


Fig. 29-2 ANATOMY OF HEARING PATHWAYS.

Hearing loss is classified into two important types:
(1) conductive, or
(2) sensori-neural.
Some patients have both.

Conductive hearing loss means failure of transmission of sound to the cochlea. This can be due to:
(1) failure of development of the external ear
(2) the ear canal being full of wax (this is unusual unless people use earbuds, which push the wax into the ear, rather than helping it to come out)
(3) infection of the ear canal (otitis externa) leading to blockage of the canal by swelling and infective debris
(4) problems with the eardrum, especially with a perforation (hole) in the eardrum
(5) damage or stiffness to one or more of the 3 ossicles, usually because of chronic infection, cholesteatoma or injury, sometimes by congenital or hereditary illnesses
(6) fluid or 'glue' in the middle ear (secretory otitis media or middle ear effusion) or pus in the middle ear (suppurative otitis media).

Conductive hearing loss is potentially curable *e.g.* by removal of wax, drainage of middle ear fluid, repair of damaged eardrum or ear bones, and treatment of otitis externa or otitis media.

Hearing aids can also help many forms of conductive hearing loss (as long as there is no active infection). They need to be fitted properly and require supplies of batteries, and it is necessary for patients to be taught how to use them. *Hearing aids will not help hearing in ears that are infected and discharging pus. If used in an infected ear, the hearing aid will usually make the infection worse.*

Sensori-neural hearing loss implies disease of the cochlea or of the auditory nerve. This can be due to:
(1) Damage to the cochlea, due to infection (viral infection such as mumps, herpes simplex, herpes zoster, measles, meningitis), injury (fracture of base of skull), loud noise or ageing.
(2) Damage to the auditory nerve, due to infection (more common) or tumour (rare).

Sensori-neural hearing loss is most often irreversible and requires hearing aids (in one or both ears), though if very severe can be cured by cochlear implantation (a procedure which involves placing electrodes into the cochlea, which is both expensive and requires prolonged rehabilitation for success).

A special form of hearing aid is bone-anchored, which can be used in infected ears, but this is expensive and sophisticated technology, like the cochlear implant.

Central hearing loss, where there is a problem in the cerebral cortex, is rare.

Prevent deafness by helping to ensure:
(1) congenital infection, such as by rubella, is prevented by maternal vaccination programmes,
(2) prompt, effective treatment of neonatal infections,
(3) prompt diagnosis and effective treatment of meningitis in children,

(4) vaccination programmes exist against meningococcal meningitis,
(5) treatment of ear infections before they develop into chronic middle ear disease, which destroys the middle ear,
(6) early diagnosis of prelingual deafness,
(7) protection against noise damage,
(8) proper dosage of ototoxic drugs, especially of gentamicin and streptomycin.

Prelingual deafness is deafness in children who have not yet learned to speak. Make every effort to identify such children and help them (by hearing aids and/or surgery) to become familiar with sound, since if the brain is not exposed to sounds by the age of 5yrs, the child may never speak at all. A child may however learn lip-reading and sign language at an early age; remember that this is different in each country. Gestuno, the International Sign Language, is heavily influenced by languages of rich countries.

Damage to hearing by loud noise is an important cause of deafness. Anyone who works in a very loud environment should wear ear plugs or muffs to protect the ears from noise damage. If, in a noisy environment, you cannot really hear what someone, standing just 1m away, is saying when they shout to you, then the noise is too loud and potentially damaging. In such circumstances, you must protect the ears. Children and adolescents (and adults) who listen to very loud music are also at risk of damage to hearing, especially if they use intra-aural (earbud) earphones.

Examining the ear

(1) Look at the outer ear: are there any abnormalities (a deformed or absent outer ear is usually associated with a congenital abnormality of the middle and/or inner ear)?
(2) Look behind the ear and feel for the mastoid bone: is it swollen or tender as in mastoiditis or a mastoid abscess?
(3) Examine the ear canal: is it blocked?
(4) Examine the eardrum (29-3). For this you need an otoscope. Pay attention to the position of the eardrum and to the handle of the *malleus*. Look for any holes in the eardrum (29-3B-D). See if the eardrum is retracted (29-3E,F). Look at the colour of the eardrum. Fluid or glue in the middle ear makes the eardrum look greyish or dull (29-3J). If the ear is painful and the eardrum is bulging, then there is likely to be pus in the middle ear (acute purulent otitis media). Look for cholesteatoma (abnormal skin in the middle ear: 29-3N).
(5) Examine if the eardrum moves inwards momentarily when the patient swallows, or outwards if he carries out a Valsalva manoeuvre (holding the nose and blowing the cheeks out): this proves the Eustachian tube (29-5) is not blocked.

N.B. The light reflex is always in the anterior inferior quadrant; *its absence does not necessarily signify pathology*.

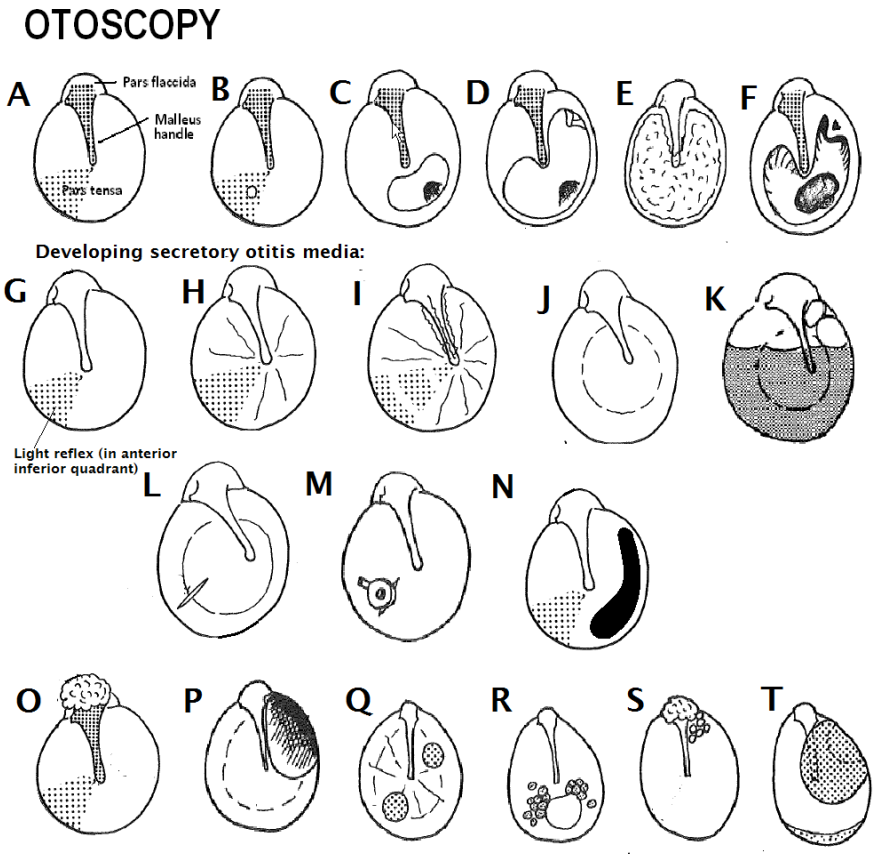


Fig. 29-3 OTOSCOPIC APPEARANCES (LEFT EAR).
A, normal. B, small perforation. C, central perforation revealing the round window at the back of the inner ear. D, subtotal perforation revealing the incudo-stapedial joint. E, tympanosclerosis resulting from chronic adhesive otitis media. F, perforated drum adherent to the medial wall and incudo-stapedial joint.
Developing secretory otitis media: thin fluid exudate in obstructed Eustachian tube (29-5) **(G); prominent blood vessels ('bicycle spokes', H); break-up of light reflex (I); bulging eardrum with increasing opacity (J); fluid level in middle ear (K). L, site of antero-inferior myringotomy (left ear) avoiding ossicles. M, grommet tube** (29.4) **inserted for aeration. N, calcified drum of tympanosclerosis** (29.4)**. O, superior (attic) perforation with cholesteatoma** (29.4)**. P, retraction posterior-superiorly from suppurative chronic otitis media. Q, bullous myringitis. R, central perforation with granulations. S, superior (attic) perforation with granulations. T, aural polyp.**
After Metcalfe S, Annotated ENT. Wilderness, Guildford, 1980.

Testing for hearing

There are two ways to test for hearing: clinically and by audiology. Clinical testing is useful but has limitations. Audiology is more accurate and important for epidemiological studies but requires an audiometer, which can measure hearing levels in each ear. If you are serious about offering services for hearing, then an audiometer and some training in audiology is essential. *Even without an audiometer, however, clinical testing of hearing is possible.*

Always do the next 2 tests as a pair; separately they will not give you the information you need.

(1) RINNE'S TEST
Strike a tuning fork gently against your knee or elbow (*not against a hard surface*, or unwanted overtones will be produced). Place the foot of the vibrating fork firmly on the patient's head, just above and behind the ear to be tested; apply sufficient pressure so that you need your other hand to support the other side of the head. Wait till he no longer hears the sound and then immediately place the fork, still vibrating, beside the ear canal. Normally he should still hear the fork vibrating (+ve Rinne test). If he does not hear the sound anymore (-ve Rinne test), his bone conduction is better than air conduction, and there is a problem with sound conduction of at least 20dB (middle ear disease).

CAUTION! If there is severe sensori-neural deafness in one ear, this may give a misleading -ve Rinne test because the sound is conducted through the head and heard in the other ear. Weber's test will distinguish this.

(2) WEBER'S TEST
Strike the tuning fork against your knee, place its foot on the middle of the forehead, and ask which ear hears the sound loudest. If there is conductive deafness in one ear (a -ve Rinne's test), because bone conduction is heard better than air conduction, the tuning fork is heard better in that ear. If there is sensori-neural deafness, the sound will be loudest in the better-functioning cochlea.

Don't be fooled that speech defects arise from 'tongue tie' (31.9)*!*

The Rinne and Weber tests help tell you whether a hearing loss is conductive or sensori-neural, but don't indicate what the level of hearing is.

Hearing is measured in decibels (dB). The more decibels a sound is, the louder it is. A whisper is around 30dB, the spoken voice around 60dB and a shout 90dB. Jet engines are in the region of 120dB. Being able to hear levels ≤30dB is generally satisfactory. The human ear should be able to hear a mosquito flying in a room 3m away.

N.B. The scale is logarithmic: 31dB is ten times as loud as 30dB.

To measure hearing, stand behind and slightly to the side of the patient, to the side that you are testing. If you are testing hearing in the left ear, stand behind and to the left side of the patient. Your opposite, right, hand is outstretched, and touching the right ear of the patient. With the index finger of the right hand on the patient's right ear, the right hand touching the back of the skull, and with the right arm straight, you are now about one metre behind and to the side of the patient.

With the right index finger, generally stroke the ear. Try this on your own ear, and you will hear a noise. This noise helps to 'mask' the hearing in the ear that you are not testing. Now whisper a number and see if the patient can repeat that number.

If the patient can hear the number whispered at 1m away, then the hearing is normal or close to normal. Then swap sides and hands, and stand behind and to the other side of the patient.

Another simple method is to ask if the ticking of a wrist-watch is audible if held up to the ear. Alternatively, rub the index finger and the thumb together. This produces a slight noise.

(3) CLINICAL TESTING OF HEARING IN CHILDREN
In a child <3yrs neither a tuning fork nor an audiometer is useful. Unless you have special equipment, you have to use: (i) the parents' account of an abnormal behaviour response, or the failure to make proper speech sounds; or (ii) the distraction test, which is effective in most young children.

THE DISTRACTION TEST is a valid screening method. Find a sensitive and understanding assistant, and practise making the test noises, which are the syllables of the word "shoe", spoken separately as two tests, a high-pitched "Shsh….." and a low, sung "Ooo…..". Make them softly, just loud enough for your assistant to hear.

Sit the child on the mother's knee facing your assistant (29-1). Meanwhile, remain out of sight behind the mother. Ask your assistant to gain the child's attention a little, by moving a toy up and down in a vertical line, while making the test sound. Then, ask him to hide the toy and break eye contact. At this exact moment, make a "Sh….." sound about 60cm from the child's ear, and level with it, while you remain out of sight. A normal child immediately turns towards the source of the sound.

Reward the child with some encouragement. Now test the other ear with an "Ooo…." sound, before returning to the first ear with an "Ooo…." sound, and then the second ear with a "Sh…." sound. To avoid false results, be sure to test the ears alternately.

If there is no response, try louder sounds. Then try a visual or tactile stimulus. If there is still no response, suspect cerebral disability, or some non-audiological problem. If there is now some response, repeat the sound stimuli at 2 or 3m, first in a louder voice, and then in a normal one.

CAUTION! These tests should tell you on which side to find the lesion.
(1) This is a very reliable test, *if you do it carefully*. Otherwise, you can easily get false results.
(2) Before the test itself, practise both the manner of attracting the child's attention, and the sound to be made.
(3) Timing is critical.
(4) Make sure the mother does not give away any clues, consciously or unconsciously.
(5) *You will get a misleading +ve* if you show yourself, or make visible the test object either directly, or reflected in a window, or some reflective object, or give some tactile clue. *You will get a misleading -ve* if the child gets bored, tired, or distracted by other things. If this happens, *don't persist*; try again later.

Management of conductive deafness
(i) OUTER EAR (EXTERNAL CANAL)
(1) WAX. It is important to understand that most wax is healthy and finds its own way out of the ear canal. *Leave most wax alone*. Wax can cause deafness, however, when it gets impacted into the ear canal.

The most common reason for this is the use of cotton buds, which pushes the wax into the canal (rather than removing it). You can remove wax carefully by using a thin hooked metal wire (but be careful not to damage the ear), by cotton wool wrapped around a thin wooden stick, by suction (but this requires a micro-sucker) or by syringing. Sometimes the wax can be too hard to remove, in which case ask the patient to put 3-4 drops of vegetable oil (thick olive oil is best) in the ear tds for 1wk.

N.B. It is legitimate to remove wax which is hiding an eardrum which you need to inspect.

SYRINGING THE EAR (GRADE 1.1). Make sure that there is no infection in the ear, and no perforation in the eardrum. Take a 20ml syringe filled with warm water, and gently flush out the ear canal, aiming the end of the syringe up or down but not directly at the eardrum.

N.B. Never point a needle into the ear canal!

(2) OTITIS EXTERNA (29.3). Infection in the ear canal can cause its wall to become swollen and filled with debris. It may then be necessary gently to re-open the canal by daily application of antibiotic and acetic acid. *Use steroids only for psoriasis*. Occasionally the canal needs clearing, if the eardrum is intact. More rarely, necrotizing infection ensues which needs radical debridement. Otitis externa pain can be severe indeed.

(3) FOREIGN BODY. This does not usually cause deafness unless associated with a blocked canal or infection or it has caused damage to the eardrum and ossicles.

(4) A TUMOUR (rare).

(ii) MIDDLE EAR

(1) A HOLE IN THE EARDRUM (A PERFORATED TYMPANIC MEMBRANE). The hole may be small or so large that it is easy to miss (you will look straight at the inner wall of the middle ear, and not see any eardrum: 29-3B,C,D). Hearing loss in ears with perforated eardrums is usually due to infection producing damaged, scarred and immobile ear ossicles. So always treat discharging ears promptly with antibiotics, but if discharge from the ear continues despite antibiotics, then it is likely that the patient has chronic middle ear disease that needs surgery. If an ear with a hole in the eardrum keeps getting infected, again surgery can be useful by repairing (and closing) the hole.

(2) FLUID OR 'GLUE' IN THE MIDDLE EAR. This is due either to a recent ear infection or to a blocked Eustachian tube (29-5). Recognizing middle ear fluid can be difficult but the eardrum may look opaque (29-3G-K). You can sometimes see an air-fluid level behind the eardrum, or you notice that the eardrum does not move on swallowing or on the Valsalva manoeuvre.

In most cases the fluid absorbs by itself, though this can take many months. Alternatively, a myringotomy (29-3L), making a hole in the eardrum, will release the fluid. *Be careful to do this anterio-inferiorly*, because you can easily damage the ossicles of the ear.

Fluid and 'glue' in the middle ear is usually the result of a problem in the throat: enlarged adenoids in children, rhinosinusitis, or sometimes (in adults, and even young people), a nasopharyngeal cancer. If due to rhinosinusitis, treating this will allow middle ear fluid to resolve.

(3) CHRONIC MIDDLE EAR DISEASE. This is disease of the middle ear which may be due to long-term tympanic perforation, scarred ear ossicles or a **cholesteatoma**. This is a white, skin-like debris that grows into, fills and destroys the middle ear (29-3N), sometimes spreading out to damage the facial nerve and erode into the inner ear and even into the brain. Though not a malignant condition, it acts locally like one and so is dangerous, destroying hearing, and may be fatal. This needs skilled surgery requiring operating microscopes and micro-drills.

Chronic middle ear disease is an important cause of deafness, important because all too often it results from acute (and simple) ear disease not being treated adequately. Chronic middle ear disease will be prevented only if you can train up otologists to do audiological testing but much can be done by treating infected ears early with antibiotics.

(4) OTOSCLEROSIS results when the ear ossicles don't vibrate because they are stuck together by bone. It causes a conductive hearing loss but when you look at the ear and eardrum, everything looks normal. Treatment is by a hearing aid. Surgical correction needs a real expert, and failure will result in a 'dead ear', *i.e.* an ear with no hearing at all.

(5) TRAUMA. Conductive hearing loss can result from a tympanic perforation caused by trauma, either a sudden loud noise, *e.g.* a gunshot close by (acoustic trauma), a sudden change in air pressure, *e.g.* a slap on the ear with the palm of the hand (barotrauma), or a penetrating injury.

N.B. When you see a perforated eardrum resulting from trauma, it is best to leave it completely alone and not even prescribe any antibiotics; almost all cases heal naturally and spontaneously.

Management of sensori-neural deafness

(1) CONGENITAL COCHLEAR HEARING LOSS, due to mumps, measles, rubella and other virus diseases, or due to inherited genetic abnormalities.

(2) BACTERIAL MENINGITIS can be complicated by sensori-neural deafness due to infection passing from the meninges to the inner ear.

(3) OVERDOSAGE WITH AMINOGLYCOSIDE DRUGS**,** especially gentamicin.

(4) HEAD INJURIES, leading to skull fractures through the temporal bone destroying the cochlea.

(5) VESTIBULO (AUDITORY) NEURONITIS, which causes sudden inflammation in the cochlea or along the auditory nerve, sometimes due to herpes simplex or herpes zoster infection.

(6) EXCESSIVE LOUD NOISE due to industrial noise, the noise associated with construction and road building, and noise from too loud music ('disco deafness'). Exposure to loud noise destroys the tiny cochlear hair cells whose movement transmits a signal to the brain. Many rock band players are seriously deaf over 4kHz.

(7) PRESBYACUSIS. With age, just as muscles get weaker and hair turns grey, so hearing can deteriorate, though it seems that this deterioration is worse in the loud urban and industrial environments of modern life. Hearing is lost first and foremost in the high-frequency range.

Apart from **speech training** and **lip-reading**, **hearing aids** are usually the only help available in sensori-neural hearing loss, but are important. They amplify sound but children with small ear canals may feel pain at volumes as low as 10dB. Better hearing aids can amplify only a selected range of frequencies to avoid increasing the background noise that often makes hearing more difficult.

A child should not be forced to wear hearing aids if managing without, although beware that he may then risk harm because he misses environmental cues. Aid in the management of all forms of deafness. Maintenance and repair of hearing aids is a skill which needs training and equipment. Many hearing aids don't work simply because there is no battery, others because they are not adapted.

If a baby is born deaf, this will usually be suspected by the family. A mother who says that her baby is deaf is usually right. *Don't ignore her*. The baby's intelligence will probably be normal, and an 'island' of residual hearing may remain. The parents and older siblings must make the most of this. Instruct them like this: "Let him watch you speaking. Use speech and signs together, because you will not know which the child will later find easiest." Speak slowly and clearly, and indicate familiar objects as you name them. If necessary, repeat the word close to the ear. Show you are pleased whenever the child tries to use a word, however indistinctly. Include the child in as much play, and as many activities, as you can. As always, "success builds on success".

CAUTION! Children who are born deaf cannot learn to speak unless they have special teaching, from their parents or someone else, from as early in life as possible.

29.3 Otitis externa

Otitis externa exists in 4 types:
(1) A furuncle (an infected hair follicle), usually *staphylococcal*, near the entrance of the external auditory canal (29-2). This is very painful, because the skin here is tightly bound down to the perichondrium of the elastic cartilage of the ear. It occurs here because there are no hair follicles to become infected in the deeper bony part of the canal.
(2) A diffuse inflammation of the whole ear canal resembling eczema. The common causes are:

(a) excessive self-cleaning of the ear,
(b) excessive humidity,
(c) associated general skin infection,
(d) eczema or psoriasis.

This may produce swelling and blockage by debris of the ear canal and, rarely, necrosis.
(3) A vesicular eruption of herpes zoster of the canal and pinna, sometimes associated with a facial palsy, dizziness or hearing loss, owing to involvement of the VIIth and VIIIth cranial nerves (the Ramsay Hunt syndrome), often related to HIV disease.
(4) In an area endemic with leishmaniasis, a well-demarcated ulcer which may cause tissue destruction.

(a) Diagnosis
There is pain on moving the pinna or pressing the tragus. There is a purulent discharge from the ear canal, which may be blocked. Sometimes there can be an associated swollen, tender lymph node behind the ear.
Don't confuse this with the tender bone of mastoiditis.

(b) Differential diagnosis of otorrhoea
This is fluid discharging from the ear. You can make reasonable deductions from its nature:
Suggesting otitis externa: non-offensive and purulent.
Suggesting otitis externa: serous,
chronic: with severe otalgia.
fungal: musky odour with mild otalgia.
eczematous: with itching.
Suggesting cholesteatoma: offensive, thick and pasty.
Suggesting chronic otitis media: offensive and purulent.
Suggesting bullous myringitis, an aural polyp, chronic eardrum perforation with granulations, or petrous bone fracture: blood-stained.
Suggesting skull fracture: CSF, or fresh blood.

(c) Treatment
(1) **If there is a furuncle** (pustule) **in the ear**, use ibuprofen and a high dose of IV or IM cloxacillin. Apply an ear wick of BIPP.

CAUTION! Don't incise it unless it is clearly fluctuant, because there is a danger of perichondritis and collapse of the pinna.
(2) **If there is diffuse otitis externa**, keep the ear clean and dry. Use ear drops of cloxacillin or antifungal agents with aluminium acetate 8% antiseptic freshly prepared, as appropriate. *Avoid neomycin or gentamicin drops*, as they may cause hypersensitivity and deafness.

If the patient is a 'self-cleaner', treat the condition that is causing him to touch his ear, and persuade him to leave it alone. If the canal is blocked, use ketamine and suction the canal with a soft curette under direct vision till you can see the eardrum. If it is intact, irrigate with diluted hydrogen peroxide, and dry the canal thoroughly afterwards. Put a wick soaked in antibiotic and steroid drops tds into the ear canal. *Never leave a wick in for >3 days.*

If there is necrosis, use IV antibiotics and arrange a debridement under ketamine. *Use steroids only for eczema or psoriasis but never for prolonged periods.*

(3) **If there is herpes zoster oticus**, use aciclovir orally 200mg x5 daily and antiseptic ointment locally.

(4) **For leishmaniasis**, use miltefosine if you can (29.18).

29.4 Otitis media

Acute otitis media is typically a disease of children. A child presents with acute earache and fever; and if very young, with vomiting or fits. At first, the margin of the eardrum and the handle of the malleus are red; later, the entire eardrum is red and bulges, so that it obscures the malleus. A few hours later the eardrum may burst, giving instant relief. Otitis media is most common in children <1yr, and is often recurrent. *Haemophilus influenzae* or *streptococcus pneumoniae* are usually responsible. Antibiotics are effective, if you use them in adequate doses promptly. *There is no need to continue beyond 48h*.

(a) Acute otitis media

TREATMENT
Use a high dose of oral amoxicillin for 2 days. Aim for good compliance over this short period. If the child is very unwell, use IV antibiotics. As first choice, avoid chloramphenicol and use erythromycin only in penicillin allergy.

Relieve the pain, and apply local heat to the ear. As soon as there is improvement, and the eardrum is no longer bulging, stop the antibiotics.

If the pain and fever continue, and the eardrum is still bulging >24h of treatment, the organism is probably insensitive to the antibiotic you are using, or you are not using enough. If the dosage was correct, use cefuroxime.

If acute otitis media fails to resolve after 3 days' antibiotic treatment, consider myringotomy and change to erythromycin or azithromycin. If this fails, *don't persist with antibiotics indefinitely*. Search for infection in the nose, sinuses, nasopharynx or mastoid.

MYRINGOTOMY (GRADE 2.3)
Incise the eardrum (tympanic membrane) if pain has not improved after 3 days of antibiotic treatment, especially with a facial palsy. You can do this best using a spinal needle.

Make sure you use ketamine, and make the perforation in the antero-inferior quadrant to avoid the stapes (29-2, 29-3L). *Don't make the incision too close to the eardrum edge* or the pus will not drain properly. Make an adequate incision if the pus is thick *but do no more than perforate the thin eardrum membrane*, and irrigate the middle ear gently. Take a pus swab.

CAUTION! Don't attempt myringotomy unless you have good equipment and light, because you can easily dislocate the incudo-stapedial joint.

If you see a child after the eardrum has already perforated, and is discharging, culture the discharge and use ampicillin for 1wk. If the discharge is smelly, add metronidazole. Instil hydrogen peroxide drops for 1min, then syringe gently with warm sterile water. Teach the parents, or a nurse, to dry-mop the discharge with cotton wool. If this is not done often enough, otitis externa and a persistent discharge may follow. Monitor hearing. A persistent discharge after acute otitis media may sometimes suggest mastoiditis.

N.B. Continuing pain suggests extension of the disease. You may need to supply cotton wool to swab the ear and to repeat this tds. Acute otitis media may be followed by a middle ear effusion (sometimes known as glue ear or secretory otitis media), in which case there may be poor hearing and recurrent earache.

(b) Difficulties with acute otitis media

If there is severe earache, with a normal eardrum, suspect referred pain from dental infection, or an impacted wisdom tooth. If these are not responsible, suspect referred pain from the pharynx, or the temporo-mandibular joints. If an adult has earache, a normal eardrum, and an enlarged node in the neck, suspect that there is carcinoma of the pharynx (29.16) or larynx (29.17).

If you see an indrawn straw-coloured opaque eardrum with a fluid level or bubbles behind it, this is SECRETORY (SEROUS) OTITIS MEDIA. This is the result of obstruction of the Eustachian tube usually by enlarged adenoids, and is common in children recovering from otitis media; it may occur spontaneously. There is usually no pain, and little hearing loss. If there are bubbles or a fluid level, enough air remains to maintain hearing; with all the air gone, deafness is more marked. Middle ear effusions usually resolve spontaneously, so wait several weeks if necessary. If the effusion persists, it may alter behaviour, and impair speech, even if it does not cause marked hearing impairment. If school behaviour or progress is poor, or the acquisition of speech is affected, consider myringotomy, perhaps with the insertion of a grommet for ventilation (29-3M).

N.B. The grommet is a tube not intended for fluid drainage: it is simply a Eustachian tube bypass for air! It has lips at its end so it will not slip out.

If there is acute otitis media and facial palsy, myringotomy is essential. Distinguish this from herpes zoster of the geniculate ganglion (Ramsay Hunt syndrome, 29.3).

If there is tenderness, redness, and swelling over the mastoid process, ACUTE MASTOIDITIS (29.5) is present. This is usually accompanied by persistent fever, and a red bulging eardrum, with pus discharging through a perforation. Tenderness is severe, high on the mastoid process. Note that otitis externa may also produce postauricular swelling, due to the infection of an adjacent lymph node.

In an infant, acute mastoiditis causes a swelling above and behind the ear, displacing it outwards and downwards. If there is a pustule in the meatus, the swelling is evenly distributed up and down the postauricular groove, displacing the ear outwards, but not downwards.

If an adult develops secretory otitis media for the first time, consider the possibility of obstruction of the Eustachian tube by a nasopharyngeal tumour (29.16).

N.B. Instruct your primary care workers to clean the ear: syringe it with a rubber rat-tailed syringe, using water or, better, 30% spirit in saline. Then to insert drops of 50% spirit and, with the ear held uppermost for 2mins, to insert 2-3 drops bd after cleaning. (*Try to train them to recognize attic disease and a cholesteatoma which should not be syringed.*)

If there is a fluid level bulging behind the eardrum, and you have fine instruments and an auroscope, make a pinpoint puncture incision postero-inferiorly.

(c) Chronic otitis media

This is usually the result of failure to treat the acute stage. It exists in 2 types:
(1) Associated with a **'safe' central tympanic perforation**, which may be small or large. Periodically the ear becomes infected and discharges pus. Infection results either from outside the ear, usually when the ear gets wet, or infection spreads up the Eustachian tube (29-5) from the nasopharynx during upper respiratory tract infections or chronic rhinosinusitis. The infections lead to damage to the ear ossicles and moderate deafness.
(2) Associated with an **'unsafe' peripheral perforation** within which dead skin from the ear canal accumulates. This is called a **cholesteatoma**. The perforation may be small but is usually in the upper part of the ear or at the back of the eardrum. Often there is little ear discharge but when present it can be very smelly. The cholesteatoma grows and, although not malignant, destroys the ear ossicles so that hearing loss can be severe.

Erosion and infection may spread:
(1) into the labyrinth (29-2), causing vertigo,
(2) through the roof of the middle ear, causing an extradural or subdural abscess in the temporal lobe, or in the posterior cranial fossa,
(3) into the lateral cerebral venous sinus (29-4), causing thrombosis, high fever, and maybe death (29.5),
(4) to the meninges; this is uncommon because infection is usually well localized,
(5) to involve the facial nerve.
All these complications need at least a mastoidotomy to deal with the underlying sepsis, perhaps with a tympanoplasty, to preserve hearing, and perhaps life.

DIAGNOSIS
Look for a perforated eardrum (29-3) which discharges continuously or intermittently, in a patient who may or may not give a history of a previous acute attack. Look carefully at the perforation. Is it surrounded by eardrum? This may be difficult to work out since the perforation may be large and the surrounding eardrum may be scarred and be calcified (**tympanosclerosis**, 29-3N). However, if you do see the edge of the eardrum all around the perforation, then this must for that reason be centrally placed.

Is the perforation at the edge of the eardrum? Is it in the roof of the ear? Does it extend to the back of the eardrum? Such perforations may be small but dangerous. Look for the white, thick, pasty, pearl-like material of cholesteatoma. If present, then the disease will progress.

The prognosis, and the urgency of definitive treatment, depend on where the perforation is in the eardrum, rather than on how big it is.

If the perforation does not extend to the edge of the eardrum, and does not involve its *pars flaccida* (the superior part of the eardrum), it is central (29-3C) and is unlikely to be dangerous. There is increasing deafness, recurrent discharge, and occasionally earache, but pain is rare. Teach the importance of a careful aural toilet.

If the perforation extends to the edge of the eardrum, and particularly if it involves the *pars flaccida* (29-3N), it is peripheral and dangerous, because it implies bone destruction. A cholesteatoma is common.

TREATMENT
Mop out the ear canal, and try to see the perforation. If there is much discharge, rinse out the ear with warm sterile water; mop the ear dry, and you will then be able to examine it.

You can syringe a discharging ear, *but it is probably wise not to syringe one with a cholesteatoma*. Try to keep the ear mopped dry with cotton wool, in the hope that the perforation in the eardrum will heal.

DIFFICULTIES WITH CHRONIC OTITIS MEDIA
Although the definitive treatment is a radical mastoidectomy, this is difficult and delicate surgery. Mastoidotomy (29.5) will buy you time for the following situations:

If there is a tender swelling over the mastoid, this is ACUTE-ON-CHRONIC MASTOIDITIS.

If there is EARACHE, this is ominous. Pus is gathering under pressure somewhere and, unless released, may track internally, with serious results.

If there is severe vertigo and loss of balance, perhaps with vomiting, this is LABYRINTHITIS. Symptoms are worse on moving the head. There is usually also increased hearing loss. Look for a fine horizontal nystagmus, and see if this is made worse when you close the ear canal with your finger, and gently press it. Use ampicillin in high doses, with chloramphenicol IV and metronidazole PR. There is a danger of meningitis if infection does not settle. Try then to drain the mastoid cortex.

If there is severe illness, headache, vomiting, fever, **with neck stiffness, photophobia and restlessness**, this is MENINGITIS. A +ve Kernig's and Brudzinski sign will be

present. Confirm the diagnosis by lumbar puncture. Examine the CSF by Gram's method, and culture it, before immediately starting antibiotics. Use ampicillin in high doses, with chloramphenicol IV and metronidazole PR. When the meningitis has settled, arrange a mastoidotomy (29-4).

If there is hemiparesis, cerebellar signs, dysphasia or depressed level of consciousness, the condition is serious, because the infection has spread to the brain. The presence of pyramidal signs (spasticity and upgoing toes) suggests a poor prognosis. Start ampicillin in high doses with chloramphenicol IV and metronidazole PR, prior to drainage of a brain abscess, possibly extradural (6.5). Check the HIV status.

If there is bilateral discharge from the ears, treat the side with pain first.

If there is fever, antibiotics alone will cure none of the complications, but always start them.

If there are fever, rigors, headache, and photophobia with ophthalmoplegia, suspect LATERAL SINUS THROMBOSIS. Use high doses of broad-spectrum antibiotic IV. Check the HIV status.

If permanent deafness develops as the result of bilateral chronic otitis media, supply a hearing aid and arrange proper follow-up.

If facial palsy develops, a cholesteatoma is invading the facial nerve. Try to arrange a radical mastoidectomy.

29.5 Acute mastoiditis

Acute mastoiditis is typically a disease of children, and may complicate neglected acute or chronic otitis media. It is rare where primary care is good. In babies, it occasionally presents as a swelling over the mastoid process.

If acute mastoiditis complicates acute otitis media (uncommon), the child continues to have fever, and the ear continues to discharge pus in increasing quantity through a perforation in the eardrum.

If acute mastoiditis complicates chronic otitis media, there is:
(1) A dull nagging pain; this may either be new, or an increase in chronic pain.
(2) Increasing discharge; if the discharge is chronic anyway, it is usually difficult to quantify.
(3) Increasing deafness; again the change may be subtle.
(4) Tenderness over the process.
(5) Oedema of the skin over the mastoid process, owing to underlying infection, giving it a 'velvety' feel.
(6) Swelling in the postero-superior wall of the meatus.
(7) Anterior rotation of the pinna, so the ear sticks out more on the affected side than normal. This is a very characteristic sign, and should make you suspect the diagnosis; it can however also be caused by a swollen postauricular lymph node, by a meatal pustule, or by cellulitis of the scalp.

If pus has gathered under the periosteum, simply open this and drain the pus, or later it may be necessary to open the mastoid cortex to allow the pus to drain.

The more radical operation of mastoidectomy, to remove all the cortex overlying the mastoid sinus, and saucerize the opening, is a highly complex operation because of the close proximity of important structures *with the danger of serious damage to hearing, the facial nerve and the brain.*

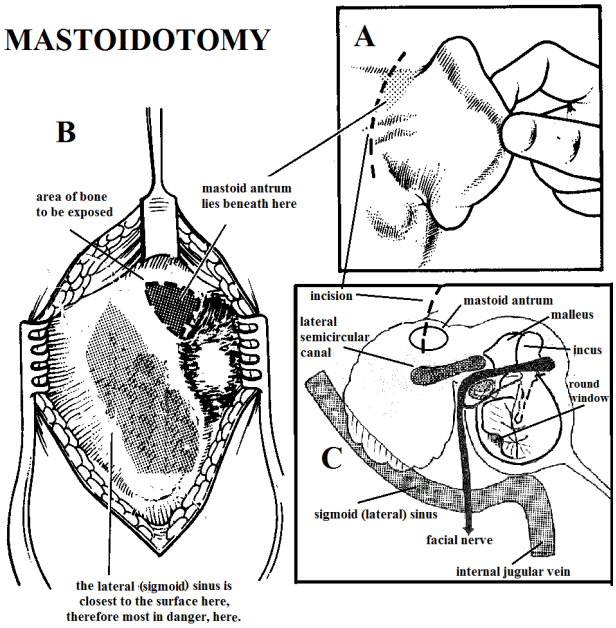


Fig. 29-4 DRAINING THE MASTOID.
A, retro-auricular incision. B,C, position of mastoid sinus and lateral (sigmoid) cerebral venous sinus. *Take great care not to damage this!*

(a) Differential diagnosis

Suggesting mastoiditis: no pain on pulling the ear. Pain on deep pressure over the *upper* part of the mastoid at 11o'clock in relation to the right external auditory meatus. *Don't test for tenderness over the tip of the mastoid*. A profuse mucopurulent discharge, a swelling on the inner bony part of the meatus at 11 or 12o'clock, marked middle ear (conductive) deafness, and cloudy mastoid air cells on radiographs.

CAUTION! The mastoid is always tender during the first few days of an attack of otitis media, before the eardrum has burst.

Suggesting postauricular lymphadenitis and swelling of the tissues round it: some septic lesion on the scalp or neck, particularly infected ringworm or impetigo, or following lice in the scalp; the pinna may be pushed forwards; no discharge or deafness, a normal eardrum. Swollen lymph nodes are usually at 8 or 9o'clock in relation to the right ear, whereas the swelling of mastoiditis is maximal at about 11o'clock.

Suggesting a pustule (furuncle) **in the external auditory meatus** (29.3, rare in a child): swelling in the outer cartilaginous part of the external auditory meatus, and the mastoid is *not* tender. Hearing becomes normal by pulling the pinna upwards and backwards. There is pain on pulling the ear and on chewing, a history of other abscesses, and a small thick discharge. The eardrum looks normal, although you may have difficulty seeing it. Radiographs show a normal mastoid.

(b) Mastoidotomy for acute mastoiditis (GRADE 2.5)
Incise behind the ear, retract the *temporalis* muscle superiorly and the *sternomastoid* anteriorly; open the periosteum (29-4A): there may be pus under it. If there is, *stop there!* If not, keeping superiorly and anteriorly *to avoid the lateral sinus*, use a gouge (7-5) to open the cortex of the bone for about 1cm, over the mastoid sinus, and expose some of the mastoid air cells (29-4B), which will be full of pus. Insert a drain and administer chloramphenicol with metronidazole. *You don't usually need to do a cortical mastoidectomy*: this is difficult surgery, and *you might damage the auditory canal, the lateral sinus and cause uncontrollable bleeding, or the facial nerve.* Be content with draining the mastoid cavity. *This could be life-saving!*

29.6 Foreign bodies in the ear

Foreign bodies in the ear are more difficult and dangerous to remove than those in the nose; the dangers include a perforated eardrum, total deafness, and a facial palsy, or all three. The middle (isthmus, 29-2) of the auditory canal is narrower than either its outer or its inner end.

If a foreign body is impacted outside the isthmus, removing it should not be difficult. *Always try syringing first*. If this fails and you have to use instruments, be sure to use GA or ketamine, especially in a child. The foreign body may be a seed, a live insect, a piece of paper or a broken matchstick. Most (70%) patients are children <5yrs.

(a) Removal of a foreign body in the ear (GRADE 1.2)

First try to syringe the ear, in a young child under ketamine, as if you were removing wax. Use a 20ml syringe, or an ear syringe, containing warm water at body temperature (37ºC). Attach a soft plastic cannula and cut off the end leaving c.1cm length. Pull the pinna upwards and backwards, and direct the stream of water up along the roof of the auditory canal, so that it gets behind the foreign body and pushes it out. Syringing will remove most foreign bodies.

N.B. Never direct the jet of fluid directly at the eardrum!

If syringing fails (rare), use GA. Ketamine is ideal. Extracting a foreign body is seldom urgent, so you have time to prepare. Lay the patient down, and use a headlamp or, better, an auroscope with an open lens, and aural speculum. Rest your hand on the patient's head.

Try gentle suction with a piece of soft rubber tubing on the end of the sucker. If this too fails, try using suction with the syringe.

Use an aural hook, a cerumen or crochet hook, or a paper clip bent as shown (29-5), held in mosquito forceps (3-3). Put the hook into the auditory canal, so that it lies against the wall.

REMOVING A FOREIGN BODY FROM THE EAR

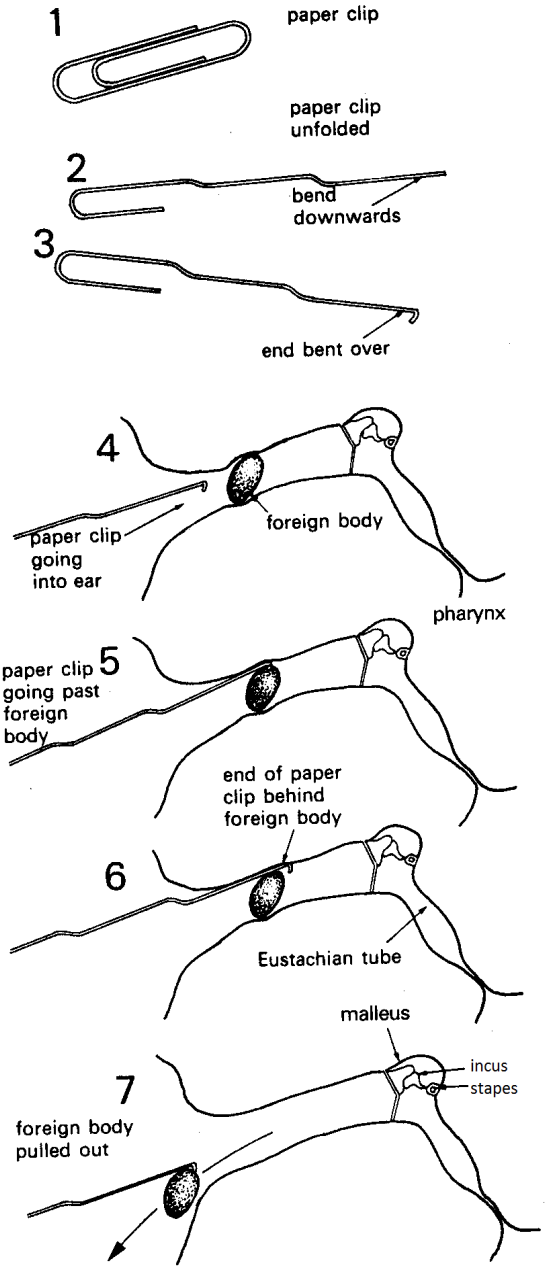


Fig. 29-5 REMOVING A FOREIGN BODY FROM THE EAR.
Lie the patient down with the affected ear uppermost. Start by trying to syringe it out. If this fails, use GA or ketamine. Make sure you have a good light. 1,2,3, if you don't have the proper hook, bend a paper clip. Smooth its ends with a file or stone. Bend it exactly as shown and hold it in mosquito forceps. *Take special care not to rupture the eardrum.* 4, gently introduce the hook into the auditory canal, along its superior wall, and 5,6, edge the end of the hook past the foreign body. *Don't do this if it is wedged against the eardrum!* If you can hook the foreign body, 7, gently ease it out, if necessary with more syringing.

Then, manoeuvre the hook past the foreign body; twist it, so that it now lies behind the foreign body, and allows you to pull it out. *Check for any more foreign*

bodies and check for damage to the tympanic membrane (eardrum)!

CAUTION! Be very gentle:
(1) *don't push the foreign body beyond the isthmus of the auditory canal*,
(2) *don't damage the tympanic membrane*,
(3) *don't try to use dissecting forceps*.

MOHAMMED (10yrs), the son of a local VIP, was admitted with a ball-bearing in the ear. The consultant ENT surgeon was on leave, and so a junior took over the case. It seemed a pity to give the child a GA, and as he seemed cooperative, the medic decided to remove the ball-bearing with a wax hook. Unfortunately, after two unsuccessful attempts, during which the ball-bearing was driven deeper in, some bleeding began, which rather obscured the view, but the ball-bearing was eventually removed. However, in the blood clot were found the remains of the malleus, the incus, and the stapes.
LESSONS (1) If you are inexperienced, simpler methods may be safer, even if they are less dramatic. Syringing is not sophisticated, but it is 'brilliant' compared with inadvertent stapedectomy. (2) The less experienced you are, the more necessary it is to remove a foreign body under general anaesthesia. A struggling child is no subject for delicate surgery. (3) *Don't try removing a round metal object with a hook!* Using a magnet may work!

(b) Difficulties with foreign bodies in the ear

If the tympanic membrane is ruptured, try to prevent infection, and let it heal spontaneously. Keep the ear completely dry for 6wks. *Don't dust it with antibiotics, or pack the canal*. Mopping is unnecessary, unless the middle ear discharges; if so, treat as for otitis media.

N.B. Direct trauma may rupture an eardrum: unskilled attempts to remove a foreign body, an explosion or blow, or penetration with a sharp object.

CAUTION! Don't syringe a ruptured eardrum, and do as few manipulations as possible.

If there is an insect in the ear, put a few drops of oil and lidocaine in the ear to kill it, then syringe it out.

If a vegetable foreign body swells, and jams itself tightly in the canal, leave it and try again later, initially with syringing. You can try using warm diluted hydrogen peroxide.

If the foreign body has passed beyond the isthmus, so that you cannot safely remove it with a hook, try syringing again at least twice. *Remember this is not an emergency*.

OPEN EXTRACTION OF AN AURAL FOREIGN BODY
If the foreign body remains *in situ*, make a small vertical incision from the back of the pinna at its attachment to the mastoid, through into the auditory canal. Control bleeding with small clips or diathermy.

Hold the pinna forwards, and remove the foreign body under direct vision. You must have suction or else bleeding will totally obscure the view. The incision is not deep but it must demonstrate the auditory canal. Inspect the eardrum, remove the foreign body and close the incision with 2 monofilament sutures. Then pack the canal with ribbon gauze to prevent oedema and granulations. If possible, impregnate the gauze with BIPP (4.11). Remove the pack in 3 days.

29.7 Epistaxis (Nose bleeding)

Nose bleeds are rare before 2yrs, common in childhood, uncommon in young adults, and more common again in the elderly. You can control them easily in most cases.

Most bleeding from the nose is from the anterior nasal septal vessels (Little's area or Kiesselbach's plexus, 29-6F); by pressing on the soft, cartilaginous part of the nose, it usually stops. You can see the bleeding point with a nasal speculum and a good light (try the sun) behind you; you will see the vessels better with a headlamp. When bleeding comes from anywhere else, it usually comes from far back in the nose. This usually occurs in the elderly and hypertensive, and may present as haemoptysis or haematemesis, or even just nausea and anaemia.

Start with the simpler methods first. If you teach these to your nurses and auxiliaries, they will be able to treat most patients. You will need suction, and if possible a headlight and BIPP (4.11).

CAUTION! Epistaxis can, in rare cases, be caused by a cancer or nasal tumour. If you examine the nose in a patient with epistaxis and see an abnormal mass, consider it a cancer until proven otherwise. Other signs that suggest a cancer are neck nodes and middle ear effusion. A very rare but serious cause of epistaxis in male children is a haemangiofibroma. For some reason, this occurs only in boys and blocks the back of the nose.

(a) Examination
Clear the airway if bleeding is profuse.
Get IV access. Sit the patient upright looking straight ahead. Ask an assistant to stand behind him, and hold the head. If there is bleeding from the anterior half of the nasal cavity, most of the blood will come from the nostrils. If there is bleeding from the posterior half, much of it will be trickling down the pharynx.
If the blood does not form clots, check its clotting time.
A child is almost certainly bleeding from the anterior septal vessels; so are most adults. In the remaining cases, the bleeding is posterior, and is occasionally caused by a systemic disease.

(b) Differential diagnosis
Did the blood come first from the front or the back of the nose? Which side? First time or recurrent? Medications taken? Trauma or foreign body? Apart from obvious hypertension, there is usually no time to speculate on the cause.

Other causes are: tumours, leukaemia, scurvy, purpura, onyalai (haemorrhagic bullae in the oropharynx with thrombocytopenia, seen in Central Africa) and the prodromal stages of diphtheria, measles, varicella, and scarlet fever. All these causes are rare.

(c) Immediate treatment
Sit the patient forwards a little, drape him in a waterproof cloth, and hold his nose over a receiver. Tell him not to

swallow the blood, but to spit it out. *Avoid a stomach full of blood!* If necessary, use the recovery position. Get suction ready.

(d) Bleeding from the front of the nose (where it is usually from the anterior part of the nasal septum): Squeeze the nose, so that you press its soft mobile parts against the septum, while he breathes with the mouth wide open. Do this yourself, or delegate a nurse to do it. If bleeding is more than minimal, keep pressing for 5mins by the clock. If it is minimal, ask the patient to do it himself. If necessary, sedate him. If squeezing fails, try it again. If you wait long enough, the bleeding will usually stop, and you will have done nothing to damage the mucosa.

If the bleeding does not stop with simple pressure, take some cotton wool soaked in a vasoconstrictor (such as diluted 1:100,000 adrenaline) and place this up against the bleeding area and again press.

If adrenaline-soaked cotton wool does not stop the bleeding, consider cautery. This can be done by silver nitrate sticks or by electrocautery. *Be careful with cautery not to make too deep a burn* and thereby cause a hole in the nasal septum. *Don't cauterize a nasal septum on both sides at the same time*, for the same reason that it is possible to cause a septal perforation.

If the bleeding still does not stop, then treat as for posterior nasal bleeding.

ANAESTHESIA

All packing, intranasal manipulation or cauterization needs *at least* LA, either by spray or on a gauze or wool swab wet with 4% lidocaine. Use ketamine or GA for simultaneous anterior and posterior packing.

ANTERIOR PACKING OF THE NOSE (GRADE 1.1)

You will need a headlamp, or head mirror with a good light shining on to it from behind your shoulder, a nasal speculum, and dressing forceps, preferably Tilley's. For each side of the nose you will need 1m of 13mm gauze packing, or a 13mm gauze roll. To make this easier to remove later, smear it with petroleum jelly or BIPP (4.11). If you lack BIPP, use 1:100,000 adrenaline solution.

Pack first the nostril which is bleeding most. With the patient sitting upright, ask an assistant to stand behind and hold his head. Warn him that this procedure may be very uncomfortable. Clear the nasal cavities by encouraging blowing of the nose, or clear the bleeding nasal cavity with a sucker and cannula. Your previously applied lidocaine pack should have produced some anaesthesia.

Focus your light on the speculum, and put it into the bleeding nostril. Grasp the end of the gauze with forceps and place it as high and as far back as you can. Try to pack the nasal cavity in an orderly way in horizontal layers, starting on its floor and working towards its roof (29-6A). This is difficult, and you will probably find yourself putting gauze wherever it will go, until the nose is full. Leave both ends of the gauze protruding from the nostrils.

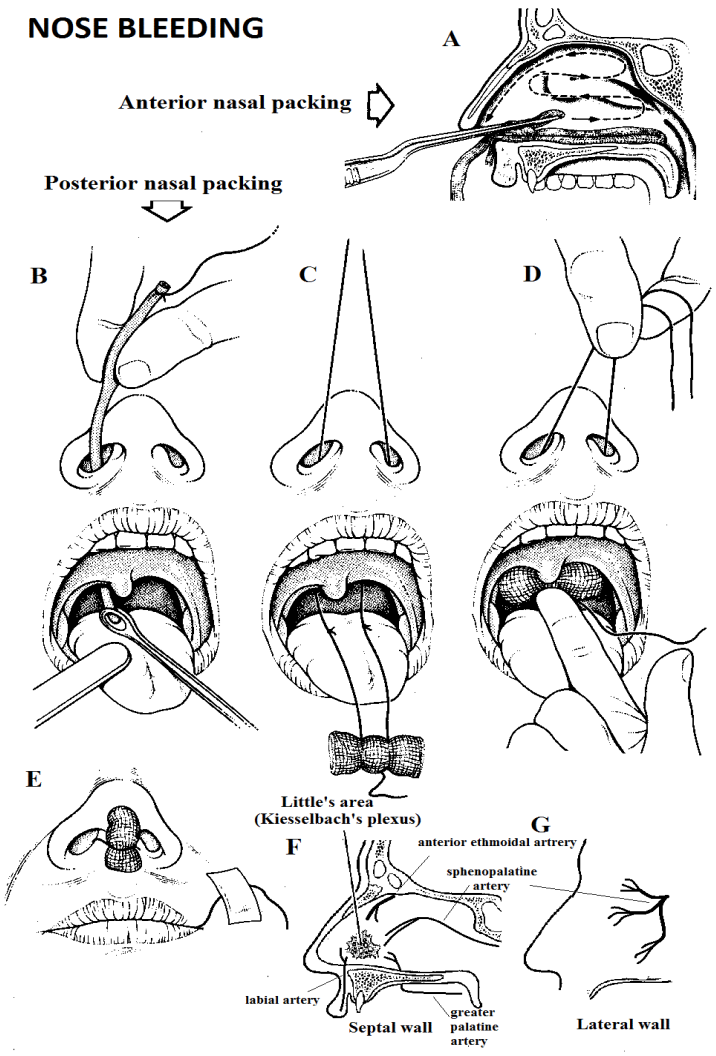


Fig. 29-6 EPISTAXIS.
A, insert an anterior nasal pack. B-E, inserting a posterior nasal pack. B, passing a catheter. C, gauze roll ready to be pulled into place. D, push the gauze roll into place. E, tie a gauze roll in place. F, vessels in Little's area (Kiesselbach's plexus) from the anterior ethmoidal, labial, greater palatine and sphenopalatine arteries are the source of the bleeding in 50%. G, bleeding from the lateral wall is mainly from the sphenopalatine artery.
After Loré JM, An Atlas of Head and Neck Surgery, Saunders Plate 51, with kind permission.

If necessary, pack both sides of the nose, and secure all 4 ends of the gauze with a safety pin. Strap a pad of folded gauze across the front of the nose, and wait 5mins.

If an anterior pack controls bleeding, leave it in place for 48h. Then gently remove it, preferably early in the day, so that you can more easily repack the nose if necessary. Observe carefully for 24h before discharge.

If an anterior pack does not control bleeding, try CAUTERIZATION (optional). Soak a small piece of ribbon gauze in 4% lidocaine and 1:100,000 adrenaline solution, squeeze out the excess, and apply this to the bleeding area for 10mins, or use a local anaesthetic spray. Use a nasal speculum, or a wide-bore aural speculum, and a good light, to find the bleeding vessels in Little's area. Touch them along their course with an applicator that has had a bead of silver nitrate fused to its tip: the mucosa will turn white.

If you fail to control bleeding, reinsert the lidocaine and adrenaline pack. If this too fails, hold a silver nitrate stick over the bleeding area for 5secs, and then roll it away to one side before you remove it (if you pull it off, bleeding may restart). *Don't use this in both nostrils at the same time,* as septal perforation may result.

If you fail again, try a galvano-cautery with a hot wire loop. If necessary, use any thin wire heated in a spirit lamp. Gently touch the bleeding area. You can also use diathermy, preferably under GA. Leave the scab, and dress it with petroleum jelly.

CAUTION! Don't cauterize both sides of the nasal septum at one time with silver nitrate or heat, because it may perforate.

(e) Bleeding from the back of the nose (*i.e.* you cannot see the source of bleeding): Infuse IV saline, cross-match blood, and administer pethidine (or sedation) 50-100mg IM, or slowly IV to facilitate your manipulations. Administer 1g tranexamic acid tds orally. Proceed with anterior packing.

If anterior packing and cautery does not control bleeding, remove the pack, insert a posterior one, and then repack the anterior nasal cavity again.

POSTERIOR PACKING OF THE NOSE (GRADE 1.2) may be necessary if:
(1) An anterior pack fails to control anterior bleeding.
(2) There is severe posterior bleeding.

Spray the pharynx and palate with 4% lidocaine. Try using a Foley catheter (often very effective). Start with this. Pass a size Ch12 or 14 Foley catheter, with a reasonably sized balloon, gently through the anaesthetized nostril, until you see its tip just behind the soft palate. Inflate the balloon with air (usual maximum 20ml), and gently withdraw the catheter, so that the balloon impacts in the posterior nasal opening. Tape it to the cheek, then pack the nose from in front as described above.

CAUTION!
(1) *Don't inflate the balloon inside the nasal cavity*, because this can quickly cause pressure necrosis of the mucosa, which may make bleeding worse.
(2) The tube of the catheter can ulcerate the rim of the nasal entrance, so spread out the pressure by putting a little gauze pad under it.

If posterior combined with anterior packing and cautery does not control bleeding, pack the nose under GA. Use a pack of folded or rolled gauze sponges of sufficient bulk to plug the posterior nares. You will need 2 packs of at least 5cm² for an adult. Tie 50cm of soft string, or umbilical tape, to a Ch16 or 18 rubber catheter (29-6B). Put this into one nostril, and pull it out of the mouth, leaving the string in place.

Do the same thing on the other side. Tie the oral ends of the strings to the pack, and tie a 3rd piece of string to it. Pull the pack up into the back of the nose, and press it into place with your finger in the throat. Make sure that it has passed behind the soft palate, *and that this has not folded upwards*. Then pack the anterior nasal cavity, as before.

Tie the nasal ends of the string over some gauze. Let the 3rd string protrude from the corner of the mouth, and tape it to the cheek. Or keep it in place with a plastic umbilical cord clamp.

CAUTION!
(1) Insert packs with great gentleness: you can easily cause more bleeding as you insert them.
(2) Withdraw the packs, or the Foley catheter, slowly after 48h. *Don't leave any pack or catheter, either anterior or posterior, in the nose for longer than this*, or you will increase the risk of suppuration, especially in the sinuses. The only possible exception is a pack impregnated with BIPP (4.11), which you can leave for 1wk. If you are using a Foley, deflate it a little first to see if bleeding is controlled.
(3) Remove a pack slowly, bit by bit.

When you remove a posterior pack, do so in theatre under ketamine, with a light and the necessary equipment ready, so that you can, if necessary, repack without delay.

Because epistaxis may recur when you allow a patient home, make sure he knows how to hold his nose, to breathe through his mouth, and to sit forwards in the correct position.

(f) General measures for epistaxis
Try to estimate how much blood has been lost. If there is severe bleeding, infuse IV saline and cross-match blood. Keep the head propped up. Use paracetamol, *not aspirin*, as this reduces platelet aggregation. Pethidine or morphine can be helpful. Monitor blood pressure, respiration, and the Hb. Most severe epistaxis is precipitated by infection, so use ampicillin or chloramphenicol and metronidazole for at least 5 days. With hypertension, bleeding may be difficult to stop unless you control the blood pressure: nifedipine is useful.

(g) Difficulties with epistaxis
If there is sudden pallor with shock whilst you are packing the nose, suspect a vasovagal attack, especially if there is a bradycardia. Put up IV saline and administer atropine.

If there is persistent bleeding, look for petechiae, ecchymoses, and a large spleen. Measure the clotting and bleeding times, the prothrombin index, and the blood urea. There may be leukaemia, thrombocytopenia, or other clotting disorder. *Bleeding may prove fatal.*

If you have properly packed the nose and it continues to bleed whenever the packs are removed, the ultimate measure is to tie the anterior ethmoidal arteries in the medial wall of the orbit.

If the bleeding is arterial and *not arising from Little's area*, it arises from the sphenopalatine artery (29-6G), a branch of the maxillary which arises from the external carotid. Clipping the internal maxillary artery behind the posterior wall of the maxillary sinus is an alternative, but unless you have experience in dissection, leave this operation for experts.

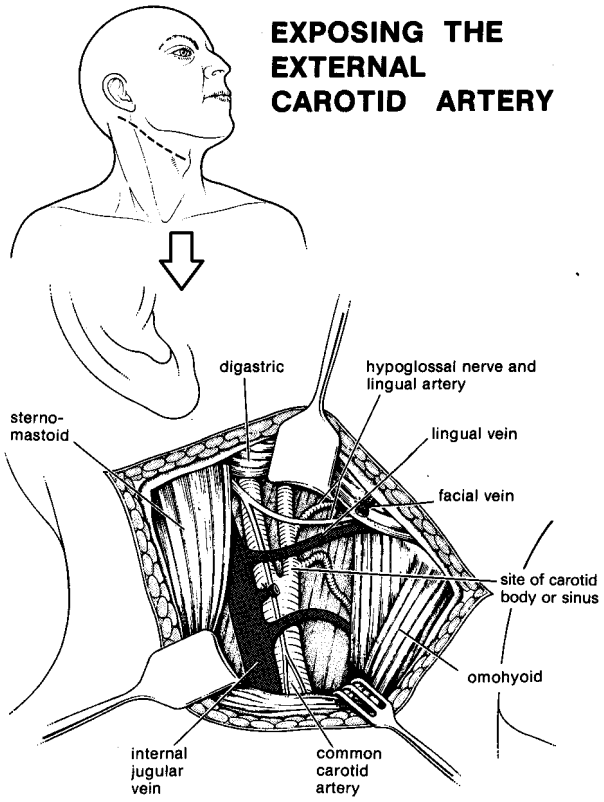


Fig. 29-7 EXPOSING THE RIGHT EXTERNAL CAROTID ARTERY.
Exposure and anatomy.
Adapted from Rintoul RF (ed) Farquharson's Textbook of Operative surgery. Churchill Livingstone, 1978 with kind permission.

EXTERNAL CAROTID ARTERY LIGATION (GRADE 3.2)

The external carotid artery via its maxillary branch supplies ½ the blood supply of the nose. Ligating the external carotid artery in the neck can help epistaxis.

This can be done under LA. It is essential, however, that you ligate the external carotid, *not the internal carotid*. The external carotid artery is recognized by having branches. It arises from the common carotid artery at the upper edge of the thyroid cartilage. It runs upwards behind the neck of the mandible, and ends by dividing into the maxillary and superficial temporal arteries. It lies under the posterior belly of the *digastric* muscle (29-7), and its upper part lies deep to the parotid gland.

Tilt the table 10º head up to minimize venous bleeding, but not more, because this increases the risk of air embolism. Turn the patient's head to the opposite side, and extend it slightly. Make an oblique incision from just below and in front of the mastoid process, almost to the thyroid cartilage.

Divide the *platysma* and deep fascia in the line of the incision, and dissect flaps upwards and downwards. Free the anterior border of the *sternomastoid* and retract it posteriorly. You will see the common facial vein. Divide this between ligatures. Carefully retract the internal jugular vein backwards, in order to see the common carotid artery bifurcating to form the internal and external carotid arteries.

If you have difficulty in deciding which artery is which, find some branches of the external carotid and follow them backwards to the main stem (*the internal carotid artery has no branches in the neck*). Pass an aneurysm needle round it, tie it with zero non-absorbable, but *don't divide it*. Tie it as close to its origin as you can.

CAUTION!

(1) Tie the external carotid just proximal to the origin of the lingual artery.

(2) *Avoid the hypoglossal nerve*, which crosses the external and internal carotid vessels and then runs anteriorly to lie on the *hyoglossus* muscle in company with the lingual vein.

(3) *Avoid irritating the carotid sinus and body* in the bifurcation of the internal and external carotid vessels, because this may trigger profound bradycardia.

29.8 Rhinosinusitis

The paranasal sinuses lead off the nose, so that disease in them usually follows disease in the nose itself. Sinusitis has some common features, regardless of which particular sinus is infected. The common presenting symptoms in the nose are:

(1) discharge,
(2) obstruction of the nasal airway,
(3) facial discomfort or pain.

(a) Acute sinusitis often follows a viral upper respiratory infection, and usually involves one of the sinuses only. It may follow a dental infection.

Presentation is with fever, copious purulent discharge, and:
(1) pain, or a sense of pressure in the cheek (sometimes wrongly thought to be 'toothache'),
(2) obstruction of the nasal airway, often without the discharge of mucus or pus,
(3) swelling of the face (this is much more likely to be due to a dental abscess, 6.9). *Tenderness over an infected sinus is not a useful sign.*

TREATMENT

No treatment may be necessary. Most cases of upper respiratory infection are viral and resolve spontaneously after 2-3wks, and require only analgesia.

If there is fever for more than a few days, or if there is severe facial pain, then consider:
(1) broad-spectrum oral antibiotics,
(2) nasal decongestants; but *don't use these for >5 days*. If you do, the nasal mucosa becomes accustomed to them and when you stop them, there is a rebound effect, which results in swelling of the nasal lining resulting in a blocked nose!

(b) Chronic sinusitis may follow acute sinusitis, or as a result of nasal polyps, which prevent the sinuses draining. Pain is not a major feature, but there may be a dull ache in the face, usually later in the day. Bending the head forward can be uncomfortable. Distinguish this from hypertension.

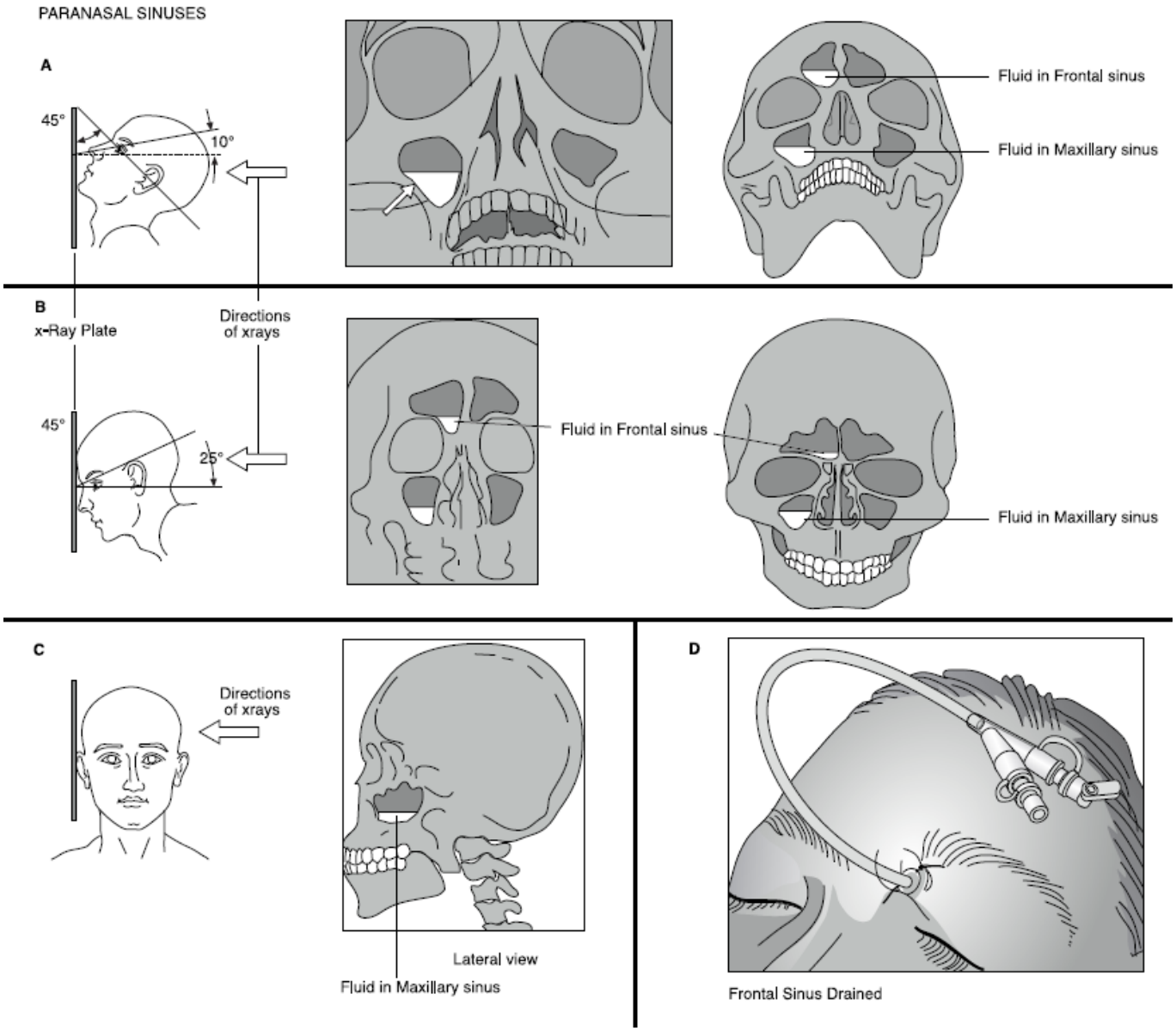


Fig. 29-8 IMAGES OF THE PARANASAL SINUSES.
A, occipito-mental (Waters/Blondeau) view (best for the maxillary sinus) showing fluid levels in maxillary and frontal sinuses. B, occipito-frontal (Caldwell/Worms) view (best for the frontal sinus) showing the same fluid levels. C, lateral view showing fluid in the maxillary sinus. D, frontal sinus drain using a Ch8 Foley catheter through a 3mm endotracheal tube.
After Ismail H, Bowden J, Hellier WP, et al. A frontal sinus drain. Ann R Coll Surg Engl 2004; 86: 306-7.

TREATMENT

Chronic rhinosinusitis is defined as disease lasting for >6wks. Examine the nose to look for nasal polyps, a deviated nasal septum or, rarely, a tumour. Use:
(1) broad-spectrum antibiotics for 2-3wks,
(2) saline nasal douching: washing the nose out with salt water (this can be made at home using 1L of clean water with 3 teaspoons of salt), either sniffed up from the palm of the hand or sprayed into the nose using a 20ml syringe,
(3) steroid nasal sprays: these sometimes help, since they dampen down the swelling and inflammation in the nose.

(c) Difficulties with rhinosinusitis

If swelling around the eyes develops with fever, this is ORBITAL CELLULITIS (6.6, 28.11). This is serious: start IV cloxacillin or chloramphenicol, and add nasal decongestant drops (ephedrine 0·5%).

CAUTION! Chronic use of vaso-constrictive or cocaine nasal sprays can lead to septal perforation.

Watch for development of a **subperiosteal abscess**, which needs draining.

If there is localized pain above the eye, suspect FRONTAL SINUSITIS. Later, gross orbital swelling, proptosis, and diplopia may develop. If large doses of IV ampicillin, chloramphenicol and metronidazole don't control symptoms rapidly, you may have to drain the frontal sinus. Frontal sinusitis is always secondary to maxillary sinusitis and obstruction of the fronto-nasal duct, so be sure to wash out the maxillary sinus also. Infection may spread to the frontal bone, causing FRONTAL OSTEITIS.

Sometimes the pus may break through the anterior skull wall of the frontal sinus and under the skin, leading to swelling of the tissues of the forehead and an abscess under the skin. This is known as a 'Pott's puffy tumour'. It requires incision and drainage of both the subcutaneous abscess and the frontal sinus. Infection may also lead to meningitis or a frontal extradural or intracerebral abscess.

If there is ophthalmoplegia, proptosis and diminished consciousness, this is a CAVERNOUS SINUS THROMBOSIS. It will probably involve both eyes. Early vigorous treatment may avoid death. Use high-dose IV penicillin with chloramphenicol or a cefalosporin, *together with diuretics* (furosemide or mannitol) *to reduce cerebral oedema.*

If a facial cyst-like swelling grows, displacing the eye, this is a SINUS MUCOCOELE, which is a late complication arising from either the frontal or ethmoid sinus. Drainage of the sinus is blocked by scarring from previous infection; the sinus lining continues to secrete mucus and produces the cyst.

(d) Drainage of sinuses

If you have a thin endoscope, it is possible to wash out and drain the sinuses elegantly; however, you can still do this without such equipment.

(1) RADIOGRAPHS

An erect plain radiograph will show an opacified sinus or a fluid level within the sinus. Use the occipito-mental (Waters/Blondeau) view for the maxillary sinuses (29-8A), and the occipito-frontal (Caldwell/Worms) view for the frontal sinuses (29-8B), and a lateral view for both to make sure. If you are in doubt, reposition the head and take another radiograph to see if the fluid level shifts.

(2) DRAINING THE FRONTAL SINUS (GRADE 1.5)

Infiltrate with 2% lidocaine mixed with 1:100,000 adrenaline and incise 1cm lateral to the midline along a line level with the upper limit of the orbit.

Use a dental drill to cut a hole in the floor of the frontal sinus, and insert a cannula attached to a syringe half-filled with saline.

Aspirate: you should see air bubbling out together with some purulent fluid. Insert a small drain. The best type is a 3mm endotracheal tube through which you have passed a Ch8 paediatric Foley catheter. This arrangement allows you to syringe out the sinus through the Foley catheter (29-8D) and the fluid can drain along its side out of the outside tube. Also, wash out the maxillary sinus through a cannula inserted through the inferior meatus.

Definitive surgery with removal of the anterior wall of the frontal sinus and its entire lining (7.13) may be necessary later.

CAUTION! Don't do this if the air cavity of the frontal sinus is situated much higher than the level of the orbit.

N.B. Remember to wash out the maxillary sinus of all patients with frontal sinus infections, because this is often the primary source of infection.

(3) DRAINING THE MAXILLARY SINUS (CALDWELL-LUC OPERATION) (GRADE 1.4)

If there is increasing pain in the cheek against a background history of rhinosinusitis, suspect maxillary sinusitis. If there is chronic repeated infection or a fluid level on a radiograph (29-8C), this needs draining.

MAXILLARY SINUSOTOMY

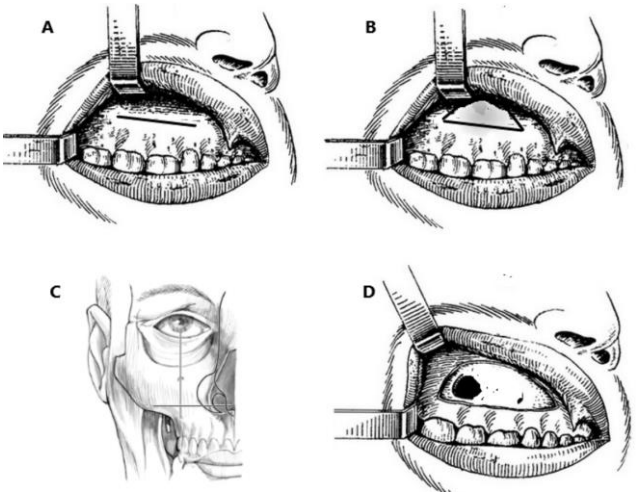


Fig. 29-9 CALDWELL-LUC MAXILLARY SINUSOTOMY.
A, make an incision above the canine to premolar teeth. B, lift a muco-periosteal flap. C, calculate the spot for the osteotomy (directly below the middle of the eye and level with the base of the nostril). D, make an osteotomy at this site.

Obtain good LA, *especially under where the trocar will penetrate; wait at least 10mins.* You may need to add sedation with ketamine also.

Infiltrate the submucosa above the canine and premolar teeth with lidocaine 2% mixed with 1:100,000 adrenaline, and apply it locally on a gauze, or as a spray.

Make an incision 1cm above the teeth from canine to 2nd premolar, and raise a mucoperiosteal flap. Use an 8mm burr (or a fine trocar) and gently make a hole at the point where a line from the middle of the eye and the base of the nostril cross.

As soon as you feel a 'give' into the sinus, stop advancing, and check that you are within the sinus. You should be able to aspirate either air, blood or pus. Try to empty the sinus, and then gently rinse it with warm saline until there is free flow of saline into the nasal cavity.

If the ostium is sufficiently blocked to prevent the water draining (rare), enlarge the hole with a bone nibbler.

CAUTION!

(1) *Don't push right through the maxillary sinus into the mouth!* You should be able to wiggle the tip of the trocar slightly when it is inside the sinus.

(2) Keep the syringe full of saline. *Don't blow air in if the ostium is blocked*; this may push air into cerebral veins and prove fatal!

29.9 Nasal obstruction

Typical signs are snoring, nasal obstructed speech, rhinorrhoea and secondary sore throats.

N.B. Look out for secretory otitis media (29.4)!

Nasal obstruction can be due to:

(1) Swollen nasal turbinates (29-13) due to allergic rhinitis. The patient often has asthma as well. The turbinates tend to be swollen and pale.

(2) Swollen nasal turbinates due to occupational exposure to wood dust, latex, animals, grains or mould. The nose often feels dry and looks crusted.

(3) Swollen nasal turbinates due to idiopathic rhinitis. The turbinates look swollen and reddish.

(4) Deviated nasal septum: the nasal septum is not in the midline and is bent or twisted. There may also be an external deformity of the nose. This may be idiopathic or traumatic.

(5) Nasal polyps (29.10).

(6) Rhinosinusitis (29.8).

(7) Nasal tumours, rare but should not be missed.

(8) Septal haematoma. Following injury to the nose, the nasal septum may swell up due to haemorrhage under the perichondrium. The swelling is soft. Infection can then lead to a septal abscess.

Don't operate for nasal obstruction unless you have some experience; even then, only when simple methods fail; otherwise the outcome may be of no benefit.

(a) Electrocautery for swollen nasal turbinates (GRADE 1.3)

Take care not to cauterize both the turbinate and the nasal septum, which could then result in adhesions.

Also be careful not to cauterize too deep or too far posterior since by doing so, there is a risk of serious bleeding. If there is excessive bleeding, pack the nose as for epistaxis (29.7).

(b) Septoplasty

Correcting a deviated septum is delicate surgery that requires training and experience: *it is easy to cause collapse of the nose or a hole in the nasal septum.*

(c) Drainage of nasal septal haematoma (GRADE 1.3)

Do this in order to avoid collapse of the nose due to pressure-induced necrosis. You can use LA. Incise through the mucosa over the septal swelling and release the blood clot. Pack the nose to push the perichondrium back to touch and adhere to the cartilage. Remove the pack after 3 days.

29.10 Nasal polyps

Nasal polyps usually present in adulthood with longstanding nasal obstruction, which becomes complete from time to time, with or without a nasal discharge. There are grey fleshy masses in both the nasal cavities.

N.B. You can mistake the anterior end of the inferior turbinate (29-13) *for a nasal polyp*, so get used to examining the nose.

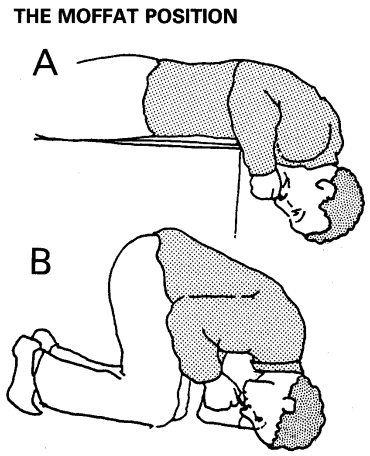


Fig. 29-10 MOFFAT POSITION FOR PUTTING DROPS IN THE NOSE. A, over a table. B, on the floor. If a patient inserts drops in either of these positions, and remains in them for 3mins afterwards, the drugs they contain are more likely to remain in contact with the mucosa for long enough to be effective.

When you remove them, they look like skinned grapes. Polyps are common and treatable. Some polyps are so large that they project through the back of the nose into the nasopharynx, and have to be removed through the mouth. If a polyp is on one side only, it may be malignant (or, very rarely, a meningocoele, 33.10), so treat it as such, until you have proved it is benign.

N.B. Some polyps may be due to *leishmaniasis* (29.18).

(a) Management

Try non-operative treatment first, using:

(1) A short course of oral steroids, 30mg of prednisolone for 1wk, which will often dramatically reduce the size of polyps; however, the polyps often return. So, following an oral course of steroids, use steroid drops to the nose. *Avoid long and repeated courses of oral prednisolone*.

(2) Nasal steroid drops alone or following oral prednisolone. Put 2 drops of betamethasone (50μg) into each nostril bd, while the patient is in the Moffat position (29-10), and ask him to stay like this for 3mins afterwards. Do this for 4wks. Steroid drops are absorbed into the bloodstream *so, again, don't use them for more than a month.*

(3) Nasal steroid sprays (beclomethazone or budesonide) puffed into each nostril x3-4 bd. Try this for 4wks, and only continue if there is improvement. Steroid sprays are, however, not absorbed into the bloodstream and in general are safe to use for the long term, but are expensive.

NASAL POLYPECTOMY (GRADE 1.4)

Premedicate the patient thoroughly; use LA. Sit opposite him, and ask a nurse to stand behind his head. Spray or douche the nose with lidocaine 2% with 1:100,000 adrenaline. Be careful not to exceed the maximum dose. Wait for 5mins.

With a good light shining over your right shoulder, open the nostrils with a nasal speculum. Pass a polypectomy snare, manoeuvre the loop to catch a polyp round its base, and remove it. If polyps don't come out with the snare, pull them out piecemeal with angled forceps.

Repeat the process, until you have removed as many polyps as you can. If there is pain, spray more anaesthetic and wait another 5mins. If there is excessive bleeding at the end of the operation, pack the nose as for epistaxis (29.7).

If you use GA, this enables you to remove polyps more completely than under LA, but there will usually be more bleeding. Avoid ketamine, because the laryngeal and pharyngeal reflexes are partly preserved, and cause trouble. Insist on endotracheal intubation, and put a large pack in the throat. Be sure it is visible throughout the operation, and has a strong thread fixed to it. Have good suction available. Large clots can form in the pharynx, and be aspirated when you remove the tube. So clear the throat first, and remove the pack with care. *It is not necessary to remove all polyps*, only the larger ones blocking the main part of the nasal cavity. Afterwards, pack the nose with an anterior pack each side (29.7). Make sure recovery from GA is in the lateral (coma) position.

CAUTION! Don't go too high up the nose when removing polyps: the brain is separated from the nasal cavity by a very thin piece of bone. Be careful when removing polyps from the lateral wall. Again the bone separating the nasal cavity from the orbit and eye (the *lamina papyracea*) is paper-thin and it is easy to start pulling out orbital fat.
If you perforate the orbital wall, *there is risk of damage to the optic nerve and bleeding into the orbit*.

N.B. Polyps often recur after surgery but usually after a few years. In children they may be the result of aspirating human papilloma virus from the mother's birth canal.

(b) Nasal tumours and cancers
Be suspicious about polyps that are unilateral and don't have the characteristic grey, oedematous, smooth appearance of common polyps. If you are concerned, carry out a biopsy: usually this can be done under LA but be careful not to biopsy a haemangiofibroma (29.7), a rare vascular tumour of male children.

29.11 Foreign bodies in the nose

Foreign bodies in the nose are not uncommon in children. The child is usually brought to clinic with unilateral nasal discharge, not nasal obstruction. In fact, it is wise to consider all unilateral nasal discharge in children as due to a foreign body until this has been ruled out by careful examination under GA. The discharge associated with a nasal foreign body is often foul-smelling.

It is rare that a child will let you remove a foreign body from the nose without general anaesthetic, and a foreign body (unless a small watch battery which will release alkali into the nose) is not an emergency operation.

Clear the nose and try to see the foreign body. Try to get the child to blow or sneeze the foreign body out. Close the other nostril and tickle the nose to make him sneeze.

CAUTION! If you suspect a foreign body, assume it is there, until you are absolutely certain it isn't.

ANAESTHESIA. LA is suitable, if you can see the foreign body, it is not too far back, and the patient is reasonably cooperative. IV ketamine is especially useful in children. If you use GA, insist on endotracheal intubation and pack the pharynx to prevent inhalation of the foreign body.

EQUIPMENT. You will need a good light, suction, angled forceps, and some kind of hook, such as a Eustachian catheter, a bent probe, or a bent paper clip held in a haemostat. Put a large speculum on an otoscope, and remove its back lens.

EXTRACTION OF A FOREIGN BODY IN THE NOSE (GRADE 1.2)
Use either a headlamp or mirror with a good light directed onto it. Use suction first: the foreign body may come out on its own. If not, try to bring the foreign body out anteriorly: if you push it posteriorly, it may be inhaled (this should not happen if the throat has been adequately packed).

If the foreign body is firm, pass your chosen hook beyond it, usually above it, turn the hook behind it and deliver it. *Don't try to grab it with forceps*, or you will push it further in. Try to draw it towards the floor of the nose, and away from its roof. You may be able to apply suction directly to the foreign body, especially if it is round, and then use an angled scoop to remove it. A foreign body is most likely to impact in the roof of the nose. *This is dangerously close to the floor of the anterior cranial fossa and the medial wall of the orbit. Check that there are no more foreign bodies* in situ, *and check for damage after your manipulation!*

If the foreign body is soft, use forceps. You may be able to suck small foreign bodies out. Otherwise, use small alligator forceps, or any forceps with blunt angulated tips.

If there is bleeding, use gentle suction. Packing (29.7) is seldom necessary.

CAUTION! Make sure there are no more foreign bodies present after you have removed an initial one.

29.12 Tonsillitis

Acute tonsillitis is common, especially in children <10yrs, and is usually due to *streptococcus*. It responds to mouth gargles and penicillin. More serious cases are caused by the Epstein-Barr virus in glandular fever (with lymphadenopathy), and *corynebacterium* in diphtheria (grey membrane on the tonsils). In scarlet fever, the *streptococcus* produces a toxin resulting in a high fever, rash and prostration.

Infection from tonsillitis may spread to the ear (otitis media: 29.4), around the tonsil (quinsy: 6.7), to the lungs (pneumonia), and in the bloodstream (rheumatic fever). It may also result in an allergic glomerulonephritis.

Acute tonsillitis does occur in adults, and is much more common in HIV disease; *it may then not respond to simple penicillin therapy.*

Sometimes the attacks of tonsillitis are frequent and debilitating; also the tonsils themselves may enlarge significantly, especially in general lymphadenopathy from other causes (17.1), *e.g.* HIV disease, tuberculosis, lymphoma and Kaposi sarcoma.

Very occasionally, in small children, large tonsils may almost meet in the midline and make eating difficult, as well as causing dyspnoea at rest, mouth breathing, otitis media, snoring at night, and even sleep apnoea.

TONSILLECTOMY (GRADE 2.4)

INDICATIONS
(1) Frequent, debilitating, recurrent attacks of tonsillitis (>6 attacks in 1yr), especially if causing otitis media.
(2) Tonsillar enlargement causing airway obstruction.

CONTRAINDICATIONS
(1) Current inflammation or abscess formation.
(2) Untreated HIV disease (5.6).
(3) Suspicion of a bleeding disorder: *you must investigate this first.*
(4) A local epidemic of poliomyelitis: *though now rare, this may still exist in certain parts of the world* (32.7).

METHOD
This is not a trivial operation, and carries the risk of fatal haemorrhage and airway obstruction; so weigh carefully the pros and cons! Make sure you can arrange blood transfusion if you need it.

The patient should be at least 3wks away from the last attack of acute tonsillitis. *You need an experienced anaesthetist who can reliably intubate children. A throat pack is essential, and a mouth gag almost indispensable.*

Make sure the head is extended and you are sitting at the end of the operating table. Insert the mouth gag to get a good view of the back of the mouth. Make sure the light and suction are optimal.

Incise the mucosa of the anterior pillar (or palato-glossal fold: 29-11B) immediately in front of the tonsil and identify the capsule of the tonsil. Use blunt dissection and so separate the tonsil from its bed (the superior constrictor muscle of the pharynx: 29-11E) until it remains attached only by its pedicle (a branch of the facial artery: 29-11E) near its lower pole. Tie the pedicle with mounted ligatures and finally remove the tonsil.

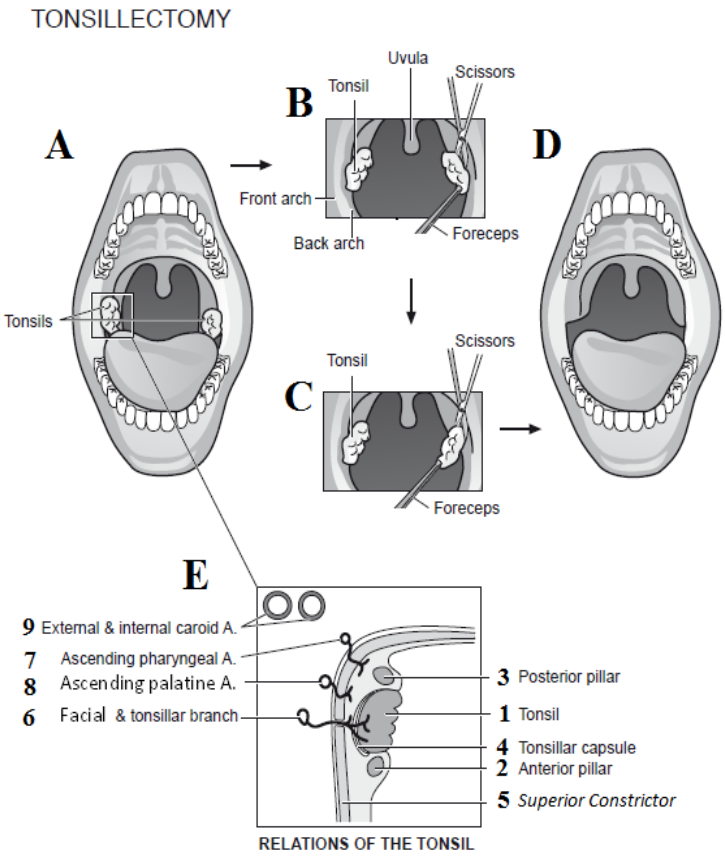


Fig. 29-11 TONSILLECTOMY.
A, view of the tonsils. B, dissecting out the tonsil. C, ligating the pedicle. D, final result. E, horizontal section: (1) tonsil. (2) anterior pillar (*palatoglossus*). (3) posterior pillar (*palatopharyngeus*). (4) tonsillar capsule. (5) *superior constrictor* muscle. (6) facial artery and tonsillar branch. (7) ascending palatine artery. (8) ascending pharyngeal artery. (9) external and internal carotid arteries. *After Ellis H. Clinical Anatomy, Blackwell 5th ed 1972 p.265 Fig 174.*

There may occasionally be feeding vessels from the ascending pharyngeal and ascending palatine arteries (29-11E) which you will need to ligate separately.

Observe the patient carefully: keep him nil orally till 6h post-operatively; then start oral fluids. Avoid opiate sedation. Use mouth gargles; chewing-gum and sweets are useful adjuncts to keep the mouth clean.

N.B. Some referred ear pain and fibrin over the tonsillar bed are normal post-operative consequences.

N.B. Soluble aspirin is useful in adults, *but don't use it in children.*

DIFFICULTIES WITH TONSILLECTOMY
If blood oozes from the mouth and nose or there is excessive swallowing post-operatively, assume there is haemorrhage. Take the patient back to theatre. Check blood clotting times and cross-match blood. Make sure you have good light and suction, hydrogen peroxide, and ligatures. Sedate the patient and remove the blood clot in the tonsillar fossa. Apply pressure with a swab soaked in hydrogen peroxide. If the bleeding is not controlled, re-anaesthetize and re-intubate the patient.

Occasionally, **if bleeding persists**, especially in a secondary haemorrhage, suture the anterior and posterior pillars (29-11E) together over a swab and add IV cloxacillin. Remove the swab after 48h.

Don't persist in trying to control haemorrhage in an unanaesthetized patient! In the case of secondary haemorrhage (3.5), *don't wait if the bleeding stops spontaneously* but examine the patient critically in theatre and be ready to suture the tonsillar pillars.

29.13 Naso-pharyngo-laryngoscopy

Examination of the throat is essential in most patients with ear, nose and throat symptoms, and one of the most useful instruments is the fibreoptic endoscope. This comes in 2 forms, a rigid and a flexible endoscope (29-12A,B). If you are fortunate to have one, take care of it; clean it properly after each use and store it carefully. You can transmit infection from one person to the next with the instrument, and you can easily damage the optic fibres. These work by internally reflecting light by way of systems of lenses and crystals: these unfortunately break up with time and use.

Alternatively, you can use an angled mirror and a headlight; *but this method (indirect laryngoscopy) is not easy*, so you will have to do it fairly often if you are going to become competent with it.

Fibreoptic endoscopes for looking at the nose, the throat and voice box are different from those used to look at the bronchi, oesophagus and stomach, since *they have no channels*; there is no suction and no biopsy channel. They are used simply to look. They are therefore narrower and simpler to clean and sterilize. But like other fibreoptic endoscopes, they are not cheap and need a special light source.

(a) Using a flexible fibreoptic naso-pharyngo-laryngoscope

The flexible fibreoptic naso-pharyngo-laryngoscope (usually abbreviated to FO endoscope) is some 30cm long with a handle and controls that allow you to focus the image and move the tip of the scope up or down. Connect it by a cable to a light source or to a battery pack.

(1) Check that the scope is clean, that the light source is working and that the image is focused.

(2) Explain to the patient what you are going to do. Decide whether he requires LA spray to the nose and/or throat. Most patients do not, but if a patient is anxious or has a narrow nose, then local spray of lignocaine or co-phenylcaine can help.

(3) Stand in front of the patient; examine one nostril and then the other with the scope, and decide which nostril is wider and will allow easier passage of the tube to the back of the nose.

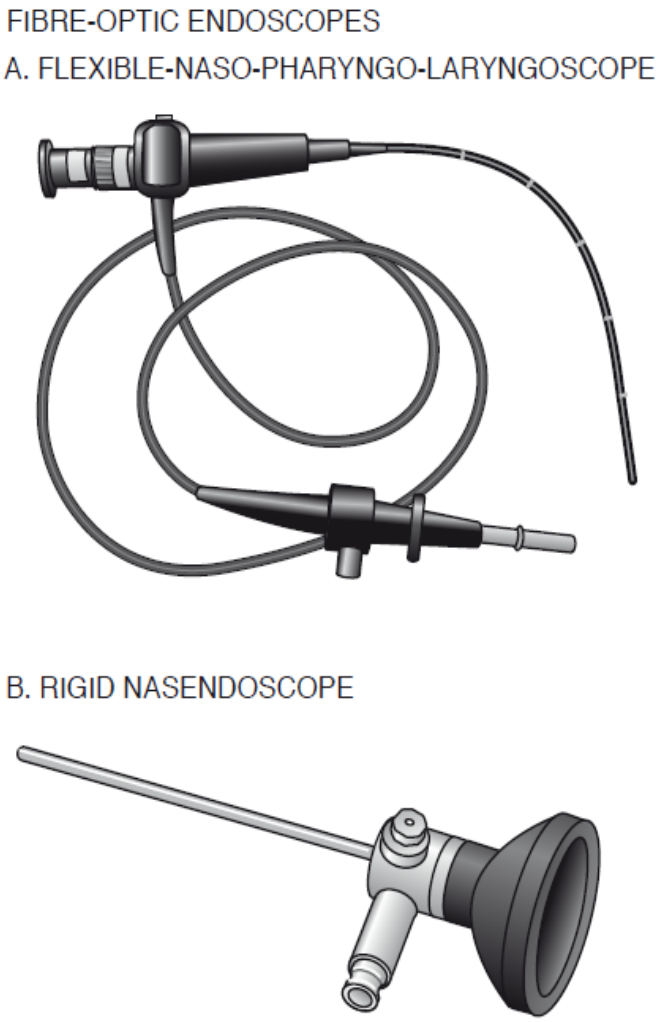


Fig. 29-12 NASO-PHARYNGO-LARYNGOSCOPES: A, flexible fibreoptic. B, rigid.

He should be sitting, preferably on a chair with a headrest, so that the head remains steady in one position. Get a nurse to steady the head.

(4) There are 4 ways to the back of the nose – the right or left nostril, and on each side either below the inferior nasal turbinate or above the inferior nasal turbinate (29-13) – and there is no predicting which way will be easiest. The narrowest part of the nose is generally at the back of the nose just before you enter the nasopharynx.

(5) As you pass through the nasal cavity, take note of the anatomy of the nose: the inferior and middle nasal turbinates and the nasal septum (often bent). Take note of any polyps (not uncommon) or cancers (rare). Benign nasal polyps are pale, oedematous, smooth and bilateral. Cancers are red, friable, irregular or bleed and almost always unilateral.

(6) At the back of the nose is the nasopharynx. Here, you will see the opening of the Eustachian tube (and by moving the end of the scope you can usually see both Eustachian tube openings whichever nasal passage you go down).

Take note of any abnormality in the nasopharynx: an abnormal swelling here may be a nasopharyngeal carcinoma.

(7) Ask the patient to breathe through the nose. This opens up the passage from the nasopharynx to the oropharynx and, by manoeuvring the tip of the scope downwards, you will be able to enter the oropharynx. You can now see the back of the tongue, the larynx and the hypopharynx from above (29-14). Often there is some mucus or saliva on the tip of the scope preventing a view. If so, angle the tip of the scope against the back wall of the oropharynx and ask the patient to swallow. This usually 'cleans' the end of the scope. Often you have to keep repeating this instruction, till eventually you get a good view.

(8) Look carefully at the base of the tongue, the epiglottis, the vocal cords and the pyriform fossae (29-15). Ask the patient to stick his tongue out in order better to see the base of the tongue and the vallecula (the space between the epiglottis and the base of the tongue).

(9) Ask the patient to take a deep breath in, so as to see the vocal cords move outwards, and then ask him to say "eee" or to count to 5 to see the vocal cords come together (29-15).

(10) Remove the scope and then pass it into the opposite nasal cavity to examine that nasal cavity; *there is no need to pass it further on the second side.*

(11) Check the patient is breathing well *and has no stridor.*

(12) Clean and sterilize the scope in preparation for the next patient.

(b) Using a rigid endoscope

This is generally used to examine the nasal cavities. It is often narrower than the flexible scope and therefore allows closer examination of the complicated anatomy of the nasal turbinates and meatuses, especially the middle meatuses.

The rigid scope can also be used to examine the larynx, in which case it is used like the laryngeal mirror. Hold the tongue with one hand and slide the scope over the back of the tongue to look down onto the larynx and hypopharynx from above (29-14). *You need a GA to do this effectively; you share the airway with the anaesthetist*.

If you do this for hoarseness and airway obstruction, *intubation can be difficult and even dangerous*, as on waking there is the danger of laryngospasm and obstruction from secretions or blood.

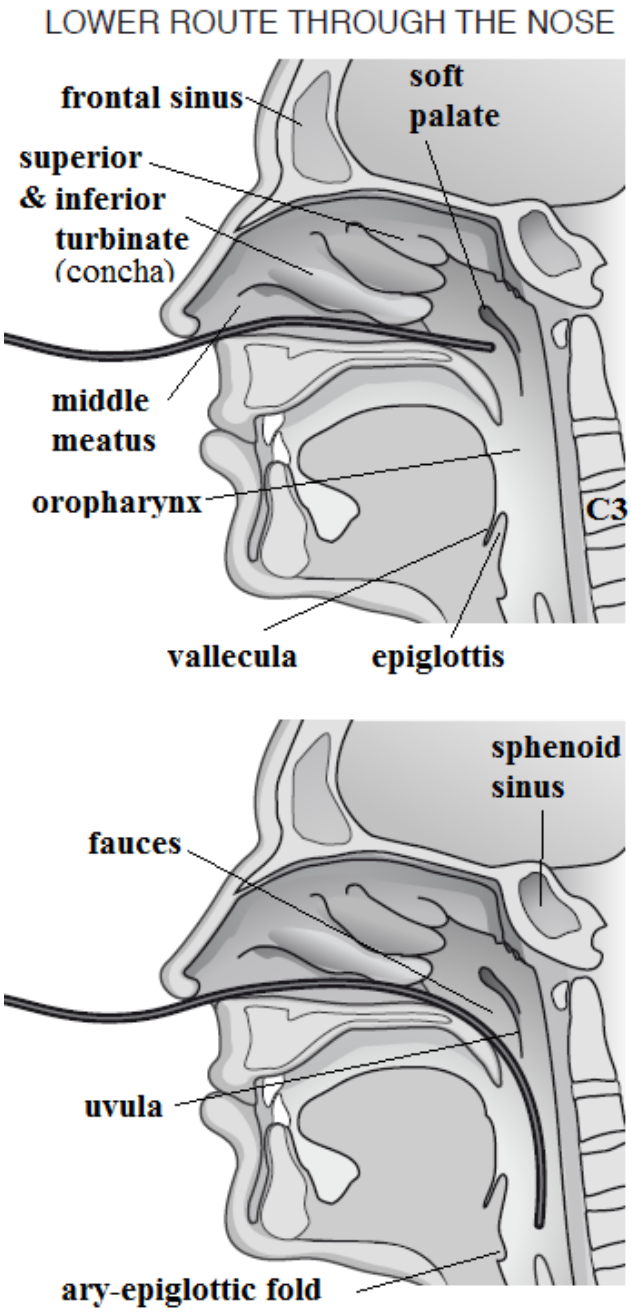


Fig. 29-13 LOWER ROUTE THROUGH THE NOSE (below the inferior turbinate)

VIEW LOOKING FROM NASO- TO HYPO-PHARYNX

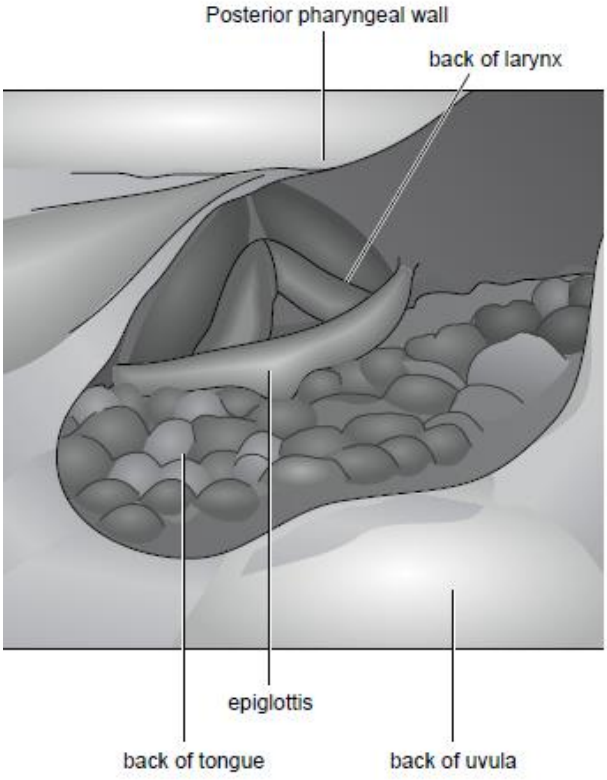


Fig. 29-14 VIEW FROM NASO- TO HYPO-PHARYNX, as seen through an endoscope introduced as in 29-13.

VIEW LOOKING DIRECTLY AT THE LARYNX

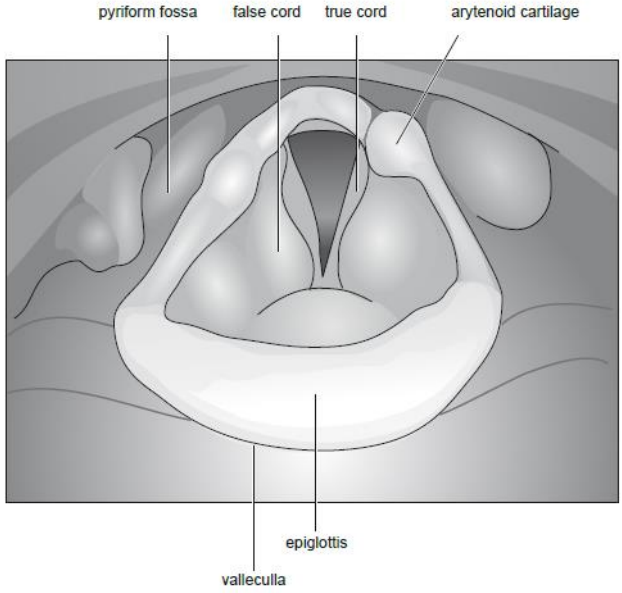


Fig. 29-15 DIRECT VIEW OF THE LARYNX, as seen through an endoscope introduced as in 29-13.

(c) Direct laryngoscopy

INDICATIONS

(1) Examination when you intend to take a biopsy.

(2) Assessing if a laryngeal tumour is operable.

N.B. Remember flexible bronchoscopy includes direct laryngoscopy!

METHOD

You need good communication with your anaesthetist for this. Position the patient with the neck flexed and head slightly extended on a pillow. Get an assistant to hold his right palm against the patient's head, and hold the thumb against the underside of the mandible. This gives vertical and lateral control.

Have the laryngoscope ready in your hand, and when you are sure the patient is anaesthetized, pass the laryngoscope into the mouth, *avoiding using the teeth as a fulcrum*. Press the laryngoscope against the back of the tongue until the epiglottis comes into view. With a curved laryngoscope blade, pick up the epiglottis with the point of the instrument, revealing the base of the arytenoids.

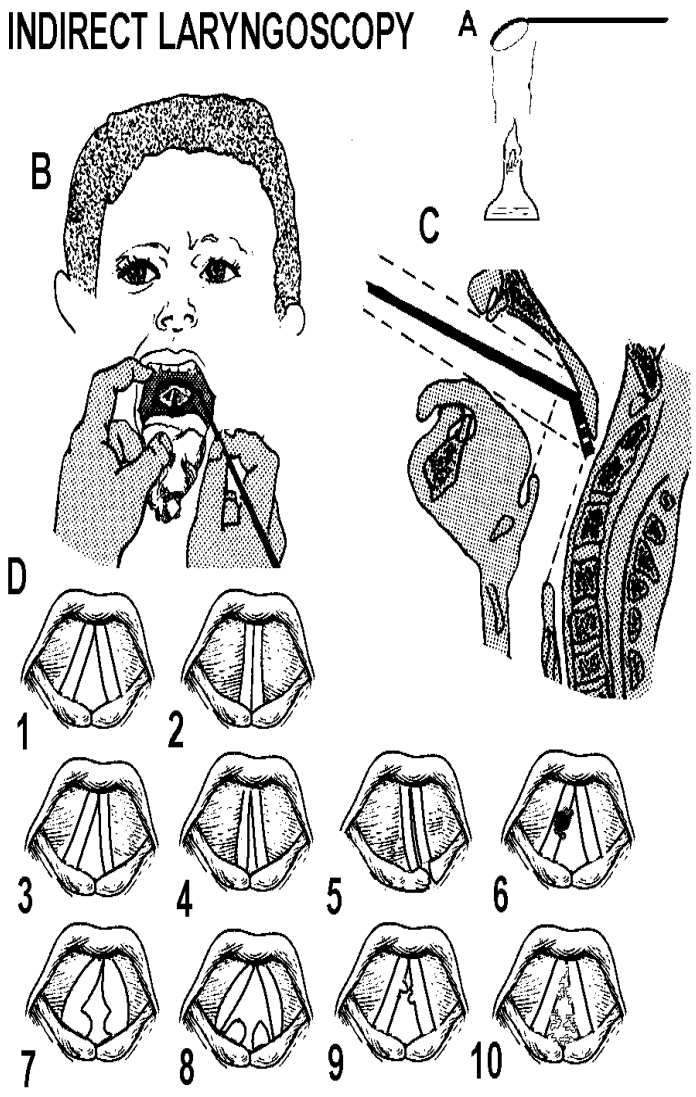


Fig. 29-16 INDIRECT LARYNGOSCOPY.
A, warming the mirror. B, hold the tongue with a swab between your fingers, and gently pass the laryngeal mirror to the back of the throat. C, note the light path to the larynx. D, views of the larynx: normal cords in inspiration (1), in phonation (2). Left vocal cord palsy in inspiration (3), in phonation (early, before 6wks, 4), and in compensated phonation (late, 5). (6), carcinoma (early and treatable). (7), swollen cords and arytenoids of laryngitis. (8), arytenoid oedema or granulomas (post-intubation or radiotherapy). (9), laryngeal polyps or singers' nodes. (10), papillomas.

Push the laryngoscope forwards with its handle: this flattens the tongue and brings the cords into view. Examine the oropharynx including the back of the tongue, and the hypopharynx including the pyriform fossa. Carefully examine the supraglottis, including the epiglottis and aryepiglottic folds. Pass the rigid scope into the larynx and examine the false cords, true cords and subglottis. Biopsy any abnormalities, but *don't disturb minor abnormalities of the vocal cords* as they are delicate structures and big biopsies of small lesions will make the voice worse.

When you have finished, allow the anaesthetist to ventilate the patient with oxygen to prevent bronchospasm.

(d) Cleaning and sterilizing
Don't leave this task to an untrained member of staff. Don't put off this job until hours after the endoscope has been used.

The 3 stages are cleaning, disinfection and rinsing. For a flexible endoscope, follow instructions as for the OGD (13.2). For rigid instruments, a multi-enzyme detergent agent is useful where immersion sinks are unavailable.

(e) Indirect laryngoscopy
EQUIPMENT. A good light coming from behind the patient and slightly to one side. A headlamp, laryngeal mirrors and a spirit lamp, 4% lidocaine in a laryngeal spray.

N.B. This is a dying art, but still very useful in places where there is lack of advanced equipment; nonetheless the success rate is only around 60% but practice makes perfect!

METHOD. Sit opposite the patient, and arrange him and the light so that it shines down his throat. Wrap gauze round the protruding tongue, and pull it forwards with your left hand. Spray the fauces, soft palate and pharynx with lidocaine.

Warm an angled mirror in the flame of a spirit lamp, and test its temperature on the back of your left hand; it should feel just warm, *but not hot*. Place the back of the mirror against the soft palate, push a little and look down at the larynx. Identify the cords.

Normal cords are white. Laryngitis makes them red, and chronic laryngitis also makes them swell. If you see a lump, it is probably a polyp. A ragged ulcer is likely to be a carcinoma. Note the movement of both the cords by asking the patient to say "eee". To examine the nasopharynx, depress the tongue with some soft instrument, and place a smaller (14mm) rhinoscopic mirror in the pharynx, so that your line of sight passes up behind the soft palate.

(f) Some conditions visible in the nose
(1) **Benign oedematous nasal polyps:** in some populations very common. These are bilateral and pale, often filling the nasal cavities (29.10).

(2) **Nasal septal perforation:** usually at the front of the nasal septum and secondary to trauma (picking of the nose with finger tips) but can be due to cancer, syphilis, leprosy, connective tissue diseases, cocaine and chronic use of ephedrine nose drops.

(3) **Nasal carcinoma:** if a polyp bleeds or is irregular, suspect cancer (29.16). This may arise from leather tanning, lacquer paint, soldering or welding fumes, and nickel or chromium exposure.

(4) **Foreign bodies:** determine if they are solid or not, and whether you can easily grasp them (29.11).

(g) Some conditions visible in the pharynx
(1) **Adenoids:** midline, usually seen in the young.

(2) **Naso- or hypo-pharyngeal carcinoma:** asymmetric, granular or ulcerating lesions (29.16).

(3) **Carcinoma of the base of tongue:** an ulcer or an irregular bleeding, exophytic mass.

(h) Some conditions visible in the larynx
(1) **Supraglottitis:** swelling of the epiglottis.

(2) **Laryngitis:** swollen, red vocal cords, usually symmetrical.
oedematous vocal cords (29-16D7)

(3) **Laryngeal papillomatosis:** laryngeal warts, sometimes very large (29-16D10).

(4) **Laryngeal carcinoma:** an early cancer is a small white plaque (leukoplakia) but as it grows, it can become thickened, ulcerating and a large mass that eventually blocks the airway (29-16D6, 29.17).

(5) **Vocal cord paralysis:** the vocal cord does not move outwards on inspiration (29-16D3), and does not move inwards on speaking or saying "eee" (29-16D4).

(6) **Foreign bodies**, *e.g.* dentures, a pen top, or a peanut.
N.B. Soaked toilet paper is particularly difficult to remove and dangerous!

(7) **Ascaris worms:** these may occasionally be aspirated when there is intestinal obstruction and profuse vomiting of a bolus of worms (12.5).

29.14 Bronchoscopy: inhaled foreign bodies

If the neck is flexed, and extended sufficiently, the mouth can align with the trachea or oesophagus, so that you can pass a rigid tube. This is the 'sword-swallowing position', and is the basis of rigid bronchoscopy (29-17) and oesophagoscopy.

In theory, these are simple procedures; the traditional type of bronchoscope is merely a long tube with a light at one end. In practice, however, removing a foreign body with one requires much skill. *Anaesthesia is difficult, and the skill of your anaesthetist is the main determinant of success.* You need a range of instruments to cover all sizes of patient, and also a variety of forceps.

There are many opportunities for disaster, particularly obstructing the airway, or tearing the lower trachea and bronchi especially if the patient is not properly sedated, so causing mediastinal emphysema and mediastinitis. If you have other instruments with fibreoptic illumination, make sure that your bronchoscope is compatible with that system. You will need good suction.

If you have a fibreoptic flexible bronchoscope, you can use this under LA quite readily. *It is much, much safer!*

Bronchoscopy is useful for:
(1) Sucking out a patient's stomach contents from the trachea, if he has been unfortunate enough to aspirate during a GA. Make this your first priority, especially if you are new to bronchoscopy.
(2) Sucking out the secretions which have gathered in the bronchi of a desperately sick patient post-operatively (11.11).
(3) Removing foreign bodies, especially peanuts inhaled by children. This is more difficult than the other indications, so if you are new to bronchoscopy, *don't start with this*, if you can avoid doing so.
(4) Diagnosing carcinoma of the trachea, carina and upper bronchi, and other diseases of the larger airways.

Inhaled foreign bodies in the larynx and tracheobronchial tree, particularly peanuts or watermelon seeds, are common. If a child is lucky, the first immediate bout of coughing expels the nut.

Otherwise, wheezing and coughing usually stop without expelling it. This may be followed by a latent interval, during which there are no signs, especially if the nut has gone far down the bronchial tree. This latent interval is then followed by fever, a cough, and the symptoms of chest infection. Antibiotics may relieve the symptoms temporarily, but they always return when treatment stops. A misdiagnosis of TB is common.

If coughing, or the 'upside down thump' or Heimlich manoeuvre described below, fail to remove a foreign body, it has to be removed through a bronchoscope. Even if you can successfully pass one, removing a foreign body is difficult, and sometimes impossible. *Leaving it inside, however, results in suppuration and chronic disability, or death.*

(a) Immediate treatment
If you are present when a child inhales a foreign body, turn him upside down and bang the back of the chest: he may cough it out. Alternatively, especially in an adult, hold him tight from behind and thrust suddenly and very firmly under the xiphisternum (the Heimlich manoeuvre: 30.1).

(b) History and examination
If presentation is not acute, take a careful history, look for impaired movement on one side of the chest, and listen for localized wheezing.

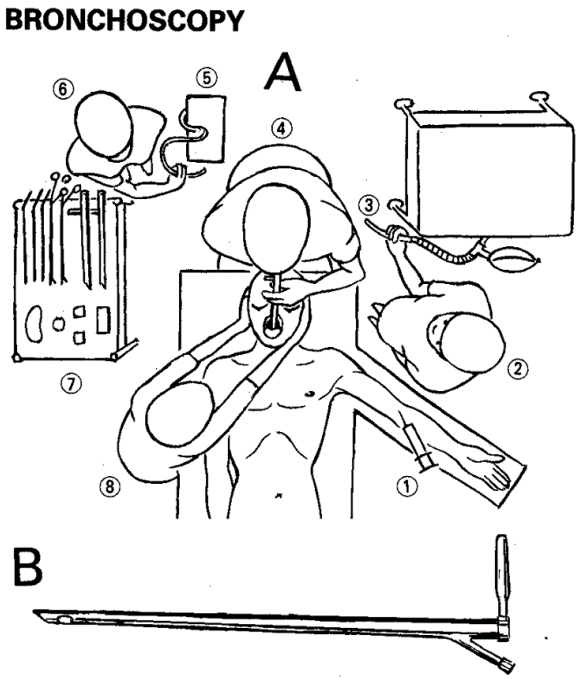


Fig. 29-17 ARRANGEMENTS FOR BRONCHOSCOPY.
A, after induction the anaesthetist moves to the patient's left. (1), cannula in a vein. (2), anaesthetist. (3), oxygen supply. (4), endoscopist. (5), suction. (6), trolley nurse. (7), trolley with instruments and tubes. (8), assistant holding the head in the midline. B, rigid bronchoscope.

(c) Radiographs
Look for a radio-opaque object, localized collapse, pneumonitis, consolidation of a segment or an entire lobe, and mediastinal shift. There will be obstructive emphysema if a foreign body allows air into a bronchus, but not out of it.

CAUTION!
(1) If a mother comes to you saying that her child has inhaled a peanut or other foreign body, believe her, as she is almost certainly right.
(2) Most plastics are radiolucent, *so you may not see them on a radiograph*.
(3) *A radiograph which does not show a foreign body* does not prove it is not there, unless its nature is known and it is definitely radio-opaque (*e.g.* cod, haddock or salmon fishbone).

N.B. Trout, herring and mackerel fishbones are radio-lucent! Ultrasound can help decide if the object is in the bronchus or oesophagus.

BRONCHOSCOPE, rigid, Negus, conventional lighting, distal illumination, complete with cords, Wappler fitting, battery box, two lamp carriers and 2·5V lamps, (a) infant lumen 5·4x4.1mm, (b) child 7x5·7mm, (c) adolescent small 8x6·7mm. If you want to remove foreign bodies from the lower respiratory tract, you will need these. This is not the complete range, which includes the large adolescent, the small adult, the adult, and several for the lower bronchus. Darken the theatre so that you don't have to use the bulbs at high voltages which shorten their lives.

N.B. The flexible bronchoscope does not require GA.

Keep some spare bulbs for it.

FORCEPS, for bronchoscope, (a) Chevalier Jackson, 2/2 teeth on 50cm shaft, (b) Haslinger tubular shaft or sliding shaft type for small bronchoscopes. If you have bronchoscopes, you will need forceps for them. Measure a length on the shaft of the forceps the same as the length of the bronchoscope; then you will know when the tip is beyond the length of the bronchoscope.

(d) Bronchoscopy (GRADE 2.5)

CAUTION! This is not an easy procedure. You will need a good anaesthetist and a good nurse.
Have a good suction ready, several sized bronchoscopes (the one suitable for a child will be of smaller diameter than the child's little finger), as well as a variety of foreign body and biopsy forceps, and a wire hook to remove a foreign body. Maintain oxygenation of the patient throughout.

Under LA. Add premedication. Use ketamine, with atropine 0·6mg for an adult. In the sitting position, inject 5ml of 2% lidocaine into the trachea with a short stiff fine-bore needle, aiming to produce a fine spray, by perforating the cricothyroid membrane. Check that you are in the trachea, by aspirating before you inject. The liquid will initiate a cough reflex. Before you pass the instrument, spray the cords with more lidocaine and wait 2mins.

CAUTION! Don't exceed the dose of lidocaine, particularly in a small child: 20ml of 2% lidocaine is the absolute maximum for an adult.

Under GA. Use a short-acting relaxant as well as GA and rigid bronchoscopy for removal of a foreign body.

CAUTION! Be sure to spray a child's larynx with lidocaine, because this will prevent laryngeal spasm as you pass the bronchoscope, and minimize difficult airway problems upon recovery.

You will need 2 connections, one for 'normal' intubation between attempts at bronchoscopy, and a larger one which fits snugly into the end of the bronchoscope, so that you can ventilate while the bronchoscope is in place, if the pulse rate falls, or if there is cyanosis. There is usually some leakage of gas, so you will need a good flow of oxygen.

Blow oxygen into the scope intermittently, watching the pulse rate meanwhile. *A falling pulse is a sign of anoxia*. Only proceed to remove the foreign body if the pulse is satisfactory. Oxygen through the side tube will not by itself provide adequate ventilation, especially if the patient is paralyzed.

The anaesthetist and the surgeon share the patient. *The anaesthetist must be in charge, and decides when he must give oxygen*.

CAUTION!
The chest must expand during ventilation. *If it does not, because you have passed the bronchoscope into the oesophagus,* remove it, let the anaesthetist intubate the child, wait and then try to bronchoscope him again.

INDICATIONS
(1) Aspiration of stomach contents
(2) Retention of secretions
(3) Removing a foreign body
(4) Biopsy of a bronchial lesion
(5) Diagnosis of tuberculosis

METHOD
Lie the patient flat with pillows behind the back. Use LA as above. Wear spectacles to protect yourself from showers of sputum. Stand behind the patient, hyper-extend his neck, and have the bronchoscope and sucker ready.

Now pass the rigid bronchoscope gently behind the tongue. Look for the uvula and the epiglottis. This will lead you in the midline to the vocal cords, as when intubating. As soon as you see them, aim the bronchoscope in the same direction as the trachea. Slip its beak between the cords and advance it downwards, sucking out the secretions as you do so.

If you have a flexible bronchoscope, pass this through one nostril (which you have anaesthetized with lidocaine spray or pack), and then keeping in the midline, through the vocal cords.

CAUTION!
(1) *Make sure you are not going down the oesophagus* (you must recognize the cords on entry).
(2) Very little movement should be possible between the sides of the trachea and the bronchoscope. So, hold its handle in your right hand. Hold its shaft between the index and middle fingers of your left hand. Rest your left thumb on the upper front teeth, and keep the lower lip out of the way with a gauze swab, held in your ring finger. If you hold the bronchoscope against the teeth like this, it and the head will turn as one and less damage is likely.

Look for the foreign body in the bronchi: the common site is just distal to the carina in the right main bronchus. This is shorter, more vertical and wider than the left. Most foreign bodies enter the right side.

If the carina is normal, pass the bronchoscope down one or the other bronchus, preferably the normal one first. When you withdraw from the right main bronchus and enter the left one, you will have to move the head to the right as you do so.

With luck you will see the foreign body, and perhaps the bronchi to particular lobes. Try to bring the foreign body out on the end of the sucker. If this fails, grasp it with the forceps.

If you cannot grasp the foreign body, try to pass down a Fogarty balloon catheter beyond it, and inflate the balloon below the foreign body, as in an embolectomy. It is usually best then to withdraw the bronchoscope and the foreign body together. If there is much pus, suck that out too.

CAUTION!
(1) Hold the bronchoscope lightly in your fingers, so that if the patient moves, it will move with him, instead of injuring the respiratory tract.
(2) *Take care not to damage the teeth*.
(3) Remove it promptly if the patient struggles.
(4) If you fail, and the anaesthetist says, "That's enough", *don't persist in your probably futile endeavours!*
(5) *Check that there are no more foreign bodies!*
(6) *Check that there is no damage to the airway!*

To identify the bronchial tree (11-23): on the right, look for the right upper lobe bronchus in the 2 or 3o'clock position, the apical lower lobe bronchus at 6o'clock, and the right middle lobe bronchus at 12o'clock. Then look into the bronchi of the lateral, anterior, posterior, and medial basal lobes.

On the left, look for the left upper lobe bronchus in the 10o'clock position, the apical lower lobe bronchus at 6o'clock, and then into the bronchi for the lateral, anterior, and posterior basal lobes.

STORAGE
Hang flexible endoscopes vertically in a lockable cupboard with good ventilation; *they should not be stored curled up in their transportation case. Biopsy forceps wires easily get tangled and caught in doors*; make sure they hang nicely on separate hooks.

DOCUMENTATION
Produce a regular form (13-10) with patient details, instructions, consent, indications for the procedure, and findings. Make sure you fill these correctly for each patient.

(e) Difficulties with bronchoscopy
If the patient is suffocating and blue from the procedure, wait and try again later. If he is suffocating because of the foreign body, you will have to persist.

If a foreign body breaks into pieces, bring it up bit by bit. If it slips off while you are withdrawing it through the cords, try again. If necessary, squirt a little saline down the bronchoscope with a syringe, and use suction.

If the foreign body rolls up and down the trachea, but you cannot get it past the cords, tip the table steeply head down, and manipulate it past them with the piece of hooked wire that you have prepared for this eventuality.

If the foreign body is up a side-bronchus, you will only reach it with a flexible scope, or if it shifts with vigorous physiotherapy.

If you are looking for a carcinoma, look for abnormalities of the wall, and biopsy any growth. It will be easier to remove a piece from the carina. Suck out the blood afterwards.

29.15 Tracheostomy and cricoidotomy

If respiration is obstructed and you cannot relieve it by simpler methods or by intubation, you may occasionally have to open the respiratory tract below the obstruction. You can enter it through the cricothyroid membrane or the trachea.

In an emergency, pass 2 to 4 large-bore (>1·5mm) short cannulae through the cricothyroid membrane. In an adult (but not in a child) you can open the cricothyroid membrane with a sharp knife.

If necessary, you can do this in ≤30secs: *it may be so urgent that you don't have time to sterilize the knife.* As an emergency procedure in an adult, this is simpler and safer than the other alternative, which is an emergency tracheostomy.

Permanent impairment of the voice or airway is unusual after a cricoidotomy. But it is a temporary procedure only, so perform a formal tracheostomy later.

If possible, *anticipate* the need for an emergency tracheostomy and do it as an elective procedure under LA, ketamine, or tracheal anaesthesia. It will:
(1) Provide immediate relief for upper airway obstruction.
(2) Reduce the dead space by 100ml and nearly double the alveolar ventilation.
(3) Provide an opening through which you can suck out secretions.
(4) Provide an airway that can be continued indefinitely.

But, a tracheostomy will also:
(1) Greatly diminish the effectiveness of the cough reflex.
(2) Short-circuit the humidifying effect of the upper respiratory tract, and so dry the tracheal mucosa and make the bronchial secretions more viscid.
(3) Make infection of the lower respiratory tract much more likely, so careful aseptic procedures are essential.
(4) Occasionally cause severe bleeding.
(5) Carry the risk of tracheal stenosis later, especially in a child.

Intubation is almost always possible, so that tracheostomy is only very rarely necessary if intubation fails or is unsatisfactory (*e.g.* during transport), has to be prolonged for >7 days, and there is no other way of maintaining the airway. This is then life-saving. Even so, *a tracheostomy has serious risks, especially when nursing care is poor*, but these may be less than intubation or doing nothing!

(a) Emergency cricoidotomy (GRADE 1.4)

INDICATIONS
Any indication for a formal tracheostomy when the patient is in immediate danger of death, and *there is no time to perform a formal tracheostomy*.

CONTRAINDICATIONS
Children (where the cricothyroid membrane is too small).

METHOD
Make sure there are already nasal cannulae in place to supply oxygen. Place a pillow behind the neck to extend it, and bring the larynx forward. Find the prominence of the thyroid cartilage in the midline, and follow it downwards to the prominence of the cricoid cartilage (29-18). Feel these landmarks on your own throat now.

Use your fingernail to mark the depression formed by the cricothyroid membrane in the midline between the thyroid and cricoid cartilages.

Insert 2 to 4 short wide-bore (>1·5mm) cannulae through the cricothyroid membrane (29-18B). Give oxygen through one of them if necessary. You can use a knife in patients >10yrs. *There is usually no time to make a vertical midline incision over the thyroid and cricoid cartilages.* If there is, insert the tip of a solid bladed knife horizontally through the cricothyroid membrane as near the cricoid cartilage as you can.

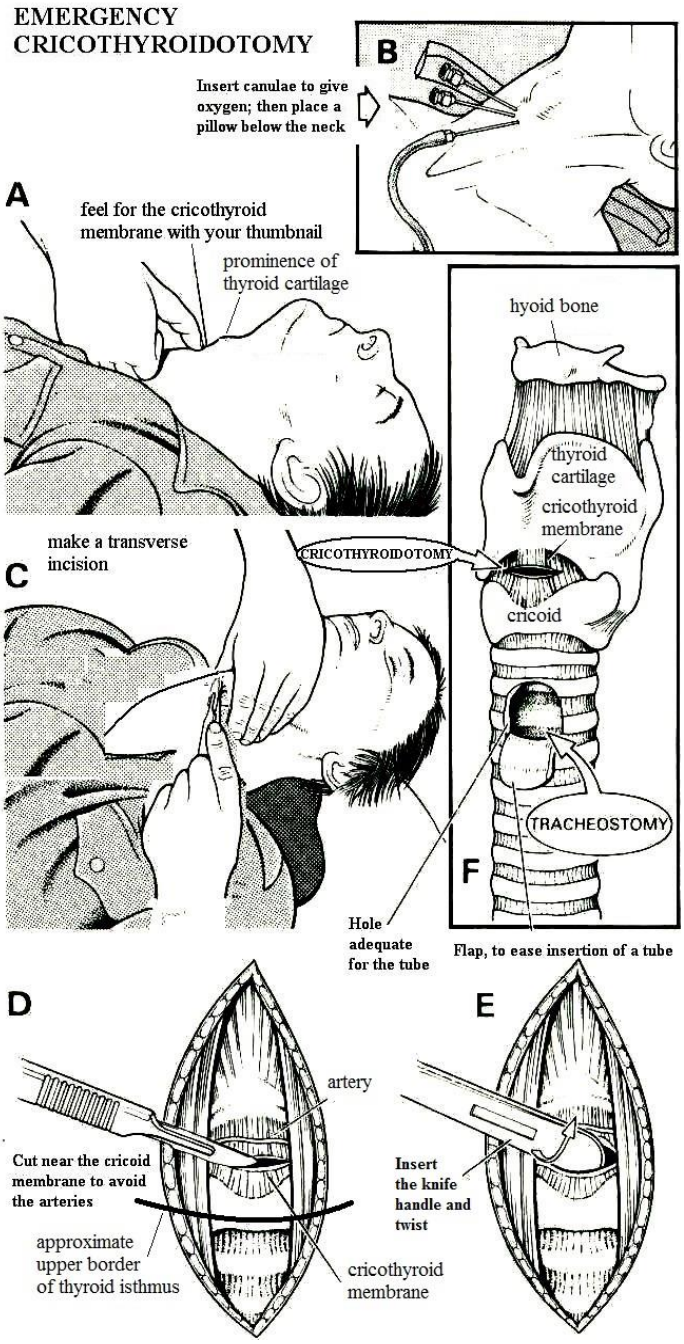


Fig. 29-18 EMERGENCY CRICO(THYRO)IDOTOMY.
A, position of the patient, feeling for the cricothyroid membrane. B, insertion of cannulae. C, making the incision. D,E,F, anatomy of cricoidotomy compared with tracheostomy.

HAMID (25yrs) heard a lion chasing his cows. He went out with a spear, but the lion leapt at him, biting his throat, and penetrating the larynx. He arrived in hospital at the point of death, with blood bubbling from the mouth. It obscured the oedematous distorted larynx, so that intubation was impossible. A tracheostomy was performed with some difficulty under LA. He immediately began to breathe normally. Much blood was sucked from the trachea, and blood stopped coming from the mouth. He recovered completely.

STEPHEN (18yrs) hit a rock on the road while riding his motorbike and flew over the handlebars, landing on his chin. The impact split it in two, lacerating the tongue down the middle. Fortunately he was brought to hospital lying on his side because every time he moved onto his back, he would stop breathing as the detached tongue fell back. A tracheostomy was done under LA with difficulty in the lateral position, and then all his facio-maxillary injuries repaired. He made a full recovery.

OMARI (36yrs) was crushed by some heavy scaffolding in a sugar factory. He was dyspnoeic with paradoxical movement on the left side of the chest, which had no breath sounds and diminished vocal resonance. It was resonant anteriorly, and dull at the base posteriorly. The trachea and apex beat were shifted to the right. Radiographs confirmed the diagnosis of multiple fractured ribs with a flail chest and a left haemopneumothorax. A chest drain connected to an underwater seal was inserted in the left mid-axillary line, and oxygen was administered by mask. Much air and a litre of blood flowed into the drain bottle, but he remained distressed and cyanosed. The chest was too painful to allow him to cough. Secretions began to accumulate, so a bronchoscopy was performed and copious sputum sucked out. Unfortunately, bronchoscopy was too traumatic to be repeated. Further radiographs showed diffuse mottling throughout both the lung fields. A tracheostomy was performed, and the trachea was repeatedly aspirated, after which his general condition improved and the cyanosis disappeared. Eight days later the tracheostomy tube was removed and 3wks after discharge, he returned to work.

This will avoid the cricothyroid arteries which run across the membrane superiorly. Twist the blade slightly and place any convenient tube into the hole (*e.g.* a ballpoint pen sheath). *Make sure this tube does not fall out! Make sure the tube is not too small to fall into the hole and then block the airway further!*

EQUIPMENT

An efficient suction machine is vital. Choose a tracheostomy tube of the size of the endotracheal tube (if present) or smaller (gauge this using the size of the patient's little finger as a guide). Use a cuffed tube if you want to ventilate the patient.

Insert a double (outer and inner) tube if the tracheostomy is likely to be long-term: you can then take out the inner tube and clean it in the sink. *Don't use too small a tube.* If it is too long, it may reach the carina and block one of the bronchi. An incorrectly-fitting tube may erode an artery and cause severe bleeding. *You don't need a special tracheal retractor or hook.*

N.B. Before any tracheostomy, warn the patient that he will not be able to talk immediately after the operation.

Fig. 29-19 EMERGENCY TRACHEOSTOMY IN A SMALL CHILD. Place the child on your lap holding the hanging head extended. Incise vertically midway between the cricoid and the suprasternal notch. *After Marsden AK, in Rutherford WH et al. Accident & Emergency Medicine, Churchill Livingstone 1989 p.120 Fig 10.26.*

(b) Formal tracheostomy (GRADE 2.3)

INDICATIONS
(1) Failed or unsatisfactory intubation, with no other way of maintaining the airway.
(2) Prolonged intubation (>7 days).
(3) After cricoidotomy.
(4) Acute respiratory infection in children, especially epiglottitis, laryngo-tracheo-bronchitis, or diphtheria.
(5) To ease suction of secretions.
(6) Respiratory obstruction following thyroidectomy (25.7).
(7) Presence of an obstructing laryngeal tumour (29.17), foreign body, or *ascaris* (12.5).
(8) Ludwig's angina (6.11) or angio-oedema.
(9) Tracheal damage.

CAUTION! Stand clear as you cut; you may be showered with droplets of blood and secretions from coughing through the tracheostomy wound.
For prolonged intubation, fashion a formal elective tracheostomy as soon as you can.
If you delay, perichondritis, stenosis, and subglottic oedema may follow.

Position the patient with the head on a soft ring support, and a cushion under the shoulders, so that the neck is extended. Make sure the head and body are lined up straight, so *you know where the midline is.*

ANAESTHESIA
Use LA by preference. Its use on a struggling patient is difficult and you may then need some sturdy helpers.

Adrenaline infiltration (1:100,000) will reduce troublesome bleeding. IV ketamine is the safest GA, unless the patient already has an endotracheal tube. A laryngeal mask is useful as it leaves the trachea free.

INCISION
Make a transverse incision 4cm long 2cm below the border of the cricoid cartilage (29-20A), *or vertical incision in an emergency*. Cut through the subcutaneous fat, and the cervical fascia (29-20C).

N.B. In an emergency, lie a small child on your lap with the head held hanging, and make a vertical incision midway between the cricoid and the suprasternal notch.

There is a fibrous median *raphe* between both right and left *sternohyoid* muscles. The *sternothyroid* muscles lie slightly deeper. Cut *in the midline* down to the tracheal rings, retracting the muscles laterally. You will now see the isthmus of the thyroid gland, which varies considerably in size.

If the isthmus of the thyroid is large and interferes with your approach to the trachea, divide it. Make a small horizontal incision through the pre-tracheal fascia over the lower border of the cricoid cartilage. Put a small haemostat into the incision and feel behind the thyroid isthmus and its fibrous attachment to the front of the trachea (29-20D).

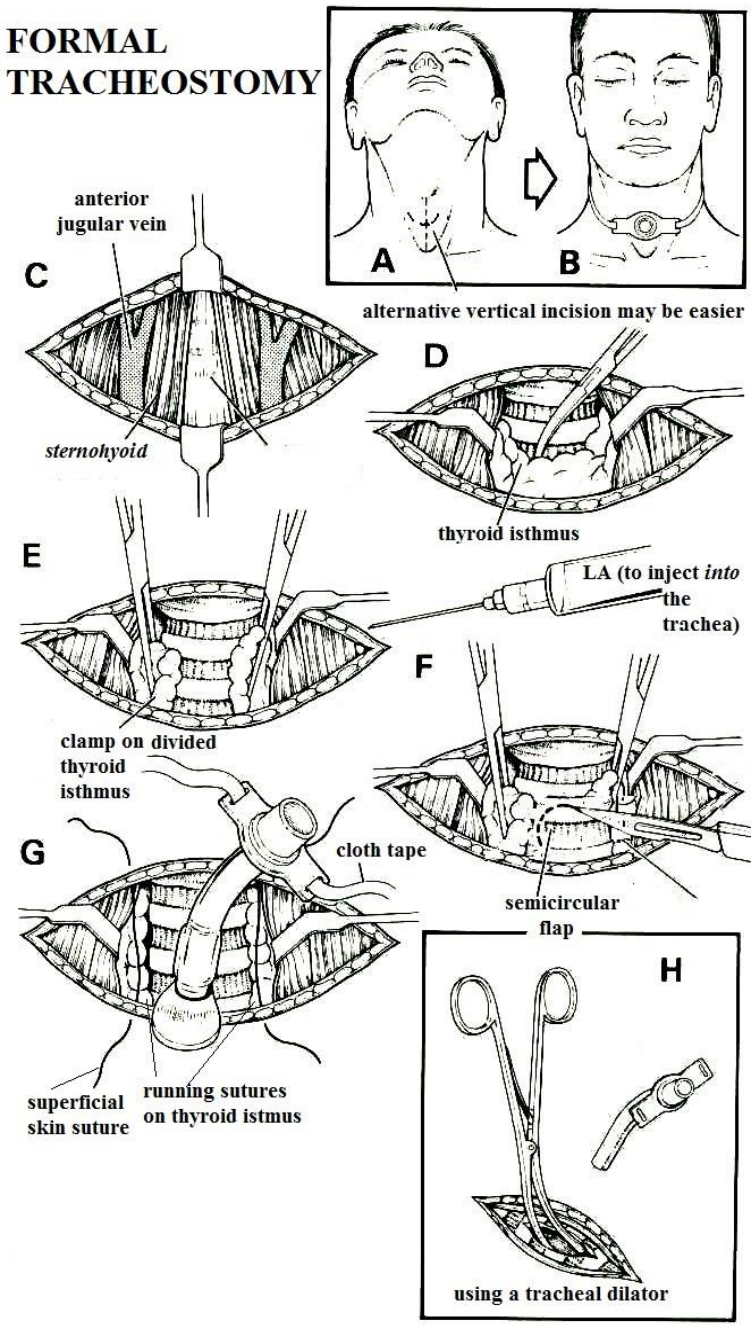


Fig. 29-20 FORMAL TRACHEOSTOMY.
A, incisions *(a vertical is easier in an emergency)*. B, tube finally in place. C, incise the skin and pre-tracheal fascia. D, after cutting in the *midline*, pass a haemostat behind the thyroid isthmus, if necessary. E, clamp the cut thyroid. F, cut a flap in the 3rd tracheal ring. G, insert the tracheostomy tube, sew up the cut edges of the thyroid isthmus, and insert superficial sutures. H, demonstrating the use of a tracheal dilator.

N.B. You may need tracheostomy equipment in a hurry, so have a set ready sterilized in the theatre.

When you have found the plane of cleavage, use blunt dissection to separate the isthmus from the trachea. Put a large haemostat on each side of the isthmus, and divide it (29-20E). Later, oversew the cut surfaces (29-20G).

Make sure you have the correct-size tracheostomy tube ready. *Test the cuff* if you are putting in a cuffed tube. Check that the suction is working. If there is an endotracheal tube *in situ*, ask the anaesthetist to suction the airway and squirt some lidocaine down the tube, and withdraw it just above the cricoid. Alternatively inject 2ml lidocaine directly into the trachea: you can confirm you are in the right space by aspirating air into a syringe filled with fluid.

CAUTION! Control all bleeding before you open the trachea. *Don't use diathermy once the trachea is open because oxygen used for ventilation may cause a fire!*

If you have a sharp hook, insert it below the cricoid and get an assistant to pull the trachea forwards. Cut an inverted U-flap (29-20F) containing the 3rd and 4th tracheal rings and insert the tube. The flap will act as a guide to direct the tube into the trachea and will make changing it easier. A flap largely eliminates the great danger of a tracheostomy, which is inability to replace the tube quickly when it has come out accidentally. You can suture the flap to the skin to keep it open.

N.B. It might be unwise to excise a flap in a child; a simple vertical incision of adequate length may be better, but re-insertion of the tube can be difficult.

CAUTION!
(1) *Stand clear.* If there is blood in the trachea, coughing will expel it everywhere.
(2) *Don't disturb the first tracheal ring*.
(3) *Don't remove any of the trachea*.
(4) *Don't incise more than 40% of the circumference of the trachea*, or severe stenosis may follow.

If you have a percutaneous tracheostomy set, proceed as above to puncturing the trachea; then pass a guide wire at the 12o'clock position into the trachea making sure it does not slip through the side hole of an endotracheal tube still *in situ*. Along this guide wire, pass the dilator 'seated' correctly on its cannula. Then withdraw the cannula and guide wire, and pass the tracheostomy tube over the dilator, finally removing the dilator.

The patient tolerates the tube more easily when the mucosa is anaesthetized with 2ml lidocaine. *The tube must not slip out*, so tie the tube in place with tapes round the neck. Tie it with the head well flexed, or the tapes may become slack when the patient sits up in bed with his head forward. Pack petroleum jelly gauze round the tube, and bring the edges of the skin incision together. Leave a little space round the tube, to minimize the danger of subcutaneous emphysema.

CAUTION!
(1) *Don't suture the skin tightly or leave it too loose round the tube*. Surgical emphysema can be caused by closing the skin too tightly round the tube (causing air to be pushed into the tissues on coughing round a partly blocked tube), or closing it too loosely, enabling air to be drawn into the tissues when on panic inspiration through a blocked tube.
(2) *Don't let the tube become blocked.*

If you use a cuffed tube, avoid too short a cuff or too high a pressure inside it. Deflate the cuff 4hrly for 15mins to reduce the risk of pressure necrosis on the trachea.

(c) Post-operative care of a tracheostomy

Blockage of the tracheostomy by crusts, especially in the first days after insertion, is a serious danger for all patients. The reason for the crusting is that air is no longer being warmed and moistened by the nose, and so cold and dry air is entering and irritating the trachea and bronchi. Secretions quickly build up which then dry and block the tracheostomy. Therefore, keep the patient in a steamy room to prevent crusts forming in the tube. If necessary, use a steam kettle or squirt a fine spray of saline into the tube every 15mins. Use humidified oxygen. Suck out secretions with a soft sterile catheter. If viscid secretions have formed, loosen them by injecting 3ml of sterile saline solution and then aspirate.
Avoid prolonged or too frequent suction.

If the tracheostomy has an inner tube, remove it regularly for cleaning (at least every 4h for the first few days). Arrange vigorous chest physiotherapy.

CAUTION! Suck out the trachea aseptically. This is no less important than catheterizing the bladder aseptically. Use a fresh, sterile catheter each time. Remove and clean the *inner* tube of a double tube every 4h during the first few days.

Deflate the tracheostomy cuff regularly for 15mins every 4h for the first 24h. Then deflate it permanently. Only keep the cuff inflated for >24h if there is still oozing or bleeding from the wound edges, or the patient is aspirating when drinking or eating, in which case inflate the cuff only on eating or drinking.

Change the tracheostomy tube regularly to clean it or to insert one that allows the patient to speak.

CAUTION!
(1) Try not to change the tracheostomy tube before the 4th post-operative day. If you take it out too soon, it may be difficult to replace. Check the tension of the tapes regularly.
(2) Minimize the risk of infection by sucking out the trachea regularly under careful aseptic precautions.

(d) Tracheostomies and speech

A patient who has just had a tracheostomy will not be able to speak if the tracheostomy cuff is inflated, as all air is coming out through the tracheostomy. A patient with a tracheostomy can only talk if air can be breathed out through the mouth.

There are 2 ways for this to happen:
(1) a narrow tracheostomy tube will allow air to pass alongside the tube up into the larynx;
(2) a special tracheostomy tube with a fenestration (hole) at its bend will allow air up into the larynx.

When the patient breathes out and at the same time occludes the tracheostomy by putting his finger over the tube, the exhaled air will travel up to the back of the mouth and allow speech.

(e) Removing tracheostomy tubes

If you think that a patient no longer needs a tracheostomy tube, then change the tube for a narrower-diameter tube, so that he can breathe around it.

Then occlude the opening of the tracheostomy with a cork or tape. The patient will then breathe with air passing around the tracheostomy. If he remains comfortable over 24h, then you are safe to remove the tracheostomy. Afterwards, apply a dressing to the stoma wound and within 2 weeks, the majority of tracheostomy sites will have completely healed.

(f) Difficulties with a tracheostomy

If there is fierce bleeding while you are inserting a tracheostomy tube, the blood may be coming from:
(1) The anterior jugular veins (29-20C).
(2) The isthmus of the thyroid.
(3) The wall of the trachea. If blood enters the trachea round the tracheostomy tube, immediately insert a cuffed tube. Get suction ready. Then open the wound and tie any bleeding vessels.

If you are not sure the tracheostomy is in the right place, check air entry in the lungs, or if possible using a flexible bronchoscope through the tracheostomy. However, if you are still in doubt, pull the tube out and start again.

If the tracheostomy tube slips out:
(1) it is in the wrong place.
(2) you have used the wrong shape of tube.
(3) the tapes round the neck were badly adjusted.
(4) the tube was not properly secured.

It may be difficult to re-insert; make sure you have suction, and a tracheal spreader ready! Change the tube for one with a better shape. With the obturator in the tube, place it in the trachea. You will find this easier if you use the tracheal spreader (29-20H). Then *don't forget to remove the obturator!* If necessary, take a soft tissue lateral radiograph of the neck, to show how the tube is lying in the trachea.

If the tube blocks, change it, humidify the inspired air, and suck regularly. *N.B. As the patient cannot talk, he may just become agitated and restless, or even confused and violent when the tube blocks. He may die if you ignore a blocked tracheostomy tube*. If you are called to see a patient fighting for breath because the tracheostomy is blocked, *you must remove it immediately*, then keep the tracheostomy wound open by forceps, until you have another tube to put in (either a new tube or the old one cleaned).

If the trachea becomes stenosed, it is probably because you left a cuffed tube in too long, or allowed the unsupported weight of attached anaesthetic tubing to rest against the trachea, or put the tracheostomy too high in the subglottic region.

If the patient cannot tolerate the removal of the tube, make sure the U-flap is not falling back inside the lumen of the trachea to block it. Otherwise, the reason may be psychogenic. In an adult, gradually reduce its size, then cork it for progressively longer periods before removing it.

If there is dyspnoea with a patent tracheostomy, there may be a pneumothorax (especially in a child): insert a chest drain (9.1).

29.16 Nasopharyngeal and maxillary antral carcinoma

(a) Nasopharyngeal carcinoma is important on a world scale. In Southern China it is a very common cancer. It is fairly common in Southeast Asia and North and East Africa. It is strongly associated with the Epstein-Barr virus, but, unlike cancers in other parts of the pharynx, not with either alcohol or tobacco.

It is more common in males, and has a peak age incidence between 40-50yrs, but is sometimes seen in older children. Some 10% of nasopharyngeal tumours are lymphomas. They spread locally by direct extension, regionally to neighbouring nodes, and distantly in the bloodstream. Distant metastases to the lung, bone, and liver occur more often from the nasopharynx than from any other site in the head and neck.

Carcinoma of the nasopharynx presents one or more of the following:
(1) Hearing loss due to a middle ear effusion secondary to a blocked Eustachian tube. *Any middle ear effusion which cannot be explained by a recent upper respiratory tract infection*, in an adult, especially from an ethnic group epidemiologically at risk, must be presumed to be due to a nasopharyngeal cancer until proven otherwise.
(2) An upper neck enlarged lymph node. Some 80% of nasopharyngeal cancers already have ≥1 enlarged cervical lymph nodes on presentation (usually unilateral but sometimes bilateral). Often the patient comes with an enlarged neck node and no other symptoms.
(3) Nasal obstruction.
(4) Cranial nerve involvement due to infiltration of cancer along the skull base. There can be vocal cord palsy (due to involvement of the X^{th} (vagal) nerve), diplopia, facial pain and numbness, or facial palsy (due to involvement of any of the II^{nd} to VII^{th} cranial nerves).

EXAMINATION
Carefully palpate the entire neck for enlarged nodes. Feel particularly for the uppermost internal jugular node, just below the tip of the mastoid process. This is often the first node to be involved when the primary is silent.

Observe the soft palate for asymmetry due to displacement by a tumour. If you are skilled, examine the nasopharynx with a mirror (29.13) or flexible scope.

BLIND BIOPSY is less satisfactory than open biopsy, but is possible under LA, using a long narrow forceps pushed along the floor of the nose.

OPEN BIOPSY (GRADE 1.5). Use GA and endotracheal intubation. Use the tonsillectomy position, lying supine with a pillow under the shoulders and with the head extended. Insert a mouth gag. Pass a catheter through the nose and out through the mouth. Use this to retract the palate. Using a warmed laryngoscopy mirror, inspect the pharynx and remove suitable pieces for biopsy.

If there is a suspicious node in the neck and you can see no obvious primary (unusual), take specimens from several suspicious-looking areas in the nasopharynx.

RADIOGRAPHS may show involvement of the base of the skull.

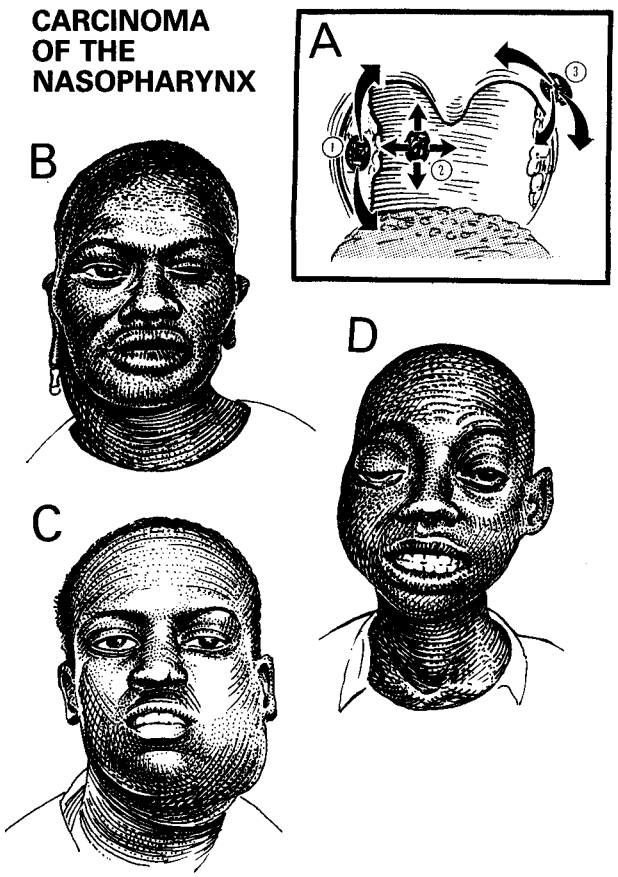


Fig. 29-21 CARCINOMA OF THE NASOPHARYNX.
A, directions of spread of carcinoma from (1) the tonsil, (2) the posterior pharyngeal wall, and (3) the soft palate. B, 45-year-old with cervical metastases and paralysis of the left IInd, IIIrd, IVth, Vth and VIth cranial nerves. C, 14-year-old boy with enlargement of the left cervical glands, but no cranial nerve lesions. D, 12-year-old boy with severe trismus ('lockjaw') from *masseter* spasm, bilateral proptosis, ophthalmoplegia and right-sided blindness from an anaplastic carcinoma of the nasopharynx, but without involvement of the cervical glands.
A, after Copeland III EM, Surgical Oncology Wiley 1982 p.127 Fig 5 with kind permission. B,C,D, after Clifford P. E African Med J 1965;42:381.

STAGING is a guide to prognosis:
TIS carcinoma *in situ*.
T1 tumour confined to one site in the nasopharynx, or no tumour visible (+ve biopsy only).
T2 tumour involving two sites (both posterosuperior and lateral walls).
T3 extension of the tumour into the nasal cavity, or oropharynx.
T4 tumour invading the skull, or involving the cranial nerves.

TREATMENT
The role of surgery is limited to biopsy. If there is a lymphoma, treat for it (17.6). If there is a carcinoma, try to refer for chemo-radiotherapy.
Treatment of nasopharyngeal cancer is highly specialized and needs to be given in centres with appropriate experience and facilities.

PROGNOSIS
Local control is possible in 60-90% of cases; 5yr survivals range from 30% (nasopharyngeal carcinomas) to 60% (lymphomas). Prognosis is dependent upon facilities for chemo-radiotherapy.

(b) Carcinoma of the maxillary sinus usually presents with one or more of the following symptoms and signs:
(1) a slowly progressive swelling of the cheek
(2) jaw pain which is not alleviated by tooth extraction
(3) a foul blood-stained discharge from the nose.
(4) malignant ulceration of the upper jaw or hard palate.
Look for swelling of the palate, epiphora (due to obstruction of the lachrymal duct), and enlarged lymph nodes behind the angle of the jaw.

Radiographs may show an increase in the size of the sinus, and later erosion of its walls. Biopsy any polyps and send them for histology. A definitive operation will usually require a total excision of the maxilla, with or without radiotherapy, for cure. The 5yr survival rate is 30-60%.

29.17 Laryngeal carcinoma

The larynx is divided into 3 parts, the glottis (the vocal cords), the supraglottis (larynx above the vocal cords) and subglottis (larynx below the vocal cords).

(a) Glottic carcinoma is the most common and usually presents early with hoarseness for >2wks. It has a 95% chance of 5yr survival with radiotherapy, so refer such.
N.B. Hoarseness in a middle-aged smoker is carcinoma of the glottis until proved otherwise.

(b) Supraglottic carcinoma often presents late with minimal symptoms until it is advanced (because there is a fair amount of room for the cancer to grow into). Then there is hoarseness, a feeling of something in the throat, unexplained pain in the ear (because of a common sensory nerve pathway), or swollen neck nodes.

(c) Subglottic carcinoma presents early with stridor and airway obstruction.

However, all laryngeal cancers may ultimately cause hoarseness and all laryngeal cancers will ultimately cause airway obstruction and stridor.

Treatment of laryngeal carcinoma is by surgery and/or radiotherapy. Treatment for cure is likely to require laryngectomy but may only be appropriate for <10% of patients. However, an early (T1) lesion confined to the true cord has a 95% 5yr survival with radiotherapy alone. Where there is airway obstruction and stridor, palliative permanent tracheostomy (29.15) will help.

29.18 *Leishmaniasis* of the nose and lips

There are a number of *Leishmania* species which are transmitted through the bite of the sandfly, principally in tropical and subtropical Central and South America, the Mediterranean basin, and western Asia from the Middle East to Central Asia. They cause single or multiple skin ulcers, and complications ensue when the parasite spreads later, usually >2yrs, but maybe up to 30yrs, to the nasopharyngeal mucosa, resulting in tissue destruction.

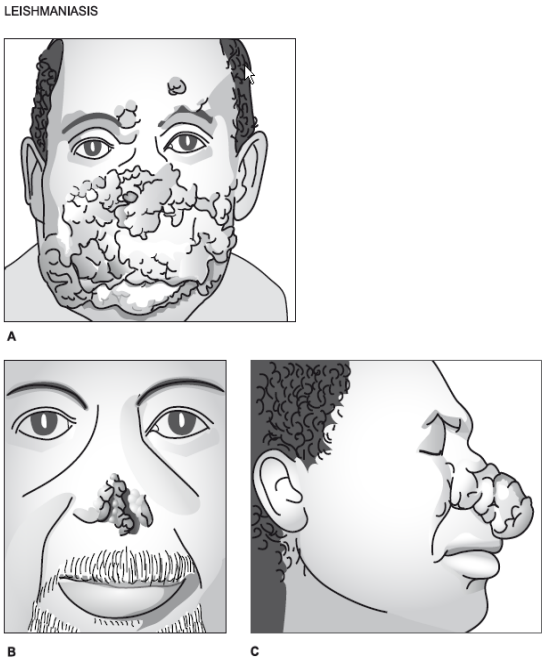


Fig. 29-22 LEISHMANIASIS OF NOSE AND LIPS.
Some examples: A, ulcerative form affecting glabella, nose, cheeks and lips. B, non-ulcerative form with hypertrophy of nasal skin and rhinophyma. C, destruction of nasal septum and of the *alar nasae.*
After Cohen RV, Aun F. Tropical Surgery, Karger Landes 1997.127-9, Fig 19.1a,2a,3a.

In the non-ulcerative form, persistent oedema, mucosal hypertrophy and upper lip fibrosis result in characteristic facies. The nasal bridge and tip collapse as the septum is destroyed, and nasal polyps may be present. Intense hypertrophy can lead to massive rhynophyma (29.20, 29-22).

In the ulcerative form, there is rapid destruction of the nasal septum from the front, and invasion of the *alae nasi*, as well as lips, tongue, palate, oropharynx and larynx. This is more common if there is also concomitant HIV disease.

(a) Diagnosis is confirmed by smears from skin and mucosa or biopsy, but signs are so characteristic in endemic areas that this may not be necessary.

(b) Treatment. Miltefosine 100mg od for 4wks is the best choice. Otherwise, liposomal amphotericin at 3mg/kg IV daily for 5 days with another single dose of 3mg/kg 6 days later is preferable because of resistance to antimony compounds.

(c) Reconstructive surgery is often required after the lesions have healed with extensive scarring.

29.19 Bronchial carcinoma

As a result of the greed of multinational companies and the inertia of governments, cigarette smoking is widely prevalent all over the world. An epidemic of smoking-related diseases has already started, among them carcinoma of the bronchus. About 75% of tumours involve the main bronchi, 10% are peripheral, and a few arise near the apex of the lung, whence they may spread to involve the sympathetic chain and the brachial plexus (Pancoast's tumour). About 50% are squamous cell, 30% are anaplastic (small cell), and 20% are adenocarcinomas: most peripheral tumours are of this latter kind, and their prognosis after surgery is relatively good.

The patient, who is usually an older man, presents with:
(1) A persistent cough.
(2) Haemoptysis.
(3) A low-grade pneumonia, as the result of a blocked bronchus.
(4) Pneumonia which fails to resolve.
(5) A solid lesion on a radiograph. Bronchoscopy is the critical investigation, and even with a rigid bronchoscope (29.14) it is possible to see and biopsy the lesion in about 75% of cases.

In countries where the disease is common and patients are aware of it, only about 20% of them are operable when they present, and of those who do survive radical surgery, only about 30% are alive 5yrs later. The chances of your being able to refer a patient for either radical surgery or radiotherapy are small. Radiotherapy is a useful palliative. Present combinations of cytotoxic drugs are of limited value.

You will probably find that most patients are inoperable when they present. So,
(1) Differentiate carcinoma of the bronchus from other, more treatable diseases which it may closely resemble, both clinically and radiologically, particularly ***tuberculosis***.
(2) Select the few 'coin-like' peripheral lesions amenable to surgery.
(3) Palliate and comfort the dying and their families.

(a) Investigations
Get an antero-posterior *and a lateral* radiograph of the chest, and a PCR test for tuberculosis (5.7).
Send sputum (after physiotherapy) for culture and AAFB.
Aspirate a pleural effusion for culture and AAFB.
Take a pleural biopsy (9.1).
Perform a bronchoscopy (29.14).

(b) Differential diagnosis includes:
(1) pulmonary tuberculosis
(2) low-grade or partly resolved pneumonia
(3) pulmonary fibrosis
(4) lung abscesses
(5) other solid tumours of the lung.

(c) Operability
The most favourable cases are those with a peripheral 'coin-like' lesion (usually an adenocarcinoma, but may be a tuberculoma, or a developing lung abscess). These may benefit from thoracotomy. Signs of inoperability include: involvement of the chest wall, involvement of the laryngeal or sympathetic nerves (Horner's syndrome: miosis, ptosis and hemifacial anhydrosis), widening of the mediastinum in a chest radiograph, secondary deposits (as in the cervical nodes), bony metastases, and a small cell carcinoma on biopsy. Operability may improve after radiotherapy, but will be difficult.

Bronchoscopic signs which suggest that a patient is not operable include: widening or flattening of the first 1·5cm of the main bronchus, widening of the carina, and distension of the trachea.

(d) Chemotherapy has a low priority. If there is an oat cell carcinoma, it will produce a remission and prolong life for 6-12 months. A few patients with small cell tumours survive much longer. Untreated, patients are likely to die in 2 months.

29.20 Other problems in the ear, nose and throat

(a) The ear

TINNITUS is a persistent noise, usually high-pitched, in the ear and is very frequently associated with hearing loss. Some medications, especially NSAIDs and anti-depressants, may be the cause. You can help patients mask the sound by advising them to put a ticking clock near the ear at bedtime, or to play music from a radio. *Sedatives are no help.* Very occasionally a specific pathology (an aneurysm or tumour) is the cause.

If a patient develops slowly progressive deafness in one ear, becomes unsteady on the feet, and has rare attacks of severe vertigo, suspect an ACOUSTIC NEUROMA (rare). Look for a loss of corneal sensation, slight facial paralysis, and an increased protein in the CSF. This needs expert intervention.

(b) The nose and paranasal sinuses

If a patient has a swelling on the nasal septum, it may be a HAEMATOMA or an ABSCESS. The same incision is suitable for both. Move the tip of the nose from side to side; you will see that swelling is continuous with the inferior margin of the nasal septum (the *columella*) on both sides, and is fluctuant.

Soak a length of 1cm ribbon gauze in 4% lidocaine, mixed with a few drops of 1:100,000 adrenaline, and place it over the red mucosal part of the septum. Wait a few minutes, and then make a vertical incision over the swelling. Remove a small piece of mucosa to enlarge the hole, and insert a drain, which you can remove the next day.

If there is a grossly thickened swollen nose, this is RHYNOPHYMA: the nose is oedematous, lumpy and irregularly swollen because of hypertrophy of sebaceous glands. Here you can shave off excess tissue and smooth it off using a sterilized disposable shaver, but *don't apply a skin graft.* In endemic areas, this may be *leishmaniasis* (29.18): take smears and a biopsy. Alternatively it may be *mucormycosis* which responds well to amphotericin B.

If there is a smelly nasal discharge with maggots, and epistaxis, this is myiasis from the deposition of eggs by flies attracted by pre-existing nasal diseases, especially in those with HIV disease. Facial cellulitis, palatal perforation, excoriation of nose and lips, and sinusitis may result. After using gentamicin and penicillin IV, spray the nose with 4% lidocaine and an ether nasal douche to paralyze the maggots and use a turpentine oil to drown them; then remove them manually. This is much helped if you have a nasal endoscope as the maggots may number hundreds and migrate to the deep recesses of the nasal anatomy! Often you need several removal sessions.

N.B. Some maggots digest live tissue, not just necrotic tissue!

(c) The throat (larynx)

There are 3 sounds of airway obstruction: stridor, stertor and wheeze. Listen carefully to the sounds of a patient with airway obstruction and try to differentiate these 3 sounds.

(1) STRIDOR is due to obstruction of the larynx, subglottis or cervical trachea. It can be both inspiratory and expiratory but is always worse in inspiration. It is a harsh sound.

(2) STERTOR is another word for 'snoring' and is due to obstruction at the base of the tongue. It may be due to something simple, such as secretions in the patient who is unconscious and unable to clear his throat, or something serious, such as a carcinoma of the base of the tongue.

(3) WHEEZE is expiratory and high-pitched and due to small airway obstruction as in asthma and chronic obstructive pulmonary disease.

If a patient of any age has the rapid onset of hoarseness and stridor, worse on inspiration, suspect ACUTE LARYNGITIS (not uncommon). Steam and antibiotics will usually achieve a cure. Tracheostomy may occasionally be necessary, but avoid it if possible, especially in a young child.

If there is a membrane in the throat, there is likely to be a streptococcal infection (common), or DIPHTHERIA (uncommon).

If a patient of any age has slow progressive hoarseness, leading to stridor which is worse on expiration, suspect a papilloma of the larynx, or a carcinoma in older smokers (both not uncommon, 29.17). Tracheostomy and endoscopic removal may be necessary. Biopsy an adult's lesion, and look for malignant change.

Recurrence is common in children, where the disease is related to aspiration of virus from genital condylomata. Symptoms may be confused with asthma, and deaths have occurred from asphyxia.

If a patient has stridor and increasing dyspnoea following extubation after having been previously intubated, suspect a tracheal stenosis. You may be able to dilate this endoscopically, but it usually needs excision.

If a patient of any age has sudden stridor, particularly on inspiration, after ingesting food, suspect a foreign body (not uncommon: 29.14).

If a patient of any age has sudden stridor, worse on expiration, following food, medicine or a sting, suspect ANGIONEUROTIC OEDEMA, causing soft tissue neck and laryngeal swelling. If there is stridor, use 1ml adrenaline (1:1000) IM. Otherwise, use an antihistamine or 200mg hydrocortisone IV. Tracheostomy may be necessary. The prognosis with treatment is good. Rarely, if there is no response, infuse 2 units of FFP, which contains the missing C1-esterase inhibitor. Advise patients about avoidance of the particular allergen, if known. Try to provide an ‘epinephrine-pen’ (auto-injector of adrenaline) for emergency home administration.

If a patient of any age has sudden severe stridor, usually without much hoarseness, suspect LARYNGEAL PARALYSIS due to infection, trauma, poliomyelitis, or nutritional deficiencies. If he has had a thyroid operation, suspect recurrent laryngeal nerve damage. Unilateral vocal cord palsy produces a characteristic prolonged wheezy cough. Re-intubation or a tracheostomy (29.15) may be necessary.

If a patient of any age develops a swollen oedematous neck related to dental sepsis, suspect LUDWIG’S ANGINA (6.11).

If a child, particularly, has progressive dysphagia, continual drooling from the mouth, stridor, cough, a red swollen epiglottis, and is ill and febrile, suspect ACUTE EPIGLOTTITIS (not uncommon), which is much more serious than acute laryngitis. If he is old enough to speak, he may have the characteristic 'hot potato' speech, which is different from the hoarseness of laryngitis. Use chloramphenicol or ampicillin IV. Be prepared to intubate him, followed if necessary by tracheostomy. *If he is not rapidly and correctly treated, the chances of death are considerable.*

If a child develops stridor with tonsillitis, suspect a peritonsillar abscess (6.7).

If a child develops stridor after ingesting an impacted fishbone, suspect a retropharyngeal abscess (6.8).

If a child (usually) has hoarseness and variable progressive stridor of rapid onset after fever, with severe dysphagia and a bleeding mouth and nose (rare), suspect GANGRENOUS PHARYNGITIS. Use IV antibiotics and oxygen. Feed him through a small nasogastric tube, and aspirate the pharynx periodically to remove blood and slough. Mortality is high.

If a child has had stridor and dyspnoea on exertion since birth, perhaps with hoarseness which is progressive with growth, suspect a LARYNGEAL WEB, a rare membrane across the laryngeal lumen close to the level of the vocal cords. Symptoms depend on the degree of stenosis. Tracheostomy may be necessary. Expert surgery can give good results.

If an infant or young child has sudden, spasmodic stridor, usually at night, which ends spontaneously with another deep inspiratory effort and collapse, suspect LARYNGISMUS AND TETANY (rare). He is normal between attacks. Use parathyroid hormone and calcium between attacks, and the prognosis will be good.

If a baby has stridor soon after birth, worse on any exertion or crying, but looks well and the cry is normal, suspect LARYNGOMALACIA (rare). Endoscopy shows a markedly folded epiglottis, with its aryepiglottic folds sucked in towards the larynx during inspiration to cause stridor. Reassure the parents that he will probably recover spontaneously between 3-5yrs.

30 The oesophagus

30.1 Foreign bodies in the throat

A patient with a foreign body in the pharynx,or oesophagus usually knows what has happened and is usually right. It can stick in the tonsils, the vallecula, the pyriform fossa (29-14), or in the lower hypopharynx. Most fish bones stick in accessible regions, usually the back of the tongue or tonsils. Foreign bodies seldom obstruct in the larynx itself, but an elderly, often intoxicated, diner may get a piece of meat stuck, as a result of which he gasps and collapses.

(a) Emergency treatment for choking

If you are present at the time and the patient is asphyxiating, sit him up, grasp the tongue with gauze, pull it forward, and tell him to keep his mouth open. Then if you can see it, *jam open the jaw*, and hook out the food bolus or foreign body with your finger. If you fail, insert a wide-bore needle (or a blade) through the cricothyroid membrane of the larynx to create an airway (cricothyroidotomy, 29.15): *this is not to remove the foreign body!* The smaller the patient is, the more difficult this will be. Follow this by laryngoscopy, and bronchoscopy (29.14), as soon as possible. *Avoid passing the bronchoscope through the tracheostomy*, which is difficult and dangerous. Only attempt it if the bronchoscope will not pass the cords.

If there is a piece of food in the larynx with persistent choking and inability to talk, try Heimlich's manoeuvre immediately: stand behind the patient, put your fist under the xiphoid and give a short sharp upward turn and thrust, whilst compressing the chest with your arms. This will exert a sudden pressure on the lungs, and may expel the food bolus with a rush of air, with immediate relief. If this fails, or the victim is too obese, sit him propped upon cushions on a chair with its backrest resting against the chest, and push him down forcefully against the back of the chair held rigid by others.

N.B. Don't try a Heimlich manoeuvre on a small child. Instead, lay him on your lap with the head hanging down, and make 5 hard slaps to the upper back; then turn him over and make 5 thrusts over the xiphoid. *Don't hold a child upside down without supporting the neck!*

If the presentation is early with 'something in the throat' without severe dyspnoea, take a careful history. Check if the foreign body is localized; where and how severe is the pain? Ask the patient to swallow 5ml water and look for a grimace. Feel the neck. Look carefully in the throat with a torch, tongue depressor, and laryngeal mirror. Is there surgical emphysema? If so, get a lateral view neck radiograph. Look for air in the tissues. If a large fishbone has stuck, you may see it, but you will not see a small one. *Don't confuse the hyoid bone* (27-19F) *for a foreign body!*

If there is no convincing evidence that it has lodged in the pharynx, and pain is mild, it probably only scratched the throat and passed on. Check for a grimace by giving 5ml of water to drink. If this is absent, treat expectantly. Encourage eating of dry bread. A small sharp object may readily pass through the entire alimentary tract without causing harm.

If you can see a foreign body in the pharynx, or there is air in the tissues, or if for any other reason you suspect it has lodged there, examine the pharynx under ketamine. Have good suction available. Use a laryngoscope carefully to search the tonsils, the valleculae, and the back of the pharynx. Take the opportunity to have a look at the larynx, even though a foreign body here does cause different symptoms (29.14). Grasp it with Magill forceps, or pull it up with a Foley catheter (30.2).

CAUTION!

(1) GA and relaxants are dangerous unless you can inflate the lungs. This will mean intubation, which may be tricky.
(2) Keep the head well down all the time, and the neck extended.

If you don't find it, proceed to oesophagoscopy.

If there is a perforation of the wall of the pharynx, spreading aerobic and anaerobic infection will be dangerous. If there is a large wound, examine it under GA and try to extract the foreign body if still *in situ*. Flexible oesophagoscopy (13.2) is helpful here. If there is cellulitis, necrotizing fasciitis, or an abscess in the neck, open up the soft tissues of the neck carefully. *Beware! The carotid artery may be injured!* Treat with IV chloramphenicol and metronidazole.

30.2 Oesophagoscopy

If you have a flexible endoscope (13.2), use it instead of the rigid instrument as much as possible. It is far less dangerous to the patient!

Passing a rigid oesophagoscope is easier than passing a rigid bronchoscope. An oesophagoscope looks like a bronchoscope except that it has no side tube for oxygen, and no ventilation holes at its distal end, because the patient does not need to breathe through it.

Fortunately, most ingested foreign bodies pass through the oesophagus unless they are sharp or too large, but if they get stuck, they have to be removed. Most (90%) stick in the upper 5cm of the oesophagus, just below the *cricopharyngeus* (30-1A) before the oesophagus enters the thorax. This is fortunate, because this is the easiest place from which to remove them. They are commonly coins, buttons, safety pins, or bones.

The patient may have almost no symptoms, or he may be distressed, refuse food, drool saliva, choke, gag, or cough (typically in paroxysms). If he is a child, he may merely 'fail to thrive'. If he is older, he may complain of pain, or the sensation of a foreign body behind the sternum.

If the foreign body is large enough to compress the trachea, he may have stridor, or episodes of cyanosis and recurrent pneumonitis (unusual).

If a foreign body gets stuck at the gastro-oesophageal junction, suspect achalasia (30.6).

If the patient is very young, or mentally incompetent, there may be no history that he has swallowed anything. Symptoms may have lasted hours or years. The diagnosis is usually obvious, but a foreign body which is missed can cause persistent dysphagia and loss of weight, so that you may suspect a carcinoma or oesophageal candidiasis.

RIGID OESOPHAGOSCOPY

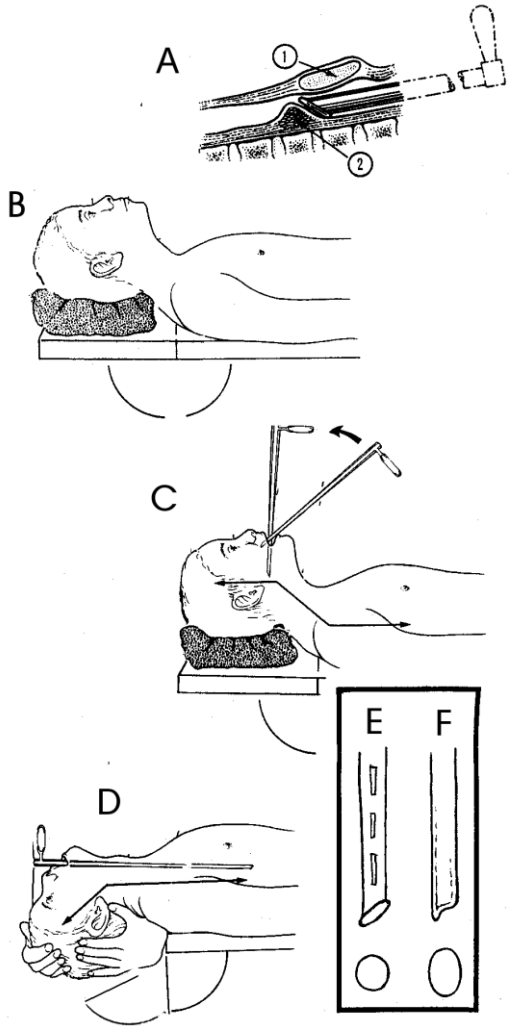


Fig. 30-1 CORRECT POSITION OF THE HEAD FOR RIGID OESOPHAGOSCOPY. A, the difficult part is passing the cricoid cartilage (1) and *cricopharyngeus* muscle (2). This will be easier if you keep the handle of the instrument up, so that it slides over this muscle. B, keep the head on a pillow with the neck flexed and the head extended in the 'sniffing position'. This position will allow you to pass the oesophagoscope as far as the deepest part of the thoracic kyphosis. C, introduce the oesophagoscope obliquely and move it vertically, as it reaches the pharynx. D, if you need to examine the lowest part of the oesophagus (less often necessary), straighten or slightly extend the neck, until he is in the sword-swallowing position. E, a rigid bronchoscope is circular, and has air holes. F, an oesophagoscope is oval, and has no air holes.
Adapted from Dudley HAF (ed) Hamilton Bailey's Emergency Surgery, Wright 11th ed 1977 p. 249,251 Figs 22.1,3 with kind permission.

A TRUE STORY. The examining surgeon at a nurse's training school (St Francis Hospital, Katete, Zambia, in its early days): "What instrument would you use for oesophagoscopy?" Enrolled nurse: "A sigmoidoscope." When it was explained that this was wrong, she repeated (correctly) that this was indeed the instrument that she had seen used at her rural hospital! LESSON If necessary, improvise.

EQUIPMENT needed
OESOPHAGOSCOPE, (a) infant, (b) child, (c) adult, with forceps and suckers that are long enough to go through them. If you don't have an oesophagoscope, you may be able to use a bronchoscope to remove coins from the oesophagus, or dilate a carcinoma before passing a Celestin tube (30-2E). *N.B. The more protruding beak of a bronchoscope is, however, more likely to perforate the oesophagus.*
BOUGIES, oesophageal, neoprene, standard set, alternate sizes only. The old-fashioned gum elastic ones are satisfactory.

THE SECRET OF
RIGID OESOPHAGOSCOPY IS TO
FLEX THE NECK AND EXTEND THE HEAD

RIGID OESOPHAGOSCOPY (GRADE 2.5)

INDICATIONS
(1) Removal of foreign bodies.
(2) Dilation for a benign or malignant stricture.
N.B. For diagnosis, flexible oesophagogastroscopy is much better and safer (13.2) because it does not need GA, and has much less risk of injury to the oesophagus.

(a) Radiographs
Many, but not all, foreign bodies are radio-opaque, but chicken and fish bones for example may be hard to see. Obtain a lateral view as well as the PA.
In the oesophagus coins lie in the coronal plane, so that you see their full diameter in a PA film. In the respiratory tract they lie sagittally, so that you see them from the side.
A barium swallow may be useful, but it makes oesophagoscopy soon afterwards more difficult; diatrizioate meglumine ('gastrografin') is better.

CAUTION!
(1) *Not all foreign bodies are visible on a radiograph.*
(2) If there is a swallowed foreign body that may cause trouble, get a radiograph of the whole abdomen and the pelvis also. *(It's obviously no use performing an oesophagoscopy for something that is already in the stomach!)*
N.B. Ultrasound may help in deciding if an object is in the oesophagus or bronchus.

(b) Preparation
Keep the patient's head on a pillow throughout. This will flex the neck. Then extend the head on the neck to achieve the 'sniffing position'.

If the foreign body is not sharp, pass a lubricated Foley catheter first using a laryngoscope into the oesophagus past the foreign body under ketamine anaesthesia; inflate the catheter balloon with enough water to occlude the oesophagus, and gently pull it out. The foreign body will come out with it, so you must grasp it quickly with Magill forceps lest it fall down the bronchus instead!
Make sure the neck is extended and the patient is 'head down'.

N.B. Don't inflate the balloon with air: the tension in the balloon will not be enough to extract the foreign body. If the Foley catheter is too big, use, if you can, a Fogarty balloon embolectomy catheter. This manoeuvre is much safer than using the oesophagoscope if you are inexperienced.

The same position will allow you to pass the oesophagoscope into the deepest part of the thoracic kyphosis. The most common reason for failure is insufficient flexion of the neck and extension of the head. The patient needs a GA: ask the anaesthetist to place his endo-tracheal tube on the left side of the mouth. Have an efficient suction machine and tubes ready.

If you need to view the very lowest part of the oesophagus (unusual), straighten, or slightly extend, the neck, so that the pharynx and the oesophagus are in a straight line to let the oesophagoscope pass (the 'sword-swallowing position'). Otherwise, keep it flexed. If your table does not have a headpiece that drops down, ask an assistant to hold the head over the end of the table to control its movement (30-1D): *this is not easy, and is potentially dangerous!*

Beware of the lady with the elaborate towering hairstyle: it will be safer to undo this, or just cut it off, than fail to extend her head.

Make sure suction is available and the light is working properly. Have biopsy forceps and foreign body graspers ready; check that these are long enough to pass through the oesophagoscope!

(c) Insertion

Aim the oesophagoscope vertically downwards at the uvula, keeping it at the back of the mouth against the palate. Angle it so as to pass the base of the tongue (aim at the foot of the pedestal of the table). When the larynx comes into view, avoid the midline, and pass it laterally, through one or other pyriform fossa (29-14), to reach the oesophagus, which is again in the midline. Going through the *cricopharyngeus* is the difficult part. If you have difficulty, pass a tube or bougie first, and use this to guide the oesophagoscope through.

CAUTION!

(1) *Never advance the oesophagoscope blind or forcefully*, or you may perforate the oesophagus.

(2) Keep the lumen of the oesophagus in the centre of your field of view, as you slide the instrument down.

REMOVING A FOREIGN BODY

METHOD

First try laryngoscopy. You may be able to feel the foreign body with a probe, and remove it with a long clamp.

If laryngoscopy and simpler methods fail, pass the oesophagoscope.

As soon as you can see the foreign body clearly (usually a coin, which will reflect the light and shine brightly as a transverse line), pass the biopsy forceps and grasp it firmly. *Don't try to use suction to pull it out!*

If it moves distally, withdraw the forceps, pass the oesophagoscope a little further, and try to grasp it again. When you have grasped it, bring it and the oesophagoscope out together. *Check for a second foreign body (or a third). Check for damage to the oesophagus!*

CAUTION!

(1) *The great danger is perforating the oesophagus*:

(a) usually at the level of the *cricopharyngeus* which keeps the entry closed, or

(b) lower down where the foreign body impacts: *beware, the aorta lies at this level!*

(2) Safety pins, bones, and lumps of food, such as meat, should, if possible, be removed by an expert: *they are particularly difficult and dangerous*. You should try to turn round a sharp object and pull it out with the point held distally, if you can do this safely. *Protect the oesophagus with the rigid scope, or with a sheath!*

(3) *If you cannot see the lumen of the oesophagus beyond, don't advance the oesophagoscope!*

(d) Difficulties with oesophagoscopy for removing a foreign body

If you don't have an oesophagoscope, you may be able to use a sigmoidoscope. Obviously you must clean it and soak it thoroughly in disinfectant before use! After you have identified the *cricopharyngeus*, use the obturator to negotiate it.

If a foreign body is too large to remove whole, as with an impacted denture, you may be able to break it and remove the pieces.

If you fail to remove a foreign body and cannot dislodge it distally *(but take great care: this may perforate the oesophagus, especially if the foreign body has sharp edges and is too large to fit inside the endoscope)*, you may be able to manipulate it into a bag. If the foreign body is blunt, you may be able to pass a Foley catheter beyond it, inflate the balloon with water and pull it out with that (as above). Otherwise, it will probably need removal, *via* an oesophagostomy, through the side of the neck (30-5).

If, soon after oesophagoscopy, there is pain in the neck, behind the sternum, or in the back, or severe dyspnoea, suspect an OESOPHAGEAL PERFORATION (30.7).

Look for air in the neck, pleural cavity or mediastinum (the earliest sign is a translucent crescent overlying the aortic knuckle) on a chest radiograph.

Consider using some water-soluble contrast medium ('gastrografin', not barium) and taking another film.

Arrange an immediate drainage (if the tear is in the cervical oesophagus, 30-5) or thoracotomy. Delay is likely to be fatal.

Try to insert a piece of sponge fixed by 2 sutures to a suction tube through a scope (rigid or flexible) to where the perforation is, and apply low-grade suction.

This is often remarkably successful. Change the sponge every 5 days till the hole is closed.

If an empyema develops, evacuate all fluid and air, insert intercostal drains, keep nil by mouth, and consider a feeding

jejunostomy (11.7): a gastrostomy is less effective because the feed can reflux into the oesophagus.

If the above symptoms are delayed, there is fever and the chest radiograph is normal, suspect MEDIASTINITIS. Antibiotic treatment is more likely to succeed. Fashion a temporary oesophagostomy tube drainage in the neck (30-5) and a gastrostomy (13.9) to rest the oesophagus. A feeding jejunostomy (11.7) in addition will allow you better to feed the patient.

If there is a retropharyngeal abscess, drain it (6.8).

If there is an oesophageal stricture, try dilating it (30.3), after extracting all the food accumulated proximally.

30.3 Corrosive oesophagitis and oesophageal strictures

Corrosive oesophageal damage is not uncommon in some communities, as the result of swallowing caustic soda (for making soap), sulphuric (battery) acid, or some other corrosive chemical. It also occurs after eating the tropical beach apple (manchineel), which has a number of highly irritant toxins. The victim is usually a child under 5yrs, or an adolescent; occasionally it is the result of a suicide attempt.

It is very useful to perform an oesophagoscopy to assess the initial damage. Often the strictures that result are severe and multiple. Keeping a passage open is vital. You can improvise bougies but remember rigid *dilation is always risky!* Do it with infinite care, otherwise you will perforate the oesophagus, and probably kill the patient. *Don't use balloon dilation.*

Oesophageal damage may also occur after excessive or misplaced injection of sclerosants for varices (13.2).

(a) The latent stage of oesophageal injury

If there is no severe complication immediately, and the pain and dysphagia improve, the victim may think himself cured. However, during the next 6-12wks, granulation tissue in the oesophagus steadily contracts, and becomes densely fibrous.

You should try to perform oesophagoscopy within 48h (43.6). If possible, use a flexible endoscope (13.2). *Don't try to inspect the whole length of the oesophagus with a rigid instrument*: use this for dilation only, though using a flexible instrument and a guide wire is much safer. Treat with cimetidine or ranitidine to lessen the risk of reflux oesophagitis and further scarring.

Persuade the patient to swallow a piece of fine string attached to a toffee, coated with honey. Keep the other end of the string outside the mouth; the string inside the oesophagus will maintain a lumen, however small, and may help with retrograde dilation later, by acting as a guide along which you can direct a bougie.

If injury to the oesophagus is likely to be severe (you can sometimes tell early by the bleaching caused in the mouth and pharynx), *don't hesitate to construct a feeding gastrostomy* (13.9), which you can use for retrograde dilation.

RETROGRADE OESOPHAGEAL DILATION

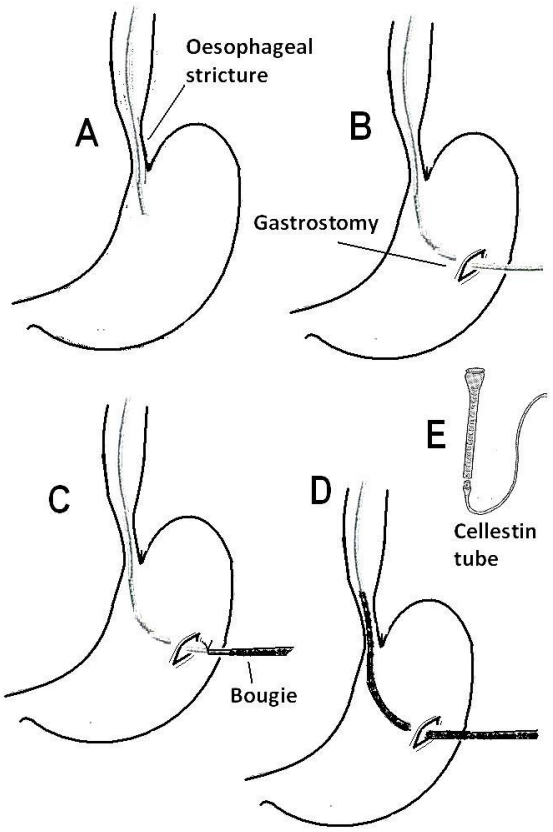


Fig. 30-2 RETROGRADE OESOPHAGEAL DILATION.
A, pass a guide wire through the stricture (if the string is not already in place). B, extract it through a gastrostomy. C, attach a well-lubricated bougie (dilator). **D, draw it gently upwards. E, attach a Celestin tube in the same way** (30.5).

(b) Retrograde oesophageal dilation (GRADE 3.3)

Dilate the oesophageal track with a bougie in retrograde fashion, using the string already in place (30-2C), 3wks after ingestion of the corrosive and then weekly for 3 months: *don't attempt >4mm dilation in one session.*

Then do it monthly for 3 months, and every 2 months for 6 months. If subsequent dilations are easy, remove the string. Perform a barium or 'gastrografin' swallow to monitor progress, and X-ray the chest, especially after a difficult dilation.

Try to provide high-energy liquidized feeds, and keep the oesophagus dilated by eating solid meals. Fizzy carbonated drinks help food go down.

N.B. Don't try to dilate multiple (>3), long (>5cm), or very tortuous strictures, or where there is a tracheo-oesophageal fistula.

(c) The chronic stage of oesophageal stricture
If the oesophagus is not dilated in the latent stage, the stricture will again get worse. The dysphagia gets worse, but because there is no pain, the patient often does not present until there is dysphagia for fluids. There is then hunger, thirst *and* weight loss, and even regurgitation due to distal obstruction in the oesophagus or stomach. The overspill of oesophageal contents, held up above the stricture, may cause aspiration pneumonitis.

Continue to dilate as often as is necessary, until you can pass a large bougie (preferably Ch40), at 4-monthly intervals, over a period of 1yr.

If you cannot pass a bougie by mouth, try the retrograde method (30-2).

(d) Reflux oesophageal strictures
Gastro-oesophageal reflux is common especially in the obese and associated with hiatus hernia; however, not everyone who has a hiatus hernia has reflux, and reflux does not necessarily imply a hiatus hernia. If the reflux of acid is severe and continuous, inflammation can lead to stricturing.

A barium swallow may give similar appearances to achalasia (30.6), and so you need endoscopy to differentiate between the two, and to dilate a stricture. This sort is, inevitably, at the lower third of the oesophagus, where dilation is especially hazardous.

(e) Differential diagnosis of benign oesophageal strictures
If there is profound wasting, and a barium swallow shows shouldering at the stricture, this is a malignant stricture (30.5).

If there are signs of HIV disease, and especially oral candidiasis, there is likely to be oesophageal candidiasis (30.4), which may be so thick as to block the oesophagus. Treat this with azole antifungal agents and fizzy carbonated drinks.

If there is regurgitation and retrosternal pain, without much weight loss, and a barium swallow shows a mega-oesophagus in a 25-35-yr-old, this is achalasia (primary, or related to Chagas disease: 30.6).

If she is a woman with iron deficiency, and a barium swallow shows a web (38.1d), this is Plummer-Vinson's syndrome, which is benign.

If there is severe retrosternal pain, and no evidence of an oesophageal fistula, especially if there is HIV disease, herpes simplex may cause ulceration. Treat with acicyclovir suspension 400mg tds for 2-3wks.

DILATING A BENIGN OESOPHAGEAL STRICTURE (GRADE 3.1)

If you can, use a flexible endoscope (13.2): it is *much* safer. Otherwise, to pass a rigid oesophagoscope, you need a GA and a short-acting relaxant, and intubation for controlled ventilation. Pass the oesophagoscope with care, using one of small diameter first. Suck out residual fluid and advance the instrument under direct vision. If entry is easy, and vision is poor with a narrow instrument, withdraw it, and try a broader one. Continue distally under direct vision, until you see the lesion, continuously sucking out further fluid. *Don't use force*. Note the position of the stricture in cm from the teeth. Note if the stricture is rough (likely to be malignant) or smooth (benign). Take a biopsy.

Try to introduce a well-lubricated bougie of size Ch10-12, with a long, strong monofilament threaded to its proximal end; this should pass easily with just a little resistance. If you have a guide wire, with a specially adapted dilator that runs over the guide wire, this will guide you down the proper channel.

Pass bougies of larger diameter gradually up to Ch24-30: some have screws to attach to the previously used bougie so that you don't have to manipulate its passage each time. Finally when the largest bougie has passed, remove the oesophagoscope.
N.B. Don't dilate small children's oesophagus >4mm.

CAUTION! If the bougie suddenly seems to encounter no resistance at all, you have perforated the oesophagus. Strictures at the lower oesophagus are particularly vulnerable because the oesophagus makes a slight curve when entering the stomach.

REMEMBER, DILATION MAY EASILY RUPTURE THE OESOPHAGUS!

DIFFICULTIES WITH DILATING A BENIGN OESOPHAGEAL STRICTURE

If you fail to pass the bougie, abandon the procedure and try again at the next opportunity: you will be surprised how often it goes easily then!

If the stricture remains impassable, try to arrange an oesophagectomy with remaining stomach or colonic replacement; this is formidable surgery unlikely to be readily available. In the meantime, provide nutrition *via* a gastrostomy (13.9) or jejunostomy (11.7).

If there is severe chest pain, odynophagia even for saliva, and shock, you have perforated the oesophagus (30.7).

30.4 Oesophageal candidiasis

A patient who is immunocompromised with a CD4 level <200/μl is at high risk of developing fungal infestation with candidiasis. Whilst it may occur with advanced cancer, steroid inhaler use, long-term antibiotics or chemotherapy, its appearance in the mouth is virtually pathognomonic of HIV disease. It may not be seen in the mouth but be found in the oesophagus. Initially you can see whitish patches

which later become confluent and thick, so much so that the whole lumen of the oesophagus may be blocked. Rarely the oesophagus may rupture. If candidiasis is limited to the lower oesophagus, symptoms may mimic peptic ulcer disease.

Treatment
Fluconazole 100mg od, miconazole 10ml qds, nystatin 500,000 units qds, or ketoconazole 200mg od for 2wks is usually effective, if the patient can swallow. Unblocking the oesophagus with fizzy carbonated drinks may be effective; otherwise perform endoscopy (13.2). The endoscope easily gets blocked with thick *candida*, so do these cases last on your list! If this treatment fails, you can try diethylcarbamazine 100-150mg orally bd for 10 days.

Relapse is frequent unless you continue a prophylactic dose or start ARV therapy.

30.5 Oesophageal carcinoma

Carcinoma of the oesophagus is one of the most common cancers in Iran, China and parts of Southern Africa, especially Malawi. It may be associated with an *aspergillus* toxin, papilloma virus, the chewing of tobacco and lime ash (nass), exposure to opium residues, exclusive eating of maize affected by a fungus, together with vitamin deficiencies; the role of nitrosamines is less well defined. In many parts of the world it is the commonest cause of dysphagia.

(a) Presentation, usually in a male and >45yrs, is with:
(1) Progressive dysphagia, first for solid food and later for thin foods and even water. If you ask the patient to point to the site where food sticks, it will usually correlate well with the site of the lesion.
(2) Regurgitation; this is common, except in the early stages; the patient may describe it as vomiting.
(3) Hunger, which can be very distressing.
(4) Weight loss.
(5) Coughing on swallowing, due either to a tracheo-bronchial fistula, or to spillage from the oesophagus, through the larynx into the trachea. This happens when the lesion is in the upper or middle ⅓ of the oesophagus.
(6) Pain is a late symptom, and is due to spread into the mediastinum.

EQUIPMENT needed
TUBES, oesophageal, Celestin, with guide.
TUBES, Procter-Livingstone, 10 mm diameter; length 11, 15 and 19cm, unflanged or flanged (30-3E,F).
STENTS, self-expanding.

Try to confirm the histology by doing an oesophagoscopy to biopsy and/or dilate the stricture (30.2), and assess its operability. Radiotherapy is a useful palliative. Chemotherapy is not a viable option in most circumstances. Palliate an inoperable carcinoma with a stent or tube by the antegrade method (*e.g.* Procter-Livingstone type, 30-3) or by the retrograde method via a gastrostomy (*e.g.* Celestin type, 30-2). This will allow swallowing mushy food and fluids, and relieve distressing hunger and dehydration. It will allow survival up to 18 months in relative comfort, and death may well be from other complications without the tube becoming blocked. Palliative radiotherapy can be given with the tube *in situ*. *Don't consider radiotherapy before a tight stricture is dilated:* oedema will make it worse initially.

(b) Radiographs
Barium swallow will show a distinctive narrowing, with shouldering of the edges (apple-core lesion, 38.1).

(c) Operability
The few cases suitable for oesophagectomy must have:
(1) A lesion <5cm long on barium swallow (microscopically, the tumour may extend double this length in each direction), which narrows the oesophagus by <50% and shows no axial deviation.
(2) No mediastinal widening (no enlarged nodes).
(3) No deformity of the trachea, carina, or left main bronchus on bronchoscopy.
(4) No evidence of a tracheo-oesophageal fistula.
(5) No pneumonia, chronic bronchitis, TB, or HIV disease.
(6) No significant weight loss (>10%).
(7) No cardiac disease.
(8) Good general health, and young age.
Don't refer cases which don't meet these criteria.

CAUTION! You should not be tempted to create a feeding gastrostomy, which will only prolong the patient's agony, and will not solve the distressing problem of the inability to swallow saliva.

(d) Stenting for carcinoma of the oesophagus
(GRADE 3.2)
INDICATIONS
Inability to swallow. Even if there is total obstruction, you may still be able to pass a tube.

CAUTION!
(1) The earlier the presentation, the easier it will be to pass the tube. *Don't wait until dysphagia is complete*: you may not be able to dilate the stricture.
(2) There is no substitute for being taught this procedure by an expert.
(3) *Don't attempt this if the patient has recently had radiotherapy:* the oesophagus becomes very friable and perforation is then a very great risk. Wait for 4wks; *even then the danger is great.*

Check the position and length of the tumour on the barium radiograph, and also note any deviation or fistulae present, which may make placing the tube difficult.

N.B. Using a flexible endoscope (13.2) and placing a stent is much easier and safer for the patient.

STENTING OESOPHAGEAL CARCINOMA

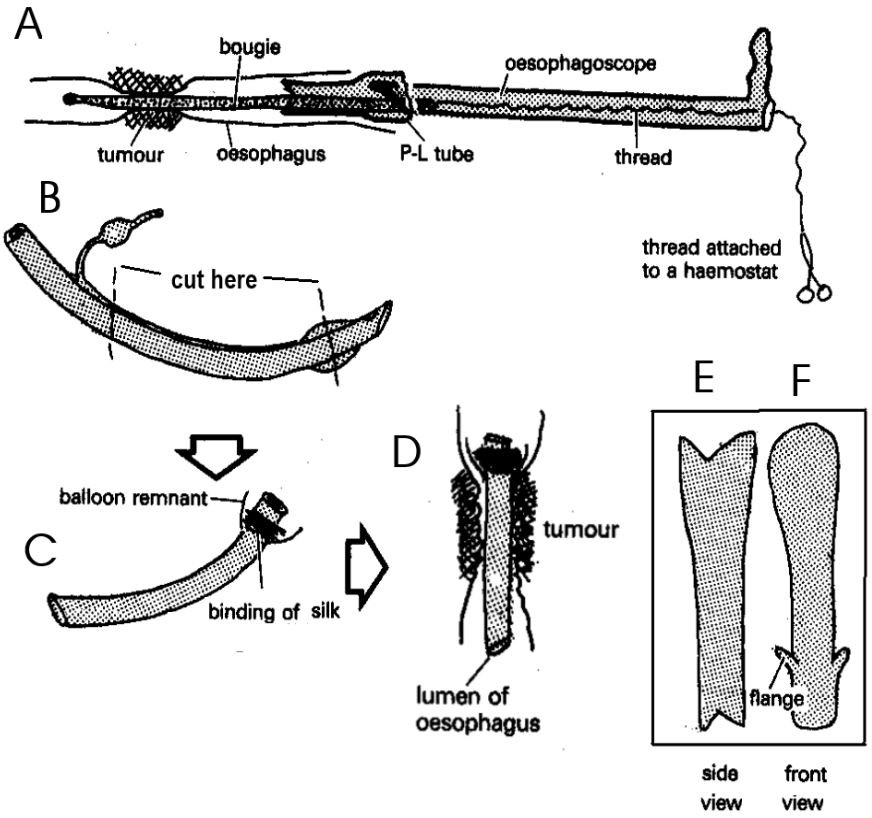


Fig. 30-3 INSERTING A PROCTER-LIVINGSTONE (P-L) TUBE. A, pass the bougie, with a thread attached to it drawn through the oesophagoscope, which is being used to insert a P-L tube. B,C, tracheal tube cut to make an improvised oesophageal tube. D, improvised tube in place with end bevelled. E,F, anterior and lateral views of a flanged P-L tube, which prevents upwards slippage. *N.B. Some P-L tubes are unflanged.*
Kindly contributed by Ian Kennedy.

Using the rigid oesophagoscope, aim to place a P-L, similar or improvised tube (30-3B,C) antegrade through the dilated oesophageal stricture; choose a tube of the right length (11cm, 15cm or 19cm). Make sure the stricture is dilated to an adequate width to accommodate the tube. Leave the dilating bougie *in situ*, and make sure a strong monofilament thread (fishing wire) is attached to it, so you can pull it out easily if necessary. Lubricate the tube well inside and out, and slide it over the end of the bougie. Guide the tube into the patient's mouth and pharynx with your left hand, and engage the oesophagoscope in the cupped upper end of the tube.

Push both gently over the bougie down the oesophagus. When the tube reaches the tumour, you may feel some resistance. Now push more firmly with a gentle twisting action.

You will feel the P-L tube passing through the stricture, until you feel its cup being stopped by the upper end of the stricture. Watch the cm scale on the oesophagoscope, as it passes the teeth, so that the tube rests not >1cm than the distance to the tumour measured previously.

Remove the bougie, untwist the oesophagoscope slightly to disengage it from the cup of the P-L tube. Look down the oesophagoscope to see that the tube is in place. Suck out blood and tumour debris. Remove the oesophagoscope under direct vision.

The P-L tube is unsuitable for very high lesions (<15cm from the incisor teeth) and low lesions (>35cm from the incisor teeth), because of the problem of acid reflux. *The mortality of insertion is c.10%.*

N.B. There are similar, safer and more reliable and lasting expandable stents which can be inserted in the same way, with the appropriate introducer; these are now not so expensive.

If you use a Celestin-type (retrograde) tube, pull it down through an open gastrotomy (30-2C). Occasionally the stricture cannot be dilated from above, but it might be possible from below. *However, the mortality of this procedure is significantly higher (20%).*

POST-OPERATIVELY
Continue IV fluids till oral intake is satisfactory: unless there are problems with the procedure, swallowing is usually possible immediately. Advise the patient, in his enthusiasm to resume eating, to chew his food well. Remember to advise laxatives, as the liquidized food taken subsequently will tend to constipation. It is best to avoid stringy vegetables.

(e) Difficulties with oesophageal tubes
If you make a false passage or tear the stricture, mediastinitis will follow and usually be rapidly fatal within 24h. Life without a tube is so intolerable that you will have to take this risk. Surgical intervention is very unlikely to be successful.

If the bougie passes, but the tube will not pass, you may need considerable force, even after what seems like good dilation. Only one diameter (10mm) of P-L tube is made, and it does not pass every stricture. You can make a home-made tube (30-3B,C). Take a soft plastic endotracheal tube of suitable size, cut off the tip with half the balloon. Wind thick silk or nylon under the balloon remnant to make a bulge. Cut the tube long enough for the stricture, and bevel the other end by cutting it obliquely, to make it easier to pass.

With the oesophagoscope in place, pass the improvised tube with long forceps under direct vision until its bulbous end is snug up to the top of the carcinoma. It will be possible to take thick liquids at least.

Or, get a long piece of wire from your workshop which will just pass down the whole length of a Ch18 nasogastric tube. Lubricate it well and pass it through the tube. Pass this wire stylet and tube through the oesophagoscope, through the stricture and into the stomach. Remove the stylet and

oesophagoscope. Pass a long nasotracheal tube down the nose, recover its distal end from the throat, and bring it out of the mouth. Push the end of the nasogastric tube through the nasotracheal tube out through the nose. Remove the nasotracheal tube. Tape the nasogastric tube to the cheek. Bandage the elbows in extension with rolled newspaper to prevent removal of the tube on recovery from GA.

N.B. In order of increasing efficacity are: nasogastric, improvised, Celestin, P-L tubes, but by far the best is a self-dilating stent.

CAUTION! Don't try to pass the wire stylet down on its own.

If you push the whole tube past the stricture (unusual), you may be able to pull it back with strong forceps. If this fails, leave it: it will pass per rectum!

If the patient regurgitates the tube, this is a nuisance, but not a disaster. If possible, replace it by a flanged tube (30-3E,F). The shorter the tube, the better it works, but the more easily it slips out. If it is too short, the tumour may grow over the end and obstruct it. Flanged tubes are more likely to stay in place.

A tube may displace if the patient vomits after the initial procedure, so keep him sedated with an anti-emetic.

If the tube blocks, try fizzy carbonated drinks to unblock foodstuffs, which are usually the cause; if this fails, perform an OGD or oesophagoscopy (13.2, 30.2).

30.6 Achalasia

Sometimes, in a patient of 25-35yrs, there is dysphagia, regurgitation and retrosternal pain; a barium swallow will initially show tapering of the distal oesophagus, which looks like a benign stricture; later the proximal oesophagus enlarges and ultimately may become enormous. There is, however, no blockage: the defect is in the function of the myenteric Meissner and Auerbach plexuses at the gastro-oesophageal junction.

This may be primary, or arise as a result of infestation by *Trypanosoma cruzi* in Central and South America (Chagas disease). In this case, other organs are commonly also affected: the heart in >30%, the pylorus in 20%, the colon in 15% and the gallbladder in 7%. Squamous carcinoma of the lower oesophagus develops in c.5% of Chagas mega-oesophagus.

It is rare in children, and does not respond readily to balloon dilation.

You can recognize 3 stages of the disease process:
(1) *incipient*, where the dilation of the oesophagus has not yet started and barium swallow appearances mimic a benign oesophageal stricture,
(2) *non-advanced*, where dilation is <7cm diameter,
(3) *advanced*, where there is gross dilation >7cm and atony of the oesophagus.

If you can get it, endoscopic injection of 1ml of 20IU *botulinum* toxin in each of the 4 quadrants at the lower oesophageal sphincter gives improvement in >50% of patients.

(a) Investigation

Barium swallow radiography gives a characteristic picture of tapering of the oesophagus in:
(1) oesophageal dilation
(2) mega-oesophagus
(3) food remnants showing as 'filling defects'.
Oesophagoscopy demonstrates no evidence of an obstructive lesion.

(b) Equipment

You should use balloon dilation for the incipient stage. In an endemic area, you should obtain the instrument designed by Pinotti from São Paulo: this is a plastic catheter with a 10cm-long heavy metal tip and a cylindrical balloon attached to the body of the catheter (30-2).

N.B. This is not the same as a Sengstaken tube (13-14).

(c) Balloon dilation of incipient mega-oesophagus (GRADE 2.4)

INDICATION. Incipient mega-oesophagus.

Fast the patient for 12h. Sit him upright and spray the oropharynx with 4% lidocaine. Introduce the instrument orally and wait till the patient feels the mercury tip has entered the stomach (30-4A). Inject 50ml water into the balloon (30-4B) and gently pull it up against the cardia (30-4C), so that it is anchored there. Then gradually inject a further 50-250ml water until the patient feels pain (30-4D), keeping it inflated for 5mins.

Then empty the balloon until only 50ml are left, thus leaving the balloon in position. Inflate the balloon twice more at intervals of 30mins, and then remove it.

(d) Difficulties with balloon dilation

If there is bloody vomiting after dilation, suspect a mucosal tear. This should heal spontaneously (30.7); if not, re-inflate the balloon with half the volume of water to compress the oesophageal mucosa for 5mins, deflate and observe.

If there is persistent retrosternal pain and dysphagia, suspect an oesophageal perforation. This should not occur if you stop distending the balloon when the patient feels pain. Confirm the perforation with a 'gastrografin' swallow. If present, an urgent laparotomy to close the perforation is necessary (30.7): *delay will be fatal*.

If symptoms recur (50% in 5yrs), you can repeat the procedure unless mega-oesophagus has developed.

BALLOON DILATION OF MEGA-OESOPHAGUS

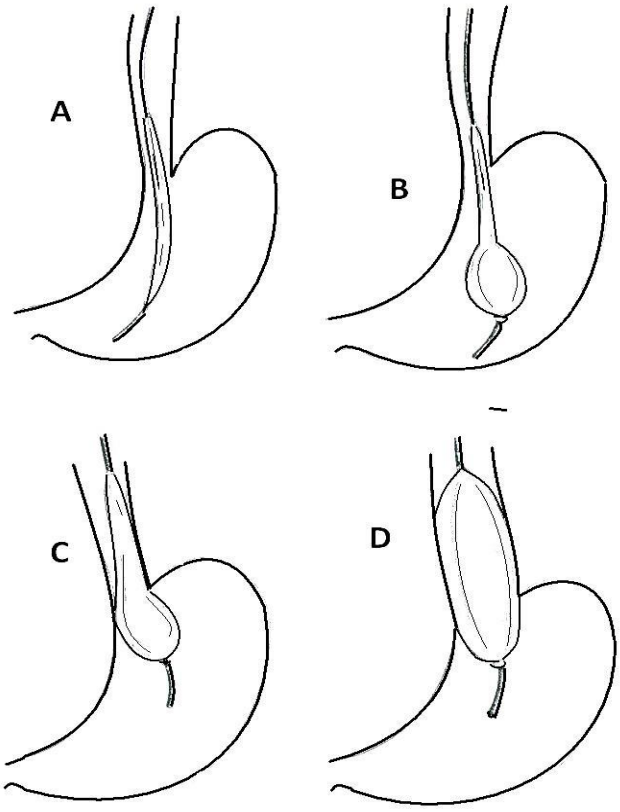


Fig. 30-4 TECHNIQUE OF BALLOON DILATION IN INCIPIENT MEGA-OESOPHAGUS.
A, introduction of balloon. B, after injection of the first 50ml water. C, anchor the balloon in the cardia. D, inject water gradually until the patient feels pain.
After Cecconello I, Pinotti HW. South American Trypanosomiasis (Chagas Disease) in Textbook of Tropical Surgery, Westminster 2004, p.986 Fig 221.2.

(e) Management of mega-oesophagus

The *non-advanced* mega-oesophagus <7cm diameter needs an oesophageal myotomy (Heller's operation) with a partial gastric fundoplication (30-6B), to prevent post-operative acid reflux.

The *advanced* atonic mega-oesophagus >7cm diameter needs an oesophagectomy.

30.7 Oesophageal rupture

Apart from iatrogenic damage at oesophagoscopy (30.2), rupture of the oesophagus is rare. It can occur in serious trauma in road accidents (usually head-on collisions), and from penetrating injuries, including foreign bodies. These are likely to be fatal. Rarely, the oesophagus can rupture spontaneously (Boerhaave's syndrome).

(a) Iatrogenic oesophageal damage

Injury to the oesophagus is most likely in the young or old, in the latter because of cervical osteophytes sticking up posteriorly distorting the oesophagus. The laceration occurs in the pharynx or cervical oesophagus just above the *cricopharyngeus* (30-1).

Instrumentation to remove a sharp foreign body and dilation of strictures are the other common causes of damage further down the oesophagus.

Symptoms will depend on the extent and position of the laceration. A substantial oesophageal injury causes severe pain and quickly develops mediastinitis or peritonitis, which present as septic shock.

Occasionally, a foreign body does not cause full oesophageal rupture. If there is a mucosal tear (Mallory-Weiss syndrome), there is only bleeding; occasionally there is a small breach that allows a leak of organisms. In such cases aggressive treatment and a feeding jejunostomy (11.7) may save the patient (43.6).

NEVER USE BARIUM IF YOU SUSPECT AN OESOPHAGEAL LEAK

DRAINING THE CERVICAL OESOPHAGUS

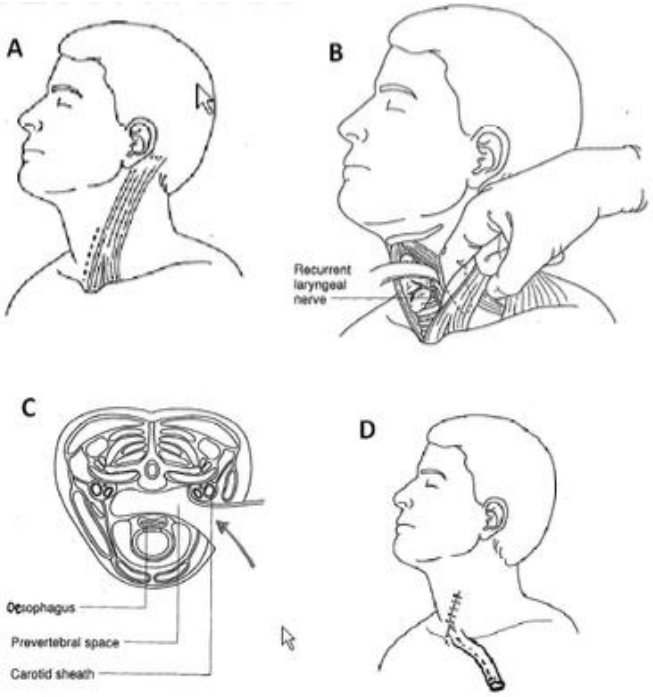


Fig. 30-5 DRAINAGE OF THE CERVICAL OESOPHAGUS.
A, make an incision parallel to the anterior border of the *sternomastoid*, from the level of the cricoid to the sternal notch. B, retract the *sternomastoid* and carotid sheath laterally with a finger, and the trachea and thyroid medially. C, dissect bluntly along the pre-vertebral fascia avoiding damage to the recurrent laryngeal nerve. D, insert a soft tube into the oesophagus if the hole is large, or a drain adjacent to it to pass through the wound.
After Greenfield LJ et al. Surgery, Scientific Principles & Practice, 2nd ed 1997 Lippincott-Rowen p.720 Fig 19-25.

(b) Spontaneous oesophageal rupture (Boerhaave's syndrome)

The patient, who is usually 20-40yrs, vomits after a heavy meal, and has an intense pain in the abdomen and left (rarely the right) chest radiating to the neck. He is intensely thirsty, but sips of water make the pain worse. Feel and listen with a stethoscope for surgical emphysema (a fine crackling) in the neck or chest. Check for absent breath sounds or hyper-resonance in the left chest. The upper abdomen may be rigid.

Radiographs show air in the mediastinum and soft tissues of the neck (usually the left side) and a pleural effusion occasionally with a pneumothorax. Confirm a rupture by repeating the radiograph after ingestion of 10-20ml of 'gastrografin'. This may show a leak.

N.B. Don't use barium as this may cause severe soft tissue inflammation.

The main differential diagnosis is a perforated peptic ulcer, but here the pain comes before the vomiting. When the oesophagus ruptures, the pain comes with vomiting. Other differential diagnoses include myocardial infarction, spontaneous pneumothorax, and acute pancreatitis.

CAUTION! Early on there are no clinical or radiographic signs in the chest; these come later when treatment may be too late. Confirm the perforation with a 'gastrografin' swallow.

Resuscitate with IV fluids and pass a nasogastric tube (check radiologically that it is not in the mediastinum). Keep the patient nil orally, start IV chloramphenicol and metronidazole antibiotics and insert bilateral chest drains.

If the perforation is in the distal oesophagus, within 12h of the perforation, perform a GASTRIC OESOPHAGEAL PATCH (GRADE 3.4).

METHOD

Make an upper midline laparotomy, extending the incision, if necessary, as a T in the subcostal region. Pull down the stomach to expose the lower oesophagus. Divide the short gastric arteries to free up the gastric fundus. The oesophageal tear is usually longitudinal just above the oesophago-gastric junction, slightly on the left. Close it with a diamond-shaped stomach patch. Put a continuous suture along the left edge of the oesophageal rupture and the right edge of the gastric patch you have outlined (30-6A). Then place several loose interrupted sutures along the right edge of the oesophageal rupture and the left edge of the gastric patch. Pull these sutures tight and tie them. Now wrap some of the posterior wall of the stomach round the oesophagus as in a fundoplication (30-6B), and suture the posterior gastric wall around the front of the oesophagus to the anterior gastric wall, allowing enough room for the oesophagus within. Cover the repair with omentum if you can.

Alternatively, for a higher perforation, use a piece of diaphragm to patch the hole: it has a good supply and you can readily make a pedicle of this (but remember its length should not be >2 times its width).

If the perforation is higher up still, a neck exploration or thoracotomy, oesophageal repair and mediastinal drainage will be needed, which may well be impossible to organize. You might just save the patient by draining the upper oesophagus in the neck (30-5) and performing a gastrotomy (13.9) and/or jejunostomy (11.7).

LOWER OESOPHAGEAL RUPTURE PATCHING

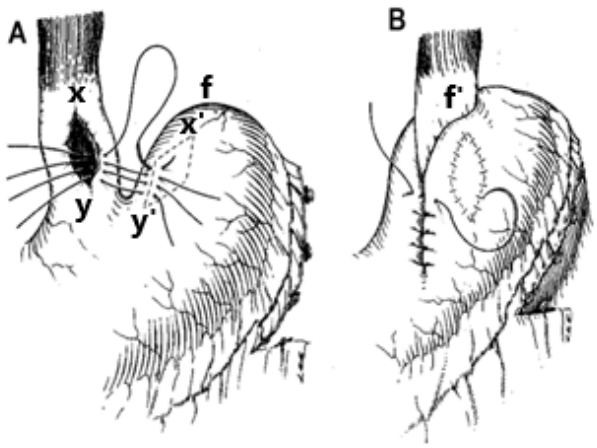


Fig. 30-6 PATCH OF A DISTAL OESOPHAGEAL RUPTURE.
A, use the stomach, so that the tear, xy, is covered by x′y′. B, the gastric fundus, f, then comes to f′. Free part of the posterior wall, by dividing the short gastric vessels, and so make a wrap round the oesophagus.
After Dudley HAF (ed) Hamilton Bailey's Emergency Surgery, Wright 11th ed 1977 p.252 Fig. 22.4, with kind permission.

(c) Oesophageal tear (Mallory-Weiss syndrome)

Following a severe bout of vomiting, a patient may suddenly produce fresh blood in the vomitus. Importantly, the vomiting starts without blood in it, unlike the haematemesis of a peptic ulcer. This is more likely to occur if there is a blood-clotting disorder, or the patient is taking anti-coagulants.

The cause is a small laceration of the mucosa of the lower oesophagus; it normally heals spontaneously, once the vomiting has stopped. You can then not usually see it at subsequent endoscopy.

Distinguish the oesophageal tear from varices (13.6).

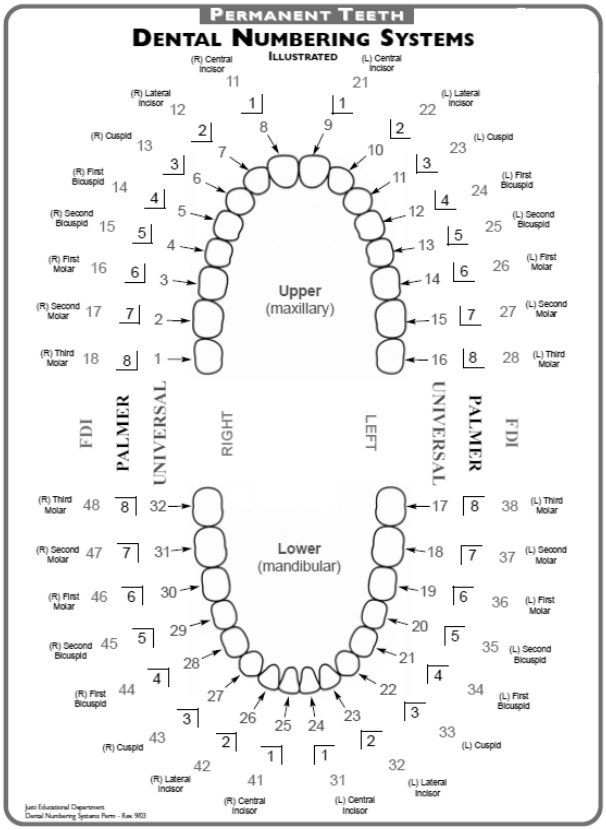
N.B. A more substantial tear may cause problems described above.

31 Dental & oral surgery

31.1 Introduction

Various dental problems may lead to serious illness; where there is no dentist, periodontologist, or oral surgeon, you may need to intervene yourself. Dental abscesses are described elsewhere (6.9). *Don't forget that a hospital can play a key role in dental health and education;* improved oral hygiene is one of the main ways to combat caries and periodontal disease, and to keep the community's teeth from falling out. When treatment is needed, make sure dental auxiliaries are trained and available for the community to provide care at an affordable cost. Try to get the WHO Basic Package of Oral Care.

DENTAL NUMBERING SYSTEMS (PERMANENT TEETH)



TOOTH NOMENCLATURE. Primary (baby) teeth are numbered in the Universal system as A-T (in the same order as the adult system), in the International (FDI) system as 51-55, 61-65, 71-75, 81-85 according to the quadrant starting top right clockwise, and in the Zsigmondy (Palmer) system as $\overline{5}|\overline{7}\ \overline{6}|\underline{8}$ A-E accordingly.

If you are not sure, it is best to describe the teeth as incisor (x2), cuspid or canine (x1), bicuspid or premolar (x2), and molar (x3).

Tooth nomenclature is complicated. The 'Universal' system (used in the USA) uses successive numbers starting at 1 with the tooth furthest back on the top right, continuing to the top left, and then from the bottom left to right. The International (FDI) system divides the mouth into 4 quadrants (1:top right, 2:top left, 3:bottom left, 4:bottom right). The Zsigmondy (Palmer) system uses a cross to designate in which quadrant the tooth lies, so $\underline{|}1$ is the 1st upper tooth to the left of the midline.

N.B. The difficulty inserting the sign before the tooth number has made this system unpopular.

(a) Anaesthesia for operations round the mouth

As so often, much of what you can do will be limited by your anaesthetic skills, or those of your assistant. You can however do much with dental, lingual, mandibular, maxillary or pterygopalatine blocks. Be familiar with the anatomy of the jaw. *Don't infiltrate LA fast, especially over the upper incisors:* the small space available distends quickly and will produce more pain if filled quickly than the pain from the bad tooth!

You can also do many procedures under ketamine; it is likely to be safer than an inexpert GA, especially for babies and children under 2yrs, who would need intubation. Bleeding and the risk of inhalation are the biggest risks from oral surgery under GA; so *always have a swab on dissecting forceps, and a sucker, with a catheter at its tip, instantly ready*.

(b) Cleaning the mouth before surgery

The mouth harbours millions of organisms, including anaerobes. Make sure the patient brushes his teeth before surgery. *Do it yourself (again) in theatre before operating on his mouth as a precaution!*

SOME DENTAL INSTRUMENTS

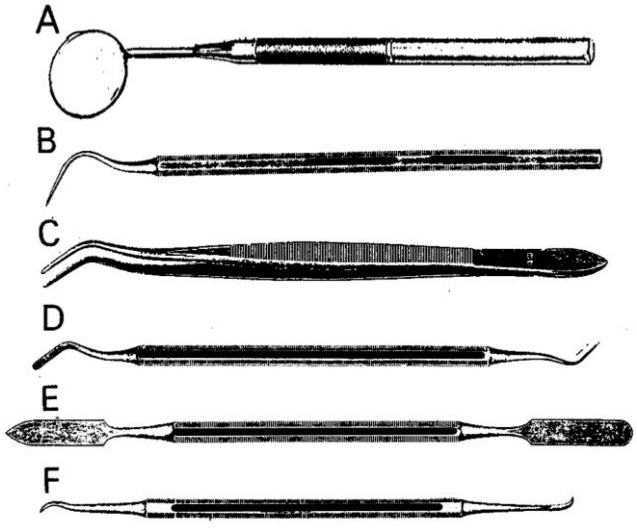


Fig. 31-1 SOME DENTAL INSTRUMENTS (shown from the side). A, mirror. B, probe. C, forceps. D, 'plastic instrument' for putting filling into a tooth. E, spatula (with end-on view). F, Cumine scaler for plaque removal (with end-on view).
From the Ash instrument catalogue.

EQUIPMENT needed
CHAIR, operating dental.
A dental chair is expensive. If you don't have one, you may be able to adapt a strong chair and fit it with a rest to support the patient's head when it is pushed firmly backwards, especially when you extract upper teeth.
DENTAL MIRROR, serrated handle with #4 size plane head (31-1A).
DENTAL PROBE, single-ended (31-1B).
DENTAL DRESSING TWEEZERS (31-1C).
'PLASTIC INSTRUMENT' (31-1D).
This is for inserting filling material into a cavity.
SPATULA, metal, for mixing fillings (31-1E).
SCALER, dental, Cumine (31-1F).
DENTAL MATERIALS, clove oil for dental use. Zinc oxide powder. When mixed together on a glass slab with the spatula listed above, these make an effective analgesic and a mildly antiseptic dressing for a dental cavity.
SUCTION MACHINE. This is essential, to prevent aspiration of liquids, pus or blood.

DETAILS OF HEALTHY & DISEASED TEETH

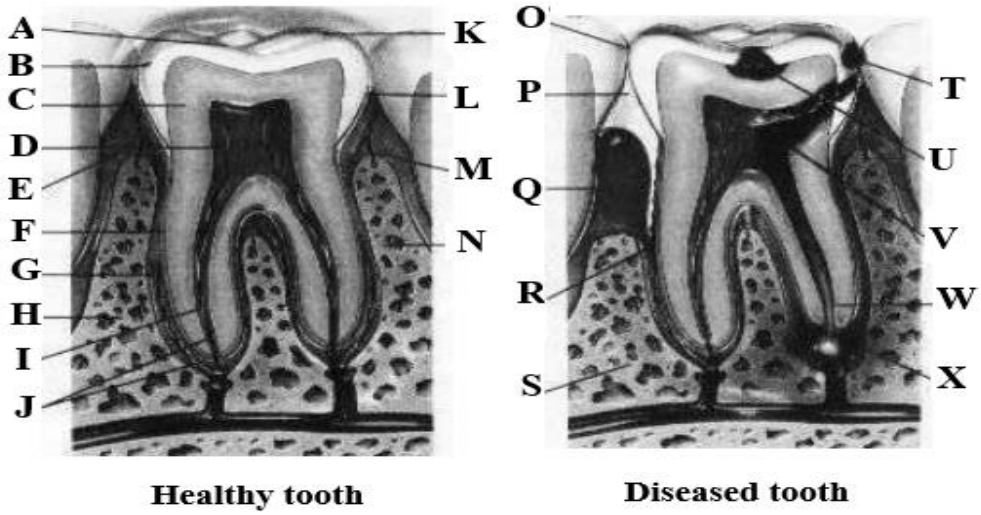


Fig. 31-2 DETAILS OF TEETH.
Healthy tooth; internal structures: (A) fissure. (B) enamel. (C) dentine. (D) pulp. (E) gum (gingiva)**. (F) cementum. (G) periodontal fibres. (H) alveolar bone. (I) root canal. (J) blood and nerve supply at root apex. External features: (K) white, shiny enamel (a crown is fixed onto this). (L) no space between tooth and gum. (M) firm, pale gums. (N) bone supporting tooth.**
Diseased tooth; stages of periodontal disease: (O) plaque (bacteria) forms and infects gum. (P) gum recedes and forms a pocket for food and bacteria to collect. (Q) gum becomes inflamed (gingivitis), bleeding often. (R) periodontal fibres destroyed; abscess develops. (S) bone destroyed (periodontitis) and tooth becomes loose and may fall out. Stages of dental decay: (T) bacteria in plaque convert sugar to acid. (U) acid destroys enamel (caries). (V) decay spreads to dentine, which becomes sensitive to heat and cold, and infection spreads to pulp cavity. (W) pulp infection spreads down root canal. (X) peri-apical root canal abscess develops.
After Ahmed MAM, Abdel-Latif, MMM in Textbook of Tropical Surgery, ed Kamel R, Lumley J, Westminster 2004 p.320 Fig 82.1.

31.2 Gum disease

The gums of healthy teeth cover their necks and those of adjacent teeth (31-3G). If gums are diseased by periodontitis, they recede and expose the necks of the teeth (31-3H,I), which ultimately become loose and fall out. Besides causing sore, bleeding gums, periodontal disease causes more lost teeth in many communities even than caries. It may be caused by *actinomycosis*.

Periodontal disease is the result of a vicious circle. Food tends to accumulate between a tooth and its gum, and causes the gum to slowly recede. This makes the pocket larger, so that food accumulates even more easily. In more severe cases the diseased gums swell, bleed easily (gingivitis), and discharge pus (pyorrhoea), often with severe foetor (31-2). Gingivitis is common in early pregnancy (<12wks), and scurvy (vitamin C deficiency), and may result in severe necrotizing ulceration with HIV disease.

Ingestion of food increases mouth acidity, which attacks the enamel lining of the teeth. Saliva exists to buffer and wash away acid, but if there is excessive sucrose and little fluoride, an exopolymer of extracellular polysaccharide matrices in a viscous hydrated phase builds up as a very sticky biofilm. This is known as plaque, under which the tooth continues to disintegrate.

The prevention of gum disease, and most of its treatment, is improved oral hygiene: better tooth-brushing (31-3D,E), increase of fibre and reduction of sugar content in the diet, and when necessary, scaling to remove hardened plaque that has accumulated in the crevices (31-3J).

You may not find yourself scaling many teeth, but there must be someone in your hospital who could do this, and you should be able to teach how to do it properly.

(a) Preventive dentistry

(1) CLEANING TEETH. Explain that this should always start with the toothbrush (or toothstick, 31-3B,C) on the gums, moving it up over the lower teeth, and down over the upper ones, at least 10 times, for at least 2mins. This is not easy behind the lower front teeth, and needs much practice. After this, it is important to rinse out the mouth. If there is no toothpaste, you can use common salt, or nothing: brushing is more important than paste.
Don't discourage the use of sticks or sugar cane!

(2) SCALING TEETH
INDICATIONS
Use the spoon end of a scaler (31-3F) to remove deep hardened plaque (31-3J). This starts just below the gum. Removal may be difficult, because the plaque may stick so firmly to the teeth that scraping it off can cause mild pain and bleeding.

Use the point of the scaler to remove plaque from between the teeth. Rinse the mouth out thoroughly, and then demonstrate how to clean the teeth properly.

CAUTION! The most important part of treatment is demonstrating how to clean teeth effectively and regularly. Advise use of dental floss and avoidance of sticky sugary foods, including most fizzy drinks.

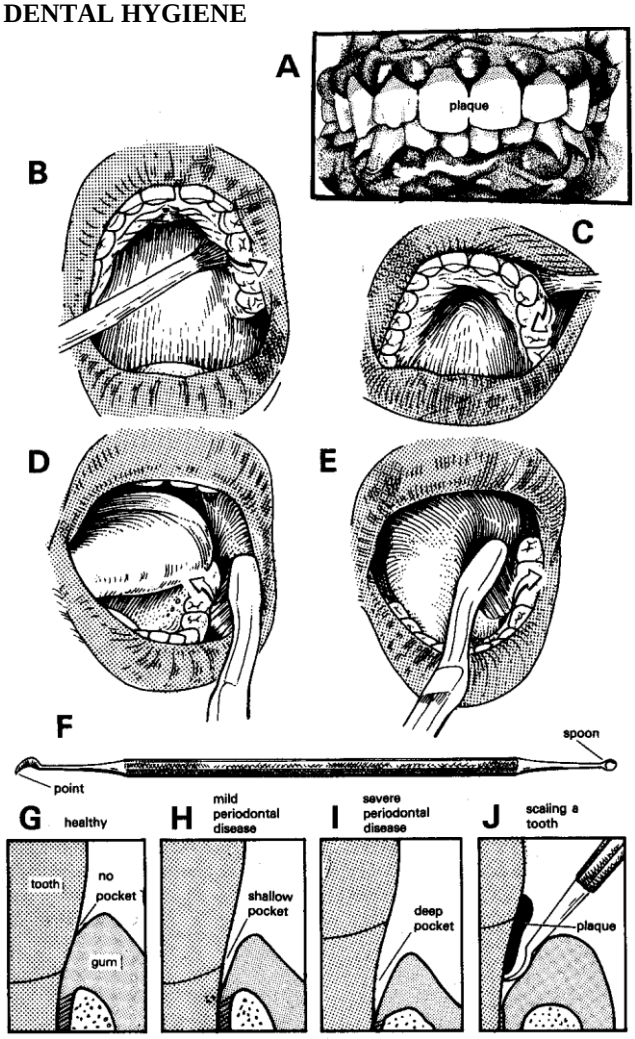


Fig. 31-3 PREVENTIVE DENTISTRY.
A, plaque around the lower front teeth. B-C, use of a toothstick on the upper teeth. D-E, use of a toothbrush on the lower teeth. Use the brush vertically from the gums up and over the teeth, and not horizontally along them. F, dental scaler. Sections of a tooth and gum: G, healthy gum. H, early periodontal disease. I, severe periodontal disease. J, remove plaque with a scaler.
After Halestrap DJ, Simple dental care for rural hospitals, Med Missionary Assoc, London, 4th ed 1975 p.198 7-11 with kind permission.

(b) Gingivitis
If this is severe, apply a strong topical antiseptic such as chromic acid 5% od, and treat with oral metronidazole. Measure the depth of the gum pockets with a special blunt probe which you can introduce under the gingiva alongside the tooth. If the pockets are <5mm, scaling may be all that is necessary, but if they are deeper than this, gingivectomy is necessary. If this is impractical, consider extraction.

(c) Caries
If you can save the tooth, scrape out the carious portion, and fill the cavity with fluoride-impregnated plastic filling: this is part of the WHO Atraumatic Restorative Technique.

31.3 Extracting teeth

You should be able to extract teeth, either for severe toothache due to irritation of the dental pulp or abscess formation or, less often, for periodontal disease. This makes teeth so loose that they often fall out on their own. Try to remove the tooth with all its roots, and without damaging anything else in the mouth.

The secret of success is to force the beaks of the forceps over the visible crown of the tooth, and under the gums, between the periodontal membrane and the alveolar bone, so as to grip its roots firmly. Then, while still grasping the tooth firmly, gently rock it or rotate it depending on the kind of tooth you are removing (31-6D,E). This will break down the periodontal membrane, and widen its socket. *The common idea of 'pulling teeth' is false*; the important movement is the early one of pushing the beaks of the forceps into the jaw around the root of the tooth.

Each forceps has handles, a hinge and a pair of blades. Forceps for the upper jaw are straight, or slightly curved; those for the lower jaw have blades at right angles to their handles.

Ideally, forceps should avoid the crown, and fit the whole surface of the neck and root of a tooth. The blades must be sharp, so that they can easily slide between a tooth and its gum. If necessary, sharpen them on the outside of their tips.

If a tooth has one root, you can loosen it by twisting it (31-6D). The teeth which have one root are: the upper incisors and canines, and the lower incisors, canines, and premolars. All other teeth have more than one root, so you cannot twist them. Instead, you have to rock them (31-6E). You will need two forceps for upper molars: one for the right and another for the left. Upper molar forceps are curved, so as to avoid the lower lip. The buccal blade with a beak on it is designed to grip the two outer roots, and the palatal blade is designed to grip the one inner root. One pair of lower molar forceps is enough. You can, however, remove *any* tooth with lower premolar forceps (31-4).

EQUIPMENT needed
FORCEPS, dental, set of six: (a) upper anteriors. (b) upper right molars. (c) upper left molars. (d) upper premolars and roots. (e) lower molars. (f) lower anteriors and roots.
Alternatively, FORCEPS, dental, universal, set of 2, upper universal, and lower universal. Dental forceps are expensive, so you may have to manage with these 2 universal forceps, but they are not so easy to use.
ELEVATORS, dental, (a) upper jaw, straight inclined plane, Coupland, (b)&(c) lower jaw, Cryer's set of 2. Coupland's elevator is a small gouge on a metal handle (31-8A,B). You will need it to remove roots; if you don't have one, you may be able to use the narrow blades of anterior forceps.
TWEEZERS.
PROP, dental. You may find this useful to keep the mouth open while you extract teeth.
DENTAL DRILL. If you are fortunate to have this, acquaint yourself with its proper use. You can attach ordinary IV tubing to it with small elastic bands in order to supply continuous water irrigation to cool the tooth and the drill bit: this is much cheaper than expensive pumps.

(a) Indications
(1) A painful, severely carious tooth.
(2) A periapical abscess (31-2X).
(3) A periodontal abscess.
(4) Severe periodontal disease.

TYPES OF TEETH AND FORCEPS

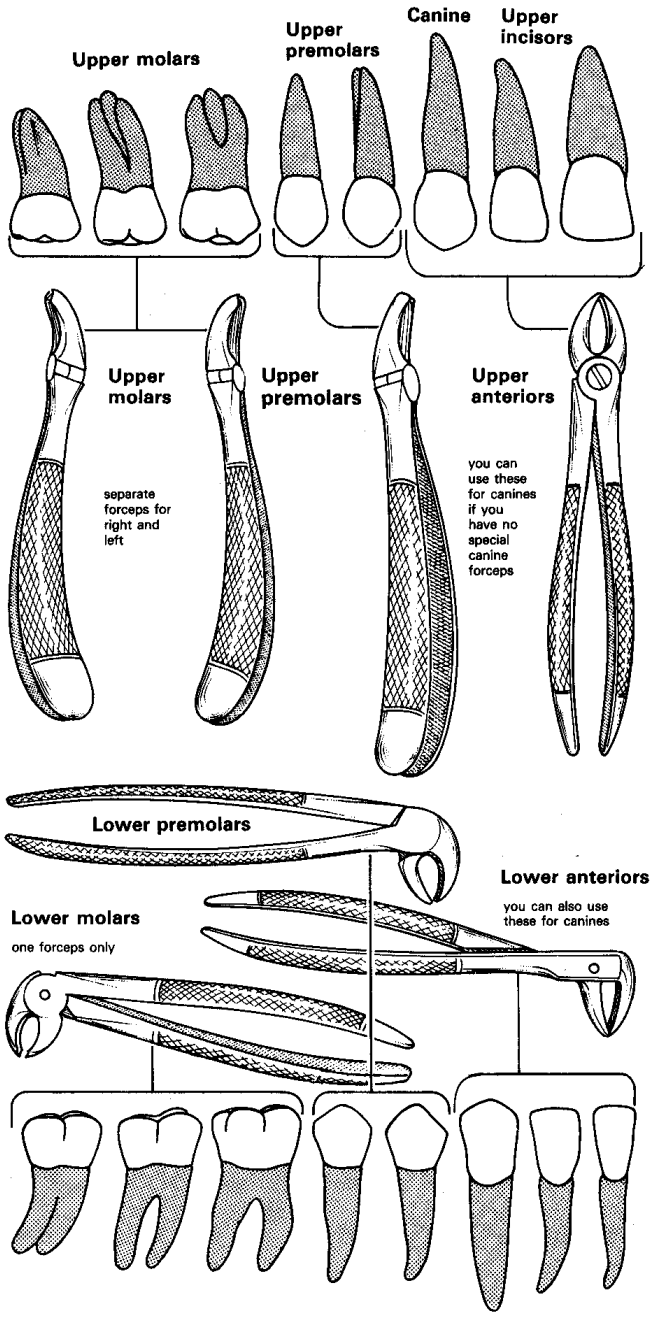


Fig. 31-4 TEETH AND FORCEPS.
Forceps for the upper teeth are straight, and those for the lower teeth are cranked. There are separate forceps for the upper molars, right and left; all other forceps can be used on either side. The beak of a pair of molar forceps is always on the outside, where it fits between the two buccal roots.
Kindly contributed by Hanif Butt.

(b) Relative contraindications
These may give you problems:
(1) Buried, impacted (31.4), or displaced teeth.
(2) Teeth which have no visible crown.
(3) Teeth in very dense bone.
(4) An uncorrected bleeding disorder.

If there is severe periodontal disease and the teeth are firm, but have swollen gums round them, leave them until other remedies have failed; you may still be able to save them. But if one or more of the teeth are loose in their sockets, and the gums are red and swollen, and bleed easily on light pressure, remove such teeth.

If a small hole in the tooth seems to be responsible for the pain, clean out the cavity. Use a mixing spatula on a glass surface to make a paste of zinc oxide powder and oil of cloves. Dry the hole with cotton wool. Pack the mixture into the cavity with a plastic hand instrument (31-1D). You can easily remove this paste later.

(c) Children's teeth
Under 12yrs, *be very careful when you remove a deciduous (primary, or 'milk') tooth*, lest you remove or damage the permanent tooth underneath.

(d) Medical history. *Don't forget this!*
Use antibiotic prophylaxis if the patient has a prosthetic heart valve.

(e) Which tooth?
If there is toothache, it is usually clear which tooth is responsible. Occasionally, however, when pain is referred, it is not clear even which jaw is affected. So, *don't necessarily remove the tooth which appears to be at fault*.
The offending tooth may:
(1) have a large hole in it. (If you cannot immediately see any carious areas, use a dental mirror to look on the adjacent surfaces of the teeth.)
(2) be broken, black, or brown.
(3) look grey under its enamel.
(4) be loose with severe periodontal disease around it.
(5) be tender on gentle tapping.
Tap each tooth in turn with the handle of a dental mirror. The most sensitive one is likely to be the cause of the toothache.
N.B. Toothache may come from an infected maxillary sinus, or the temporo-mandibular joint!

(f) Radiographs
If a tooth is displaced or impacted (31.4), get a radiograph.

(g) Antibiotics. If there is an apical abscess (6.9), use cloxacillin for 24h beforehand, and continue for 3 days afterwards.

(h) Anaesthesia. Use LA. Make sure that a tooth is properly anaesthetized, by pushing a blunt probe into the gingival crevice (sulcus) on its outer (buccal, facial, labial) and inner (palatal, lingual) surfaces. If there is pressure but not pain, anaesthesia is adequate; otherwise inject more LA.

(i) Position. Keep the patient seated so that his head is level with your chest as you stand. Position him and yourself correctly (31-5B, 31-6B).

(j) Extraction of a lower tooth (GRADE 1.2)
Use right-angled forceps and press downwards.

If you are extracting a lower front tooth or a lower left molar or premolar, sit the patient upright in the chair, and low enough for the mouth to be level with your elbow. If he is too tall, stand on something. Grip the tooth socket

between the index and middle fingers of your left hand, and put your thumb under the mandible (31-5A).

If you are extracting a lower right premolar or molar tooth, stand behind the patient (31-5B).

If you are left-handed, stand behind the patient whilst extracting all lower left premolar and molar teeth. For all others, stand in front, but on the left side.

EXTRACTING LOWER JAW TEETH

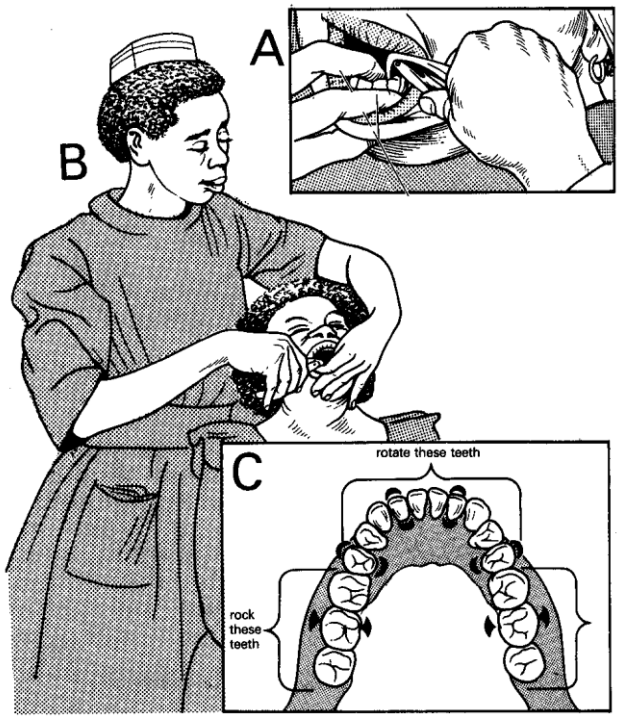


Fig. 31-5 EXTRACTING A LOWER RIGHT TOOTH.
A, support the jaw with one finger inside and one outside. B, stand behind the patient. If you cannot get high enough, stand on a step or something stable. C, rotate the incisors and rock the molars. *After Common oral diseases, WHO, Fig. 2.9.3. with kind permission of Martin Hobdell.*

The beaks of lower molar forceps are both pointed to fit between the 2 flattened roots. Use small rotating movements combined with a sideways movement between the tongue and the cheek. Use constant downward pressure, and support the jaw in your other hand.

To extract a lower incisor tooth, stand in front, push the beaks of the forceps down round the root, and apply a gentle backwards and forwards movement.

To extract a lower canine, which has a more rounded root, use gentle rotating movements (31-6D).

To extract lower left premolars, turn the head towards you and use gentle rotating movements.

To extract lower right premolars, move to the right, or even stand behind the right shoulder.

To extract a lower right molar, stand behind the right shoulder, and use a side-to-side rocking action (31-6E).

If you have difficulty extracting the lower 3rd molar, this may be because its roots are deformed, and need to be dissected out with bone chisels (31.4).

(k) Extraction of an upper tooth (GRADE 1.2)
Tilt the head backwards. If your chair does not have a head rest, support the patient's head against a wall, or ask your assistant to support it. Stand upright and to the right in front of the patient. For all upper teeth, put the finger and thumb of your left hand on either side of the gums.

CAUTION!

(1) Make sure that the long axis of the tooth forceps blades is in the long axis of the tooth.
(2) *Don't grasp the tooth and the gum together*.
(3) Carious teeth are brittle and will break if you put too much sideways pressure on them: *don't use the forceps as a 'nut cracker'*.
(4) *Don't start extracting movements when you have only grasped the crown of the tooth*.
(5) When you rock a tooth, feel if it is responding to reasonable pressure; if it does not respond and seems very firmly fixed, abandon the attempt.

To extract an upper incisor, or canine, which have a single conical root, rotate the tooth at the same time as you press it firmly in the direction of its apex (31-6D). Finally, tilt it outwards.

To extract an upper premolar, which has delicate roots (the 1st premolar often has 2), be as gentle as you can. Make small side-to-side and rotating movements while you push upwards with considerable force. When the tooth is loose, pull it downwards.

To extract an upper molar, which has 3 roots, 2 on the outer side, and a single large one on the inside next to the palate (the roots of the 3rd molar are sometimes fused together), choose the correct molar forceps (right or left), so that the pointed blade slips down outside the crown between the roots on the outer side.
Press upwards firmly until the beaks are beside the roots, while you make slight side-to-side rocking movements to loosen it (31-6E).
Finally, increase these movements, and exert pressure in an outward direction, until you can draw the tooth out of its socket into the cheek.

CAUTION! Make sure you support the socket firmly between your finger and thumb, because you can easily break off part of it, especially when you extract a 3rd molar, and break the maxillary tuberosity posteriorly.

EXTRACTING UPPER JAW TEETH

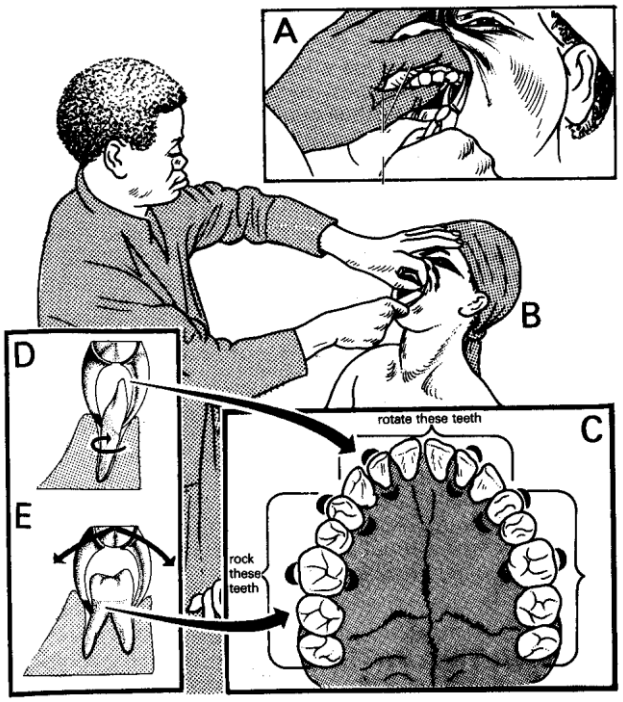


Fig. 31-6 EXTRACTING TEETH FROM THE UPPER JAW.
A, support the gums between the finger and thumb of your left hand. B, tilt the head backwards and support it against a wall, or ask an assistant to support it. Note the excellent position of the operator in this figure. C, the teeth to rotate (those with single roots), and the teeth to rock (those with >1 root). D, rotate a tooth with a single root. E, rock a tooth with >1 root.
After Common oral diseases, WHO, Fig. 2.9.2, with the kind permission of Martin Hobdell.

(l) Post-operatively after tooth extraction
Rinse out the mouth *once only*. Remove loose bits of bone and tissue. Push the inner and outer sides of the empty socket together. Place a tight ball of gauze over the socket, and tell the patient to bite on this for 15mins; make sure he presses on the gauze. Ask him not to spit or wash out the mouth again for 24h; it may wash away the clot, which should be filling the empty socket. The following morning, start rinsing out the mouth with water, using a small spoonful of salt to a cup of water. Tell him not to touch the socket, or play with it with his tongue.

CAUTION!
If you have extracted a tooth for an abscess, commence antibiotic treatment if the swelling does not rapidly improve. Examine the tips of the roots you have removed to make sure they are complete.

N.B. If the empty socket does not bleed after you have removed the tooth, use a dental probe to scratch around inside it until it does bleed. A socket which does not bleed is more likely to become infected ('dry socket').

(m) Difficulties during tooth extraction
If there is a constant oozing during the operation, swab, suck, and apply packs. If necessary, press a dry pack over the wound for 2mins *timed by the clock*.

If the tooth is immovable, and fails to yield when you apply reasonable force with forceps or an elevator (31-8), it probably needs dissection.

If the enamel crown or root breaks, examine it carefully to see how much you have left behind. What you should do depends on how much is left. If it is only a root apex, <5mm in its greatest dimension, leave it. In a healthy patient, the retained apex of a living tooth is unlikely to cause trouble. If the root is >5mm, try to extract the fragment of the broken root with a Coupland's inclined plane elevator (31-8B). Wiggle the elevator between the root and its socket. Otherwise, wedge a blunted 26G needle or wire, braided if available, firmly into the exposed root canal of the tooth fragment, and pull on this to extract it. Alternatively use a large burr to grind out the socket (broken root included) so that no tooth fragment remains.

CAUTION!
(1) *Hold the elevator with your index finger near its tip to stop it slipping.*
(2) *The upper premolar and molar roots are very close to the maxillary sinus, so you can easily push a root into it.*

If the socket breaks as you remove the tooth, examine the bone. Remove any bony fragment which has lost >½ its periosteal attachment. Grip it with haemostatic forceps, and dissect off the soft tissues.

If you displace a tooth into the sinus or if, while you are extracting an upper molar, you feel supporting bone move with the tooth, the MAXILLARY TUBEROSITY IS FRACTURED, and you have, technically, opened the sinus. If only a small piece of tuberosity has broken off, remove it. If a larger piece has broken, this will need a mucoperiosteal flap made to cover the gap. Warn that if the same teeth are extracted on the other side, the same thing may happen again.

If, when you remove the upper molar, you suspect you have produced a fistula, ask the patient to grip his nose and to try to blow air through it. This will raise the pressure in the maxillary sinus, make blood in the socket bubble, and deflect a wisp of gauze you hold over the socket. The fistula needs to be closed with a flap. *Don't allow rinsing of the mouth until you can arrange repair of the defect*, and *don't put any instrument through the fistula*: you may infect the sinus.

If you have produced a fistula, and this presents within 24h, close it immediately, by incising the periosteum and advancing a buccal mucoperiosteal flap over the defect (31-8E), and suture it in place. Post-operatively, use cloxacillin and inhalations of tincture of benzoin.

If a fistula presents after 24h, the edges of the wound will probably be infected, so *don't suture it*. Advise oral intake of a sloppy diet. Allow the area to heal, excise the fistulous tract, and close the fistula with a buccal flap. You can remove most teeth or roots from the sinus through the original defect enlarged if necessary.

If you lose a tooth while you are extracting it, immediately bring the head forwards, and ask the patient to cough it out. If you don't find it, X-ray the socket *and* the chest.

If it has been inhaled, try to remove it by bronchoscopy (29.14) as soon as possible, before a lung abscess develops.

If you break or dislocate the mandible, you will need to fix it internally and reduce it.

If you injure the tongue, and the wound is small, it needs no treatment except mouth washes. If it is larger, pull it forwards, inject some lidocaine with adrenaline and repair it with absorbable sutures.

If there is an extra tooth, it is usually conical, and may present almost anywhere on the jaw, and even in a nostril. Removing it may call for skill and ingenuity. If necessary, use a dental elevator to clear away the soft tissues of the gum before you apply forceps.

HAEMOSTASIS FOR A BLEEDING SOCKET

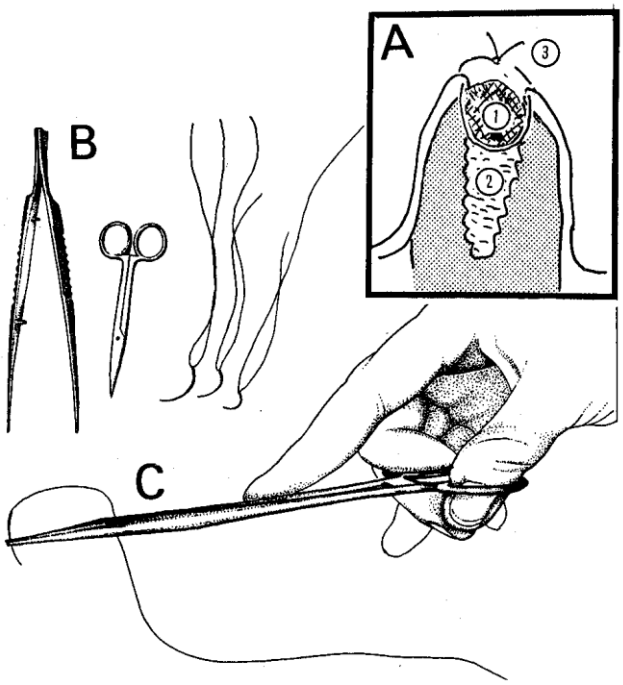


Fig. 31-7 SUTURING A BLEEDING SOCKET. A, ball of haemostatic gauze (1), soaked in adrenaline and plugging a clot-filled socket (2), which is closed by sutures (3). B,C, equipment needed; hold the needle at an angle.
B, from Common oral diseases, WHO, with the kind permission of Martin Hobdell.

(n) Bleeding after tooth extraction
Bleeding during the first few hours is likely to be reactionary haemorrhage. Later bleeding is the result of infection (secondary haemorrhage, 3.5).

If there is continued haemorrhage, tell the patient to bite on a rolled gauze, soaked with a 1mg ampoule of adrenaline, in the socket for a further 30mins. Make sure the pad really does press onto the socket this time. (If the socket is infected, use hydrogen peroxide.)

If there is persistent haemorrhage, suture the gums. Use a half-circle cutting needle and 3/0 black waxed non-absorbable suture. Pass 3 such sutures through the gum one side of the tooth socket, and out on the other side. Place a plug of haemostatic gauze over the bleeding socket (31-7A), and tie this in position. If you don't have haemostatic gauze, use cotton wool; *but be sure to remove the pack after 48h.* Alternatively, bring the edges of the gum together by chipping away bone from the crests of the socket. This will put the gum under tension, and make it less likely to bleed. *Be careful not to suture foreign material under the gingiva!*

CAUTION! Don't be content with inadequate suturing; it will only cause more problems later.

(o) Infection after tooth extraction
Diagnose infection when there is:
(1) **Pain and bleeding.** Irrigate the socket, remove clot and food debris, pack it with haemostatic gauze, and suture this in place. Place a firm gauze pack on top and ask the patient to bite on this. Use metronidazole. *Don't allow rinsing of the mouth*, which may restart the bleeding; instead, clean it with wet gauze.
(2) **An acutely painful empty socket, without any clot in it.** This is a DRY SOCKET. It is a local osteitis of condensed bone. The danger is that osteomyelitis may follow. Irrigate it with warm water and remove any food and degenerating blood clot. Under LA, scratch around inside the dry socket to make it bleed. Try to excise any sharp bone spurs. If it bleeds and a clot forms, it will probably heal. A dry socket is very painful, so make sure you provide adequate analgesia.
(3) **Fever and a very painful socket, a mandible which is exquisitely tender, and perhaps numbness of the lips** (owing to involvement of the mental nerve). This is acute OSTEOMYELITIS (7.15).

(p) Broken roots after tooth extraction
If a root breaks off, leave a small piece (<⅓ of a root) in place. Remove a larger piece. Try to do this with the narrow blades of a pair of anterior forceps, or by passing Coupland's inclined plane elevator between the root and its socket (31-8A). Push the elevator towards the bottom of the socket, while you press it firmly and rotate it a little each way. As you do so, hold it with your thumb near its tip to prevent it doing any unnecessary damage (31-8B). It should act like a wedge and move the root out of the socket. You can also use this elevator for loosening very firm teeth.

If you fail to remove a root, use a large surgical pneumatic burr to drill vertically into the root, thus destroying it, but not drilling surrounding bone. This avoids the risk of thermal osteonecrosis, and requires no flaps. The mucosa will grow over the defect. Furthermore the patient can then have an implant inserted.

CAUTION! Don't try to remove a fractured maxillary root by passing instruments up the socket. You may enter the sinus and produce a fistula (31-8D). This is much more likely to occur with molars and premolars than with incisors and canines.

DIFFICULTIES WITH ROOTS

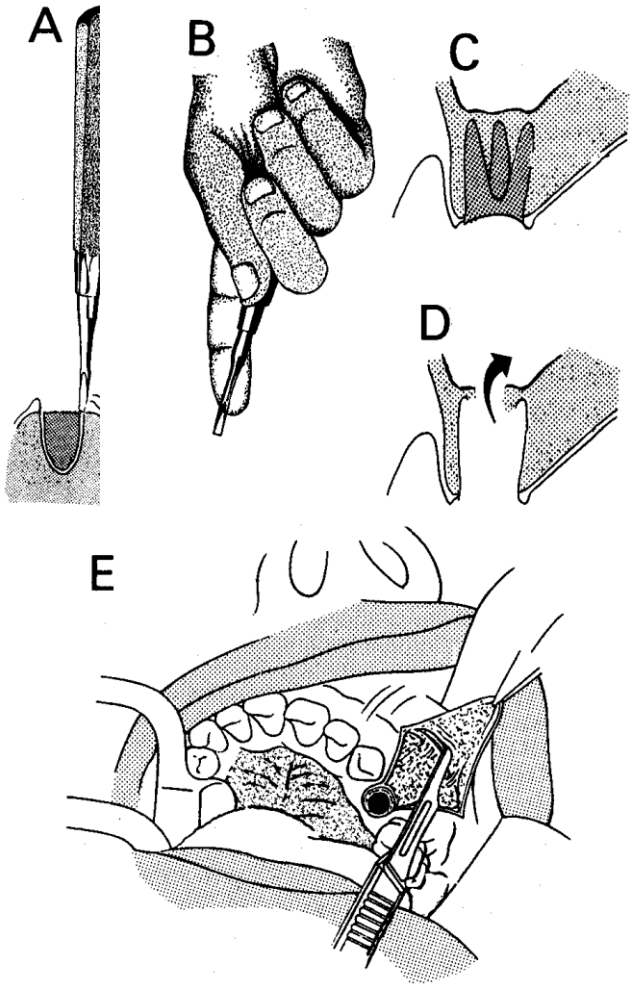


Fig. 31-8 DIFFICULTIES WITH ROOTS.
A, Coupland's inclined plane dental elevator. B, how to hold this with your finger close to the end, to act as a guard. C, roots of a patient's upper teeth are close to the maxillary sinus. D, be careful *not to cause a fistula into the maxillary sinus*. E, make a relieving incision through the periosteum (only) on the undersurface of a mucoperiosteal flap, and move this across to close an oro-antral fistula.
A-D, kindly contributed by DJ Halestrap. E, after Dudley HAF (ed) Hamilton Bailey's Emergency Surgery, Wright, 11th ed, 1977. p.182 Fig. 16.45 with kind permission.

(q) Other difficulties with carious teeth
If there is a small discharging granuloma with underlying induration, on the lower face, jaw, or chin, or inside the mouth on the surfaces of a dental socket, it is probably a DENTAL SINUS. An abscess around an infected residual root has caused osteomyelitis in the bone under it, and pus has tracked through the soft tissues to discharge on the gums or on the surface of the face.

Radiographs show a carious tooth, or a residual root, opposite the sinus. Using GA or ketamine, remove the root with a dental elevator and forceps. Curette away the granulation tissue on the face. Advise oral intake of a sloppy diet. The discharge should stop in 48h, and the granuloma should not recur. Treat with cloxacillin for 6wks.

DENTAL SINUSES

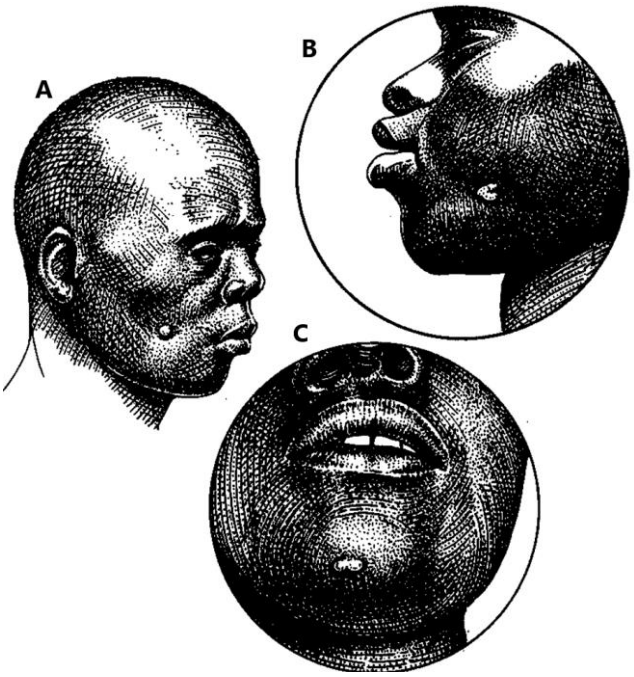


Fig. 31-9 THREE DENTAL SINUSES.
These are caused by a chronically infected residual dental root which has caused an abscess in the bone around it. This erupts A, on the gums, or, B, less often, on the surface of the cheek, or C, the chin.
After Bowesman C. Surgery and Clinical Pathology in the Tropics, Livingstone 1960, permission requested.

31.4 Impacted 3rd molar ('wisdom' tooth)

A lower 3rd molar sometimes fails to erupt because it faces forwards, or lies horizontally impacted against the second molar. A pocket or flap of gum (operculum) may overhang it, so that food is trapped and inflammation results. The patient, usually a young adult, complains of pain, which may be referred to the ear, and sometimes has trismus (lockjaw). Secondary infection may follow.

Gently syringe the space between the crown of the patient's unerupted tooth, and the flap of gum over it, with warm water. Then insert a pledget of cotton wool soaked in oil of cloves under the flap. Use metronidazole for infection, and ask him to use hot antiseptic mouth washes. The infection may settle down.

If infection does not settle, you may be able to incise the gum round the edge of the apex of the tooth, so that food no longer impacts around it.
If a 3rd molar is pressing on the gum flap, and making the condition worse, control infection and trismus with mouth washes, syringing, and antibiotics. If this fails, introduce an inferior alveolar and lingual nerve block.
If the 2nd molar is carious, remove it to leave space for the 3rd.
If the 2nd molar is normal, and the impacted 3rd molar is at a nearly normal angle, use bone forceps or dental forceps to nibble away the jaw behind it.
If the 3rd molar is completely horizontal, split it with a chisel, and then extract it in 2 parts, with any convenient forceps.

AN IMPACTED 3RD MOLAR

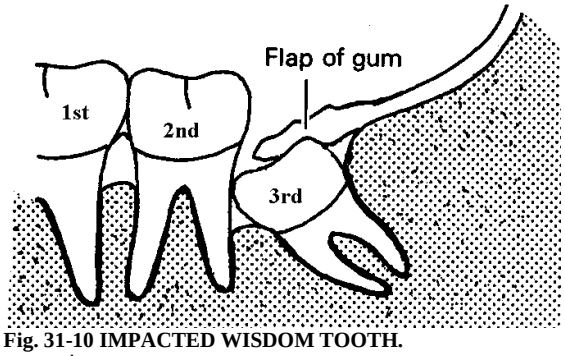


Fig. 31-10 IMPACTED WISDOM TOOTH.
This 3rd molar is lying obliquely in the jaw and is covered by a flap of gum. Food may be trapped and inflammation results.
Kindly contributed by James Gardiner.

31.5 Cancrum oris
(Gangrenous stomatitis, Noma)

Cancrum oris is not uncommon. It is a gangrenous process of the mouth which starts suddenly, rapidly involves the adjacent tissues of the face, quickly becomes well demarcated, and then spreads no further. It most often affects one or both sides of the jaw, and occasionally the front of the face (mouth, lips, nose, and chin). Mixed organisms including *Fusiformis* and *Borrelia* are mostly responsible, but it is not contagious. It is a necrotizing fasciitis, and may be associated with simultaneous extra-oral gangrenous lesions of the limbs, perineum, neck, chest, scalp, or ear, etc, especially in the presence of HIV disease.

Although cancrum oris can occur at any age, it is most common in poor malnourished children from 1-5yrs whose general health has been further weakened by some infectious disease or depressed immunity. It was endemic in Nazi concentration camps during World War II.

The lesion starts inside the mouth, in association with acute ulcerative gingivitis, and then spreads to the lips and cheeks. The earliest stage, which you rarely see, is a painful red or purplish-red spot, or indurated papule, on the alveolar margin, most often in the premolar or molar region. This lesion rapidly forms an ulcer, which exposes the underlying alveolar bone. You can stage the extent of the disease by how much of the face is affected – grade 1(<¼), 2(¼-½), 3(½-¾), 4(>¾) – and describe the part affected: N(nose), O(outer cheek), I(inner cheek), T(trismus), U(upper lip), L(lower lip).

At this stage, there is a sore mouth, a swollen, tender, painful lip or cheek, profuse salivation, and an extremely foul smell, with purulent discharge from the mouth or nose. Within the next 2-3 days, a bluish-black area of discoloration appears externally on the lips or cheek. The gangrenous area is cone-shaped, so that much more tissue is destroyed inside the mouth than the external wound might indicate. After separation of the slough, the exposed bone and teeth rapidly sequestrate.

Quite extensive superficial lesions can heal surprisingly well. But destruction of the deeper tissues, teeth and skeleton can produce such appalling disfigurement that expert plastic reconstruction will be necessary. This may have to include: correction of gross mutilation, 'dental anarchy', trismus (particularly difficult) and a salivary leak. You can however treat the acute stage. Untreated cancrum oris is almost always quickly fatal, owing to associated illness (*e.g.* measles, typhoid, diarrhoea, pneumonia) or a complication, such as septicaemia or aspiration pneumonia and malnutrition. Secondary haemorrhage is most unusual.

CANCRUM ORIS

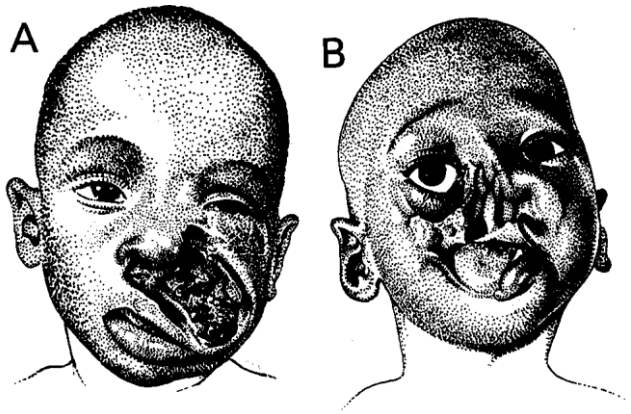


Fig. 31-11 CANCRUM ORIS.
A, cancrum oris in the acute stage, showing well-demarcated gangrene of the upper lip and adjacent cheek. B, typical example of the gross facial mutilation that follows.
After Tempest MN, Cancrum Oris, Tropical Doctor 1971;1(4):164-9 with kind permission of the editor.

Early treatment of cancrum oris
Start emergency treatment *immediately*, and aim to build up the child's immunity. Correct protein energy malnutrition and electrolyte losses, by normal feeding if possible. If the mouth is too sore, start nasogastric feeding. Correct the anaemia using folic acid, iron, vitamin C and B complex, particularly B3 and niacin, and blood transfusion if necessary. Treat with penicillin in large doses and metronidazole. Repeatedly irrigate the lesion. Debride septic surfaces; use hydrogen peroxide. Pack cavities with gauze pads soaked in hypochlorite ('Eusol'), saline, or BIPP. Change these dressings often, and keep them moist by adding more solution to the outer layers. Chewing raw pineapple, or slices of orange, will help to clean the mouth. *Avoid petroleum jelly gauze* (which acts like a foreign body), *especially when it has been impregnated with antibiotics.*

In a fit child, cut away any separating dead tissue, and remove any loose teeth or sequestra (dead bone). When quite large sequestra are ready to separate, you may be able to remove them under ketamine.

In an unfit child, allow the dead tissues to separate spontaneously. Sequestra occasionally drop out. More often, they have to be removed after 3-4wks, when the child's condition has improved enough for surgery to be safe. Check for intestinal worms and schistosomiasis.

CAUTION! There is no place for radical surgery at this stage, except to control bleeding (rare).

Try to organize reconstruction at 3-6 months, before marked trismus develops. This will allow the scars to mature, the local tissues to become supple and soft, and the child's health to improve. Meanwhile, maintain good nutrition and oral hygiene.

These children have major psychological difficulties of adjustment; do all you can to help them.

31.6 Jaw swellings

Lesions which make the jaws swell, apart from trauma, are:
(1) Infection: an alveolar abscess (6.9), a dental sinus, actinomycosis and osteomyelitis (7.14).
(2) Various types of dental cyst.
(3) Tumours: Burkitt's lymphoma (17.6), ameloblastoma, carcinoma, salivary tumours (17.7), and giant cell tumours (31-13D).
(4) A complex group of fibro-osseous lesions.

Actinomycosis classically follows oral surgery, or may complicate poor dental hygiene; infection arising from *Actinomyces israelii* spreads across tissue planes and results in a woody hard swelling around the mandible, resulting in sinuses discharging yellow 'sulphur-like' granules.

N.B. These are not sulphur at all, just yellow in colour; they stain blue under microscopy.

Osteomyelitis results, and infection may spread to the base of the skull, giving rise to cranial nerve lesions. There is no lymphadenopathy early on as the bacillus is too large to pass in the lymphatics; lymphadenopathy implies secondary infection.

You should drain pus, excise fistulae and remove necrotic tissue; however, the definitive treatment is till acute infection has settled and then oral penicillin for 2-6 months. In case of penicillin allergy, you can use erythromycin or doxycycline.

A dentigerous cyst forms round the apex of a chronically infected, and usually non-vital, tooth, commonly in an older patient. Chronic infection causes the epithelial remnants in the periodontal membrane to grow and become cystic. Dental cysts are usually quite small, and are commonly symptomless. Occasionally, they grow large enough to expand the alveolus in which they arise. In the maxilla they may extend into the sinus or the nasal fossae. It expands the lateral aspect of the jaw while the stronger medial side resists deformation. The tooth which forms the cyst usually fails to erupt, and you can see that it is missing from its normal place in the mouth. If this tooth is normally placed, opening the cyst may allow it to erupt. Often, it is so misplaced that it cannot erupt, and needs enucleation. The fluid it contains is usually clear, but may contain cholesterol crystals. The lesion may be hard, tense, or fluctuant. If the bone over a cyst is thin, it may crackle like an eggshell when you press it. Radiographs show a clearly defined, well-corticated, unilocular radiolucency, unless the cyst is infected, which causes it to lose its cortex.

An odontogenic keratocyst (rare, developmental) is filled with keratinized epithelial squames. These make the contents creamy, so that it looks like pus, and can only be distinguished from pus microscopically. *Don't confuse this cyst with an abscess*; there are no signs of infection. Radiographs show a well-corticated uni- or multi-loculated radiolucency. These cysts are particularly likely to recur after they have been removed (20-60%), so need radical surgery.

Developmental cysts (rare) are not associated with teeth. The commonest one is a nasopalatine cyst, which develops from epithelial remnants in the nasopalatine canal, immediately behind the upper front teeth. If it is causing problems, it should be enucleated. If this is impractical, you may have to open it out, taking care not to injure the incisor teeth and their supplying vessels.

Treatment of jawbone cysts
Simple methods are to:
(1) Marsupialize a cyst, by removing the mucosa over it, together with the immediately underlying bony wall and lining, washing it out, and then suturing the lining of its floor to the surrounding mucoperiosteum. This relieves tension, stops further expansion, allows drainage, and lets the space the cyst occupied slowly fill up from the bottom.
(2) Lay a cyst open.
(3) Decompress a dentigerous cyst, by opening it, and allowing the tooth in it to erupt.

Dentigerous cysts
(1) EXAMINATION
Stand exactly in front of the patient and inspect the face carefully for asymmetry, especially of the mouth, nostril, and the level of the inner canthi (corners of the eye). Feel the mass carefully. Most dental cysts which arise from an apical infection are small (<1cm), most dentigerous cysts are quite large (3-8cm). Examine and count the teeth.

If a tooth is missing (and has not fallen out), it may be hidden in a dentigerous cyst.

If one tooth in a line of permanent teeth is much smaller than the others, it might be a persistent milk tooth, with the missing permanent one hidden in a dentigerous cyst. Aspirate and examine the fluid from the swelling with a wide-bore needle. If you withdraw clear yellow fluid, it is a cyst. If you withdraw a substance that looks like pus, it is either true pus from an infection, or a mixture of keratinous squames from an odontogenic keratocyst. Microscopy will tell you which of these it is. Look for dental sinuses (31-9) on the gums or face.

(2) RADIOGRAPHS
Take films in 2 planes. Compare the density of the sinus shadows on either side. A cyst is an area of radiolucency surrounded by a radio-opaque line. If there is a tooth in the cyst it is dentigerous, otherwise it is probably dental.

MARSUPIALIZING A CYST

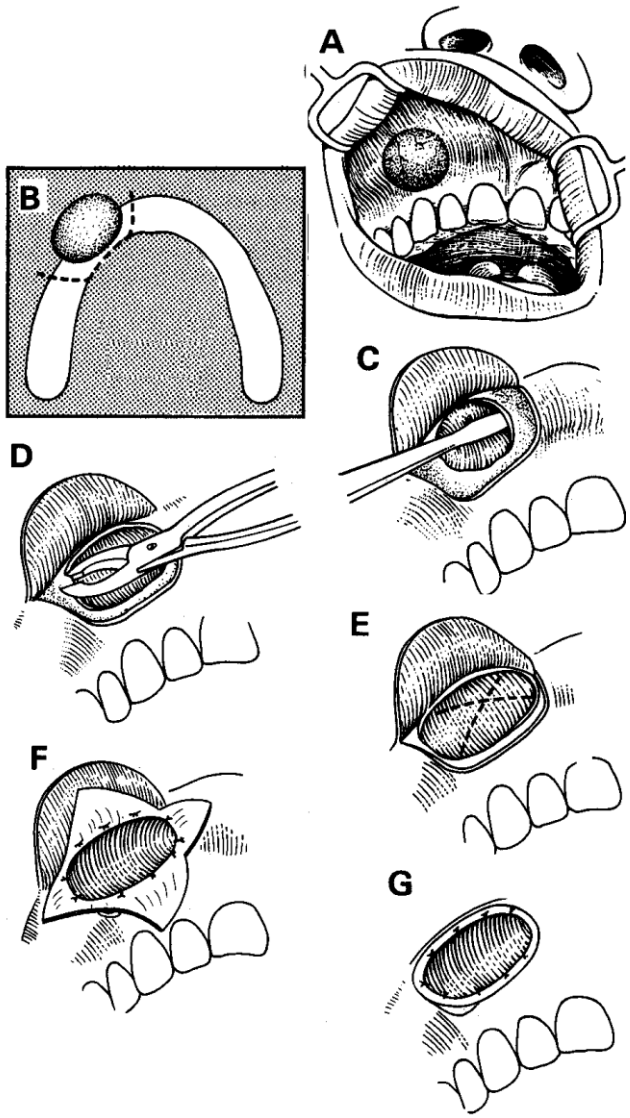


Fig. 31-12 MARSUPIALIZING A CYST.
A, a cyst on the gum. B, reflect the area of the mucoperiosteal flap. C, remove the bony roof of the cyst. D, trim the bone from the edges of the cyst. E, prepare to incise the lining of the cyst. F, open the cyst. G, marsupialization complete.
Adapted from Howe GL, Minor Oral Surgery, Wright 2nd ed 1971 p.194 with kind permission.

CAUTION!
(i) Be careful to distinguish a cyst in the maxilla from a normal part of the maxillary sinus: this can be difficult.
(ii) The signs that indicate that the lesion is not a simple dental cyst but a more aggressive lesion are:
(a) A multilocular ('honeycomb') radiolucency, indicating an ameloblastoma, an odontogenic keratocyst, or a giant cell tumour.
(b) A loss of cortex, indicating an aggressive lesion, particularly a carcinoma. If a benign cyst is infected, it may also lose its cortex.

(3) MANAGEMENT
If a dentigerous cyst is small and symptomless, leave it.

If it is small but is causing symptoms, remove the tooth and curette the cyst.

If a dentigerous cyst is large, and especially if it is in the upper jaw (unusual), remove the tooth. The danger is that you may produce a fistula between the mouth and the nose or the maxillary sinus. This usually needs surgical closure.

If the dentigerous cyst is <1·5cm diameter, marsupialize it, especially if it is in the lower jaw.

(4) MARSUPIALIZING A CYST (GRADE 2.4)
INDICATIONS
(i) An easier alternative to enucleation for a larger dental cyst.
(ii) A small dentigerous cyst.
(iii) An elderly patient, in whom there is a risk of pathological fracture.

ANAESTHESIA. Use a combination of LA and regional blocks. You may prefer to add ketamine. Thoroughly clean the mouth first.

INCISION
You can approach all cysts from inside the mouth, unless you need also to resect the jaw.

Approach the cyst from the side of the jaw on which the swelling is greatest. If it is equal on both sides, approach it from the buccal side. Reflect a large mucoperiosteal flap (31-12B). Remove bone over the same site (31-12D). Remove the superficial part of the lining (31-12E), so as to expose the cavity widely, and render the deeper part of its lining continuous with the oral mucosa. Wash out the cyst, and examine its lining for signs of neoplastic changes. If there is more than a little tissue in it, suspect that it might be an ameloblastoma. Send any material you remove for histology.

If you are marsupializing a dentigerous cyst, be sure to remove all the epithelium, or it may grow again. To do this, remove all the soft tissues on the outside of the bony cyst wall. Remove the tooth at the same time. Leave it open to granulate.

If a dentigerous cyst is related to a permanent tooth, the tooth is likely to be non-viable. It might be saved by root canal treatment, but you will probably have to remove it. If it is related to a deciduous tooth (unusual), remove the tooth.

If the bone is much expanded and the bony wall of the cyst is thin, consider compressing it to reduce its size.

Pack the cavity, and remove the pack at 48h or earlier. Continue with thorough mouthwashes until it has healed. Insist on washing out the mouth after meals.
CAUTION!
Be sure to make a wide opening. If it is too small, it will close, and the cyst will recur.

(5) LAYING OPEN A CYST
INDICATIONS
An infected or 'messy' cyst, with a lining which you cannot completely remove, or a flap which you have to sacrifice.

METHOD
Remove the tooth associated with a dental cyst, unless it can be root-treated. Open the cyst, remove as much of its lining as you can, and then pack it with BIPP-impregnated gauze. Reduce the bulk of this over 4wks, to allow the cavity to granulate slowly from its base.

(6) ENUCLEATING A CYST
More advanced methods include enucleating a cyst by reflecting a periosteal flap, opening it, removing all its lining, and then replacing the flap. This is more difficult than the preceding methods. GA with tracheal intubation is essential.

Approach a cyst in the upper jaw through the outer aspect of the socket. Approach a cyst in the lower jaw through an incision 1cm below the lower border of the mandible, or inside the mouth, through the labial side of the socket. Infiltrate the tissues with adrenaline in saline (3.1).

Clear the bony covering of the cyst, fracture its eggshell surface, and remove a piece of bone from its most prominent part. Nibble away more bone, and push the cyst off the bony wall of the cavity in which it lies. If it is a dentigerous cyst, its lining will be held round the tooth it contains.

BENIGN TUMOURS OF THE MOUTH

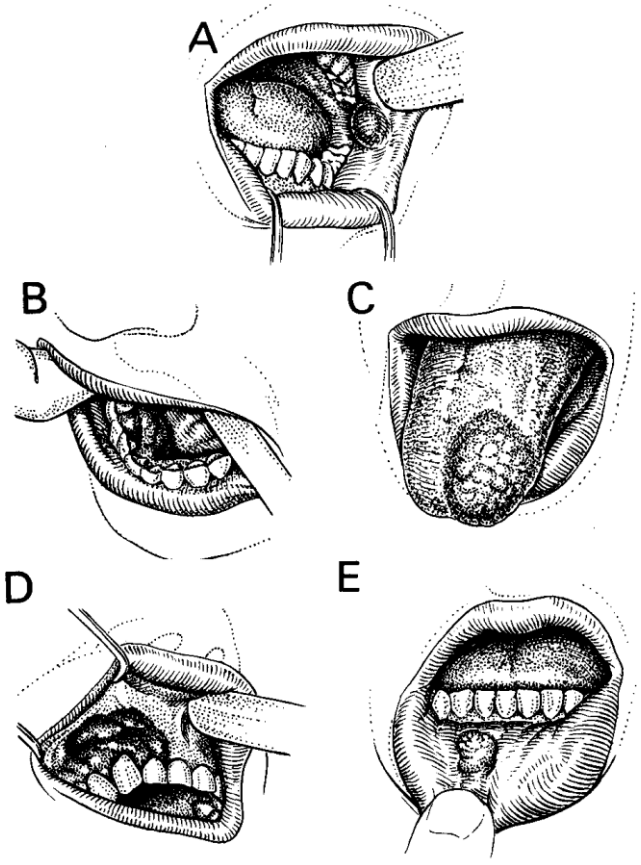


Fig. 31-13 BENIGN TUMOURS OF THE ORAL CAVITY.
A, a fibroma. B, a pregnancy epulis (a benign gingival growth, 31.9**). C, a haemangioma of the tongue. D, a giant cell tumour. E, a hard papilloma** (31.9)**.**
Adapted from drawings by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

You need an expert to excise a piece of jaw with a tumour. This may be:
(1) A **giant cell tumour** which is only locally invasive, but may grow very large if it is not treated (37-4).
(2) An **ameloblastoma** (adamantinoma), which arises inside the jaw from the enamel organ of a tooth, and slowly destroys the surrounding bone. It may be solid or cystic, it is locally invasive like a basal cell carcinoma, and does not metastasize.
You are unlikely to miss an ameloblastoma if you remember that: (a) the radiolucent lesions it produces are commonly multilocular (the cysts described above are mostly unilocular), (b) the solid tissue from around any 'cyst' should be sent for histology, which is the only certain way of making the diagnosis (the cysts described above are filled with liquid). An ameloblastoma requires radical removal.
(3) An **odontogenic keratocyst**, an **ossifying fibroma**, a **carcinoma**, or a **fibrosarcoma**.

31.7 Cleft lip and palate

(a) Cleft lip
This may be variable in extent, and is often associated with cleft of the tooth socket or palate. It is usually unilateral, but may be bilateral. Defects of the midline or oblique facial clefts are more complicated and of different embryology.

The Millard rotation advancement repair is the most popular; you should only attempt correction if the baby is in good nutritional state, and preferably >9 months old.

Make sure you have a fine marking pen and indelible ink or dye. *Don't attempt this operation if your experience is limited and your supply of fine sutures limited*: getting a good cosmetic result on a re-do is very difficult.

MILLARD CLEFT LIP REPAIR (GRADE 2.5)
Mark the points 2 to 4 on the child (31-14A), and infiltrate with lidocaine/adrenaline solution (3.1).

Incise through full thickness of the lip at points *4f* and *5g* so their thicknesses are equal, and along the dotted lines *4h* and *5e*, these also being equal. Make a curved extension *4a* and an equal curve laterally *dc* under the *ala nasae*.

Preserve a small 'C' flap so that *cd'e5* fits into *4'ab4*. This allows for closure of the nostril. Close the flaps with buried knots using 4/0 absorbable suture so that points *45* and *fg* align. Suture the skin with 5/0 nylon, and paint the wounds with chloramphenicol ointment qds. If you leave one length long, you can use this as a stay suture for easy retraction whilst you complete the remaining sutures.

Restrain the child from tampering with the wound by splinting the elbows at 45° flexion; make sure he is fed a sloppy diet with a spoon and uses mouthwash after eating. Remove the sutures after 4 days, preferably under ketamine.

If there is a bilateral cleft lip, repair the more severe side first, and then a month or two later do the other side. If the philtrum protrudes anteriorly, you can strap it back for a few months before surgery; protruding teeth will need to be removed because they will get in the way. (It is possible to repair a bilateral cleft lip in one sitting, but this is for the expert.)

MILLARD CLEFT LIP REPAIR

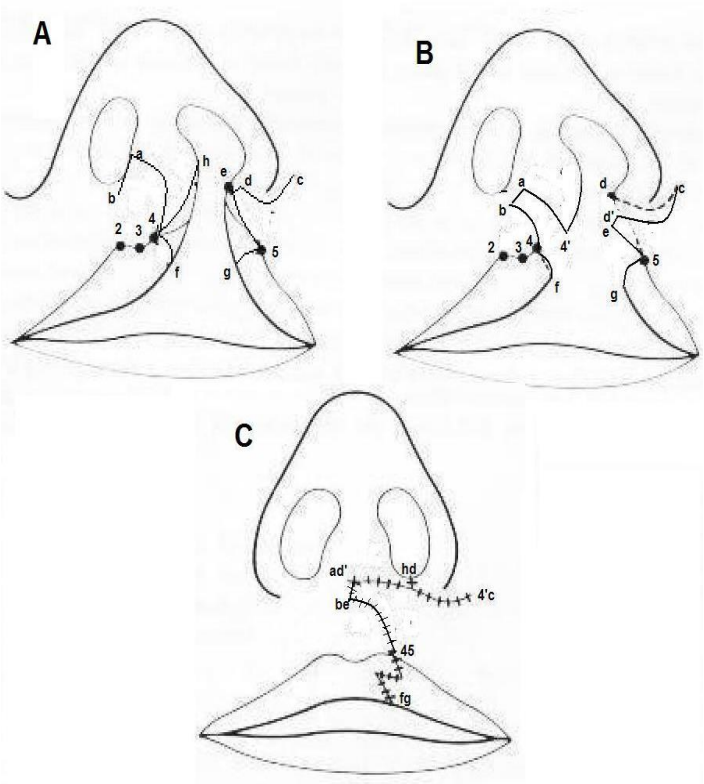


Fig. 31-14 MILLARD CLEFT LIP REPAIR.
A, *3* is the centre of the lip (Cupid's bow); *2* is the peak of Cupid's bow on the normal side; *4* is the projected point equivalent to the point *2* on the affected side, so that *23=34*; *5* is the start of the mucosal thickening of the cleft side, so that *5g=4f*; *h* is the medial extremity of the open nasal sill on the cleft side, and *e* the lateral extremity. The lip vermillion does not usually reach these points. B, intra-operative view: divide the upturned cleft lip across *4f* and *5g* such that these are equal. Cut *4h* so *4h=5e*; cut a curved extension *4a* where *a* is the medial border of the nasal sill on the unaffected side; this effectively removes the abnormal lip tissue *(and makes the defect look bigger and worse!)*. Cut a C flap to allow plastic nostril closure: cut *cd=a4*. Extend an incision along the nasal sill *ab=de* to make the C flap fit nicely. C, end-result: points *fg*, *45*, *be*, *ad'*, *hd*, and *4'c* are all joined together. The Z-plasty closing the upper lip is optional: *omit it if you are unfamiliar with it or you don't have very fine instruments.*
After Kirk RM, Williamson RCN. General Surgical Operations Churchill Livingstone 2nd ed 1987 p.563 Fig 31.26.

(b) Cleft palate

If milk comes from a baby's nose as he sucks, suspect that there is a cleft palate. This is often, but not always, associated with a cleft lip. It may be unilateral or bilateral. Cleft palates are much more difficult to repair than cleft lips. You should not operate before c.12-18 months, before the child tries to speak, and when he should be fit enough for a major operation, if he has been adequately fed. Breastfeeding is a major problem, except for minor clefts of the soft palate only, which need no treatment.

Try a cup and spoon. Alternatively make a feeding spout to fit a standard feeding bottle (31-15). If this fails, he will have to be fed through a nasogastric tube, until he is stronger, when cup and spoon feeding may be possible. A special plate, supported by 2 wire arms on the cheek, can be constructed in the first few days of life, to bridge the gap in the cleft palate.

SPATULATE FEEDING SPOUT

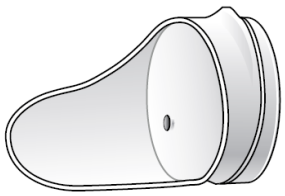


Fig. 31-15 SPATULATE FEEDING SPOUT.
Attach this to a feeding bottle and squeeze it to deliver a bolus into the spout.
After Jones PG, Woodward AA. Clinical Paediatric Surgery, Blackwell 3rd ed 1986 p.210 Fig 24.4.

Good dental care is essential. These children are prone to recurrent ear, nose and throat infections, and particularly get 'glue ears' (29.2,4).

Experts can do the surgery in theatres with basic facilities: if a programme for cleft repairs can be set up in your district, this is the best chance for your patients.

31.8 Oral tumours

Carcinoma of the mouth accounts for c.30% of malignant tumours in India. It is a disease of the poor, of both sexes, and is caused by:
(1) Chewing *paan* (betel leaf) with tobacco and slaked lime. *Paan* masala (spiced betel nut) chewing is popular in India, and results in oral submucous fibrosis: this is pre-malignant and ultimately results in cancer.
(2) Reverse or 'chutta' smoking.
(3) Smoking cheap 'bidis' (rolled tobacco leaves).
(4) Poor oral hygiene, especially associated with vitamin A deficiency, and low iron levels.

The patient, who is usually elderly, presents with:
(1) An area of dry, blanched leathery mucosa subsequently becoming thick or indurated,
(2) Inability to open the mouth (trismus), whistle and blow out the cheeks.
(3) A painless mass.
(4) An ulcer.
(5) A sore throat or dysphagia (late).

Because the disease is painless, poor patients are usually unaware of the danger and present late. Yet the mouth is easily accessible, so teach health workers to examine the mouths of their patients always.

Carcinoma can occur anywhere on the lips, or inside the mouth. It most commonly involves the buccal mucosa, but it may involve any part of the tongue, the floor of the mouth, the alveolus, or the hard palate. It commonly occurs in the gingivobuccal groove, where betel leaf is kept to chew later.

There are several kinds of tumour:
(1) Squamous cell carcinomas (90%). A few of these are slow-growing, cauliflower-like 'verrucous carcinomas' with a good prognosis.
(2) Adenocarcinomas arising from ectopic salivary glands (5%).
(3) Kaposi sarcoma, associated with HIV disease: these are typically purplish raised lesions.
(4) Other sarcomas (rare). Most tumours spread to the lymph nodes on the same side, and bloodstream spread is rare. The prognosis depends on the extent of the local disease, and whether or not there is cervical metastasis.
(5) Melanoma; most black-pigmented spots though are benign.

Precancerous lesions occur as:
(1) leucoplakia (white patches: dyskeratosis),
(2) erythroplakia (leucoplakia interspersed with reddish spots),
(3) submucous fibrosis,
(4) chronic dental ulcers.

Surgery and/or radiotherapy gives excellent results in early cases. In late cases radiotherapy reduces bleeding, discharge, and smell, and is useful palliation. If the buccal mucosa is involved, radiation is enough, but if bone is involved, resection of the jaw is also necessary.

MALIGNANT MOUTH TUMOURS

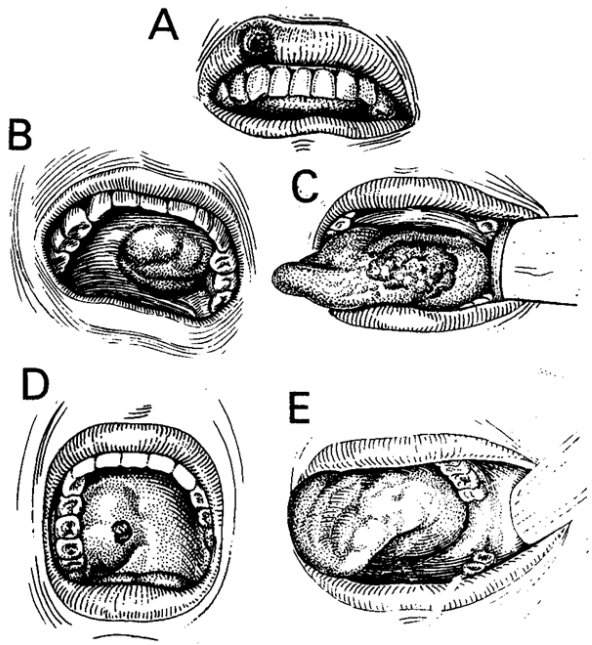


Fig. 31-16 MALIGNANT TUMOURS OF THE MOUTH.
A, early carcinoma of the lip. B, mixed tumour of the palate. C, carcinoma of the side of the tongue. D, carcinoma of the palate starting to ulcerate. E, leucoplakia of the tongue (this is precancerous).
Adapted from drawings by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

(a) Examination. Any ulcer or lump in the mouth which does not respond to treatment in 2wks, you should suspect as being malignant and biopsy it. Feel for enlarged nodes in the neck.

(b) Special tests. Confirm the diagnosis with a punch or incision biopsy. X-ray the mandible, or maxilla, to detect local infiltration, and the chest for distant spread.

(c) Management. You can only satisfactorily treat these patients in the earliest stages.

If there is a lesion <1cm on the lips or tongue, excise it with a margin of at least 1cm. All other patients need chemotherapy, followed by radiotherapy and/or surgery. Verrucous carcinoma is best treated by surgery only, because radiotherapy causes it to become a rapidly growing anaplastic lesion.

If there is cancer of the mouth, and you can feel nodes in the neck on presentation, consider your options carefully. Patients with a potentially curable lesion with mobile nodes and no distant metastases may have a 25% chance of cure with skilled surgery. If nodes appear months or years after the primary has been treated, *and are still mobile*, radical dissection has a 30-50% chance of cure. Node biopsy may be useful. The nodes may be enlarged by infection. If in doubt, try antibiotics for a few days, and see if they become smaller. Adequate surgical excision may mean using an extensive delto-pectoral or forehead rotation flap.
CHEMOTHERAPY is not curative, but there is a 20% response rate to x2wkly IV methotrexate at 40mg/m^2, with little further benefit from multi-dose regimes or more expensive drugs.

31.9 Other dental and oral problems

The range of possible oral pathology is large; some of the more important lesions are tumours.

(a) Mouth ulcers

If a patient has a recent, shallow, painful ulcer in the mouth, it is likely to be an APTHOUS ULCER, or a recurrent HERPETIC ULCER (both very common). The distinction between them is not important, since there is little you can do about either of them, and they will resolve spontaneously. Advise mouth washes, and try folic acid 5mg weekly, both as prevention and treatment. These ulcers are common in people taking the anti-malarial prophylaxis, proguanil.

If there is irregular ulceration of the gums, cheek or the floor of the mouth, suspect that this is a CARCINOMA (uncommon), especially if it has a raised edge. Send tissue for histology and arrange deep radiotherapy, or radical surgery.

Mucocutaneous *leishmaniasis* (34.7) is endemic in Bolivia, Brazil and Peru, and is transmitted by the sandfly from other infected humans, dogs or rodents. Itchy papules arise at the mucocutaneous junction of the lips and nose, and ulcerate. With spread by lymph and blood, tissue destruction may become extensive, requiring plastic reconstruction.

Paracoccidiomycosis *(blastomycosis)* is frequent in parts of Central and South America, particularly Brazil. It occurs between the ages of 20-40yrs. The fungus is inhaled from vegetables or the soil, and implanted through breaks in the skin or mucosa, resulting in haemorrhagic papules which soon ulcerate. Regional lymphadenopathy is common, and the intestines may also be involved. Use ketoconazole 200-600mg od if you can make a diagnosis early. The problem with the disease is gradual destruction of the mouth and nose, requiring plastic reconstruction as for cancrum oris (31.5).

(b) Lumps arising from the alveolus

If there is a firm lump on the gum, it is probably a FIBROUS EPULIS (common, especially in pregnancy). Very few of these lesions are fast-growing, and if a lump is soft, bluish, and grows rapidly, it may be a sarcoma (very unusual). Excise it and send it for histology. If it is very extensive, try to refer to an expert periodontologist (if you can find one!) or a maxillofacial surgeon. It may be one of a wide range of obscure, rare, fibro-osseous lesions.

If there is a soft swelling on the gum, between two teeth, or on the palate, and associated with chronic infection, it is probably a PYOGENIC GRANULOMA. It is related to HIV disease, when it is often very vascular, and may simulate a malignancy. Pyogenic granulomas are common inside the mouth, and can also occur on the tongue. *Don't try to excise this during pregnancy.* Otherwise, excise it using infiltration with adrenaline, and provided the infection is eradicated, it will not return. Send any material you obtain for histology. Make sure there is no underlying osteomyelitis (7.14).

If a child has a loose tooth with a swelling of the jaw, suspect that this is BURKITT'S LYMPHOMA (17.6) if it is common in your area. Typically, the teeth are displaced.

(c) Lumps arising elsewhere in the mouth

If there is a pedunculated swelling on the cheek (or tongue), it is probably a FIBRO-EPITHELIAL POLYP or a fibroma (31-13A). This is commonly associated with repetitive irritating trauma, particularly that from an ill-fitting denture. Excise it and it will not recur, provided the trauma is removed.

If there is a papilloma (wart) inside the mouth (31-13E), it may be viral (verruca vulgaris or condylomata associated with HIV disease), and there may be similar lesions on the hands and genitalia. If necessary, excise the oral lesion.

If there is an expanding tumour of the mandible, with a radiograph showing large loculi and a honeycomb appearance, suspect that this is an AMELOBLASTOMA (31.6).

(d) Cysts of the mouth

If there is a bluish, translucent, raised vesicle 0·2-2cm diameter, it is probably a MUCOUS RETENTION CYST (31-17C). These cysts may arise from the mucous glands anywhere inside the mouth, including the tongue, but are most common inside the lower lips. They may arise in a few days, persist for months, periodically discharge their contents, and then recur. Try to excise the lesion; if you merely incise it, it is likely to recur.

If a child has a circumscribed, fluctuant, often bluish swelling of the alveolar ridge, over the site of an erupting tooth, it is probably an eruption cyst. This is common, usually symptomless, and bursts spontaneously to allow the tooth underneath to erupt. If it does not, under ketamine, grasp it with toothed forceps, and excise it. A little dark blood will escape, and the underlying tooth will erupt within the next few months.

If there is a slowly enlarging painless unilateral swelling on the roof of the mouth with normal mucosa over it, it is probably a RANULA (31-17A). This is a particular form of retention cyst, arising from the inferior aspect of the tongue, and caused by blockage of the submandibular duct. If you remove it entirely by careful dissection, it will not recur. If this is difficult, deroof it; it may, but will probably not, recur.

It may also be a SUBLINGUAL DERMOID CYST (31-17B), which is a rare developmental cyst in the line of fusion of the 1st branchial arches. The epithelium lining it is thicker than that of a ranula. Although it arises in the midline, it usually displaces the tongue to one side. Dissect it out cleanly, and take care not to injure the submandibular duct.

CYSTS OF THE MOUTH

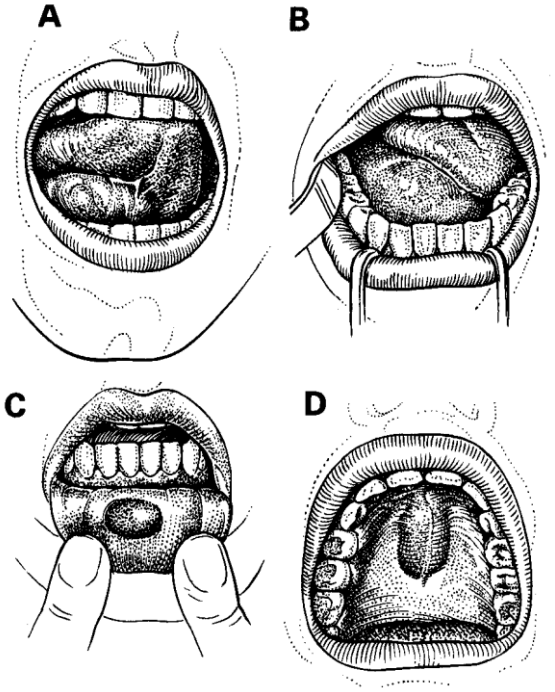


Fig. 31-17 CYSTS IN THE MOUTH.
A, ranula. B, sublingual dermoid cyst. C, mucocoele of the lip. D, secondarily infected nasopalatine cyst.
Adapted from drawings by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle, Switzerland.

If there is a midline swelling in the middle of the mouth, it may be a rare NASOPALATINE CYST (31-17D), which may have become secondarily infected. More likely, it is a pleomorphic adenoma (mixed salivary tumour) in an ectopic site.

(e) Tongue-tie (Ankyloglossia)

In neonates the lingual fraenum seems short and attached to the tip of the tongue; appearances change in the next 1-2yrs and the tongue is actually perfectly mobile protruding easily over the lower incisor teeth.

Poor speech is almost invariably due to *hearing deficiency* or *brain damage*. True tongue-tie is very rare and causes the tongue to indent deeply in the midline on protrusion, and requires a Z-plasty correction: this is a procedure that requires proper GA, mouth packing, good lighting and good equipment!

(f) The mouth in facial palsy

If a patient cannot close the mouth because the facial nerve is paralyzed, because of leprosy, a stroke, or parotid disease or surgery, the gums may dry and he may dribble food and drink and become socially outcast. His teeth will also be more susceptible to caries.

One solution is a *plantaris* or *fascia lata* tendon transfer to support the lip, by slinging it from the zygoma or temporalis fascia on both sides. Although this is a static sling, it will keep the mouth closed, improve its appearance, and stop the dribbling. If there is no lagophthalmos (lid-lag: 28.17), try to arrange a temporal muscle transfer to reactivate the mouth.

32 Orthopaedics

N.B. Trauma is dealt with in Volume 2.

32.1 Muscle and joint contractures

(a) A contracture is a deformity which prevents the movement of a joint through its normal range. Structurally, contractures are the result of shortening of the soft tissues of a limb, and/or tightening of the ligaments of a joint. This can happen as the result of:
(1) Ischaemia, which can occur in compartment syndromes due to neglected crush injury, burns, tourniquets, or snake bite,
(2) Soft tissue or bony injuries, especially burns and fractures,
(3) Neuropathies, including leprosy,
(4) Poliomyelitis, and other lesions affecting peripheral nerves (lower motoneurone), which weaken one muscle group more than another,
(5) Spastic paralysis of an upper motor neurone lesion, *e.g.* after a cerebrovascular accident (stroke), cerebral birth injury, encephalitis, or any head injury,
(6) Osteomyelitis (7.2),
(7) Arthritis (7.16),
(8) Soft tissue infections (6.22) and other unknown causes, *e.g.* Dupuytren's contracture (34.2).

(b) "Prevention is better than cure" is never more true than with contractures. If a joint is to remain useful, it must move regularly through its full range. Anything which prevents it from doing this eventually causes a contracture. The soft tissues surrounding a disused joint become shorter, and less elastic, and its muscles waste and will not extend normally. Ultimately, its bones change their shape, and become deformed; it lacks a full range of movement, or becomes fixed near one end of its range, usually flexion.

The two important principles in prevention are:

(1) Most importantly, to keep all joints moving whenever you possibly can. For example, a patient lying prone for several weeks may keep the elbows flexed and never move them. This will result in severe contractures in both elbows, which were perfectly normal on admission. A burnt child may develop contractures in joints unaffected by the burns simply because he did not move them. Contractures like these happen quite unnoticed, and when you do notice them, *it may be too late*.

(2) When movements are temporarily difficult, or inadequate for any reason, prevent deformity by splinting or skin traction, as with the burnt child.

PREVENTING CONTRACTURES IS EASIER THAN TREATING THEM

Most contractures can be prevented by:
(1) Putting the joints through their full range of active and passive movement, several times a day, as with paraplegia. This is such a simple measure, yet it is so often forgotten. You may not have physiotherapists, but this is something that all nurses can do; so, show them how.
(2) Appropriate splinting, as for burns, tuberculosis of the knee (32.3), or a radial nerve palsy (causing wrist drop).
(3) Skin traction for burns.
(4) Early movements in bone and joint injuries, as with Perkins extension traction using a Steinmann pin through the upper tibia.
(5) Early drainage of pus, as with septic arthritis of the hip, which readily causes a flexion contracture (7.18).
(6) Early grafting of wounds and burns over joints.
(7) Early manipulation and immobilization, as for neonatal club foot (32.10).

Practise several of these preventive measures at the same time: *e.g.* combine splinting with active and passive movements.

In polio, start to assess the power of the muscles (32-1) as soon as tenderness allows, usually about 3wks after the start of paralysis. Assess the degree of recovery regularly; you will then be able to judge how far full recovery is likely. The joints must be stretched in the direction opposite to that in which a contracture might form, preferably qds (*e.g.* stretch an *equinus* ankle contracture dorsally, 32.9). Fit a calliper (32-13) as soon as the tender muscles will allow. In the acute stage, leave this on for most of the day and the night. Or, use a plaster gutter splint. After 3 months from the onset of paralysis, you will know whether long-term callipers are necessary or not.

(c) Assessment

Where relevant, make the assessment lying, sitting, standing, and walking. Remember that abduction is movement away from the midline, and adduction is movement towards it. *Varus* is a deformity where the distal part is more medial than it should be, and *valgus* (32-11A) where the distal part is more lateral. In an *equinus* deformity of the ankle, the foot points downwards, like that of a horse; in a *calcaneus* deformity the foot points upwards so that the calcaneus bone is pointing downwards.

Contractures involving the tendons or nerves (as in the popliteal fossa) are more difficult. Involvement of a joint can be due to:
(a) Mild or dense adhesions.
(b) Shortening of capsule or ligaments.
(c) Destructive changes, as the result of past infection.
(d) Ankylosis (fixed joint due to fibrous or bony tissue growth).

If the joint is merely stiff, exercising it should not be too difficult. If only the skin, subcutaneous tissues, and muscles are involved in a contracture, you should be able to release them. If the bones are deformed, an osteotomy will be necessary.

N.B. A contracture of one joint can affect movement in another, so take this into account.

(1) **If you are assessing a flexion contracture of the hip**, flex the other hip as far as it will go. This will correct any lumbar lordosis, which may disguise as much as 60º of fixed hip flexion. Extend and abduct the hip, because a tight adduction contracture may be responsible for most of the deformity.

(2) **If you are assessing a flexion deformity of the knee**, do so with the hip in both neutral and flexed positions. Assess a *varus* or *valgus* deformity from the line of the shaft of the femur. Assess backward or lateral subluxation of the tibia on the femur as mild, moderate, or severe. Assess external rotation of the tibia on the femur with the knee extended as much as possible. Be careful to assess whether an immobile stiff straight knee may be more of a hindrance in a rural setting than a fixed flexed knee.

(3) **If you are assessing an *equinus* deformity of the ankle**, do so with the knee flexed and extended, because this will help in deciding management. If the deformity is in the ankle joint, it will be the same whether the knee is flexed or extended. But if the deformity is in the *gastrocnemius* muscle (35-20B), which spans both the knee and the ankle, as in polio, the range of movement in the ankle will vary with the position of the knee. So, if this is short, an *equinus* deformity of the ankle will be less if the knee is flexed than if it is extended, because the *gastrocnemius* is not being stretched by an extended knee.

(d) Range of movement. In the anatomical position all joints are at 0º, so record the movements there from this position, and state whether they are active or passive.

For example, the range of movement for a normal hip could be: flexion 0º/120º, that is from 0º to 120º. Its other movements might be extension 0º/10º, abduction 0º/40º, adduction 0º/30º, internal rotation 0º/30º, external rotation 0º/60º. 'Normal' people vary somewhat.

A patient with a flexion contracture might have: flexion 30º/110º, extension -30º/-30º (this means that there is no extension in the hip, movement starts at -30º of extension and ends there), abduction 0º/20º, adduction 0º/20º, internal rotation 0º/10º, external rotation 0º/40º. This means that the hip is flexed, but will not extend at all; it will flex a bit more, but not as much as normal. In other directions its movements are slightly limited.

(e) Charting muscle power

This is important if there is a lower motor neurone lesion, such as that following polio, or an upper motor neurone lesion as the result of spinal cord injury.

Muscle power is graded from 0 to 5. The important grade is 3, because this is the grade at which a muscle is just able to do its work against gravity.

MUSCLE POWER

Grade 0	no power, not even a flicker.
Grade 1	a flicker of movement, but no more.
Grade 2	movement with gravity eliminated.
Grade 3	movement is just possible against gravity.
Grade 4	movement is possible against gravity and some resistance.
Grade 5	full normal power.

It varies with the muscle; the quadriceps, for example, has to lift a heavy leg against gravity, whereas the extensor of the little finger has only a finger to lift. Any muscle which can lift its part of a limb against gravity must have a power of at least 3. Charting is difficult to do accurately (32-1), especially in young children. In an older patient, tremors, rigidity owing to Parkinsonism or a patient pretending disability can easily deceive you.

MUSCLE POWER CHARTING

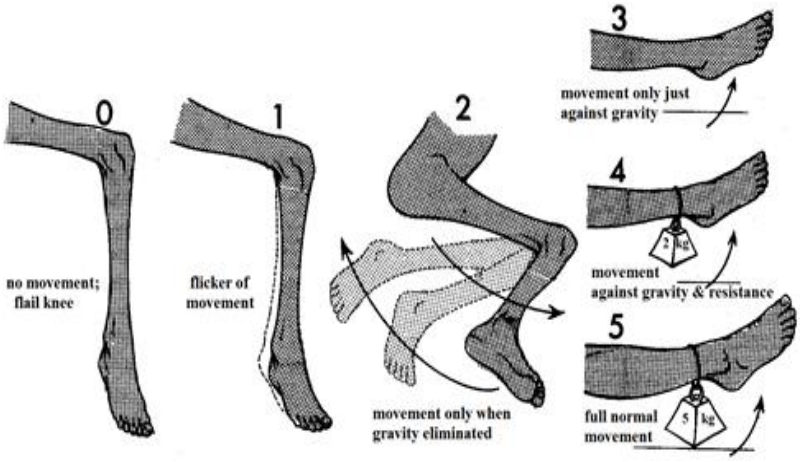


Fig. 32-1 MUSCLE POWER CHARTING.
Grade 3 is the critical level, because this is the grade which allows a patient to lift the limb against gravity. You can test all other muscle groups in the same way.
Kindly contributed by Ronald Huckstep.

(f) Radiographs

If you think the contracture involves more than muscle, X-ray the bones and joints involved. Look for: deformity of the joint surfaces, evidence of active disease, and the degree of osteoporosis.

(g) Treatment

NON-OPERATIVE METHODS

Try these first. You have several choices:

(1) You can use active and passive movements. These might seem the simplest, but they need a determined physiotherapist, or someone, such as a nurse, with some physiotherapy training.

(2) You can apply skin or skeletal traction.

(3) You can manipulate a joint.

(4) You can apply serial corrective casts.

Manipulation and casts can often be usefully combined. For example, you can manipulate a joint, and then apply a cast almost at the limit of its range of movement. Later, you can manipulate the joint again, and replace the cast with another one, in which the joint is nearer to the limit of its normal range of movement. If manipulation is to be thorough, you will have to use GA. The danger is that, during manipulation, a joint may bleed, or a contracture split and ultimately cause more adhesions. You can easily break a bone when you manipulate it, so follow the instructions we give, which are designed to prevent this happening. You can also introduce an angle in a cast, by

putting in a wedge, and combine it with manipulation by applying a ratchet.

The need for treatment usually means that prevention has failed. Intervene when contractures result from burns (34.2), polio contractures (32.7,8) and paraplegia.

(1) ACTIVE AND PASSIVE MOVEMENTS FOR CONTRACTURES
These may gradually stretch shortened soft tissues and correct the deformity. If possible, encourage active movements, or alternatively passive movements (done by someone else). Most useful are assisted active movements:
(i) Support the limb while the patient gently moves it himself. This eliminates gravity and gives him a greater feeling of security.
(ii) At the extremes of movement use a little passive movement in addition to active movement. Chart the range of movement weekly.

(2) TRACTION FOR CONTRACTURES
If satisfactory correction is not possible by exercises alone, consider skin or skeletal traction.

(3) MANIPULATION FOR CONTRACTURES
This is often combined with casting.

INDICATIONS
(i) Joints in which active and passive movements or traction have failed, or are not possible because the deformity is too great.
(ii) Hip contractures of <45º.
(iii) Knee contractures of <30º.
(iv) Ankle contractures of <20º.

METHOD. Press firmly for at least 5mins in a direction opposite to that of the contracture. If necessary, repeat the manipulations every 2wks.

CAUTION! Before you begin, remember that a bone which has not been moving is osteoporotic and breaks easily. To prevent this, reduce the leverage that you can exert, by holding the bones close to the contracted joint (32-2).

(i) HIP
Flex the opposite hip to eliminate a lumbar lordosis (bent back). Press the upper ⅓ of the thigh backwards, to bring the leg down on the table in slight abduction. This will also stretch the adductors, which will probably be tight.
Laying the patient prone is a very useful nursing procedure for preventing and treating flexion contractures of the hip. If tolerated, use the prone position with a pillow under the lower thigh. This uncomfortable position is more acceptable if the head faces towards the middle of the ward, rather than the wall.

(ii) KNEE
Hold the knee close to the joint; otherwise you may break the tibia or the femur, displace the epiphyses, or sublux the tibia on the femur.

MANIPULATING JOINT CONTRACTURES

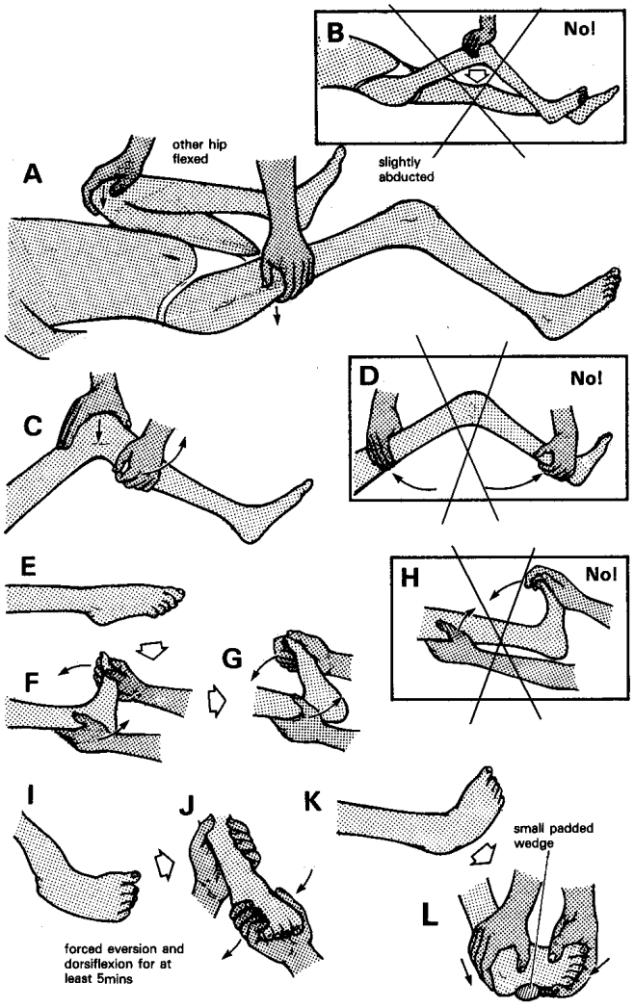


Fig. 32-2 MANIPULATING CONTRACTURES.
When you manipulate a joint under anaesthesia, *always exert pressure close to a joint,* or you may break a bone or displace the epiphyses. A, when you manipulate the hip, flex the opposite hip, and grasp the thigh. Push it down in extension and slight abduction. B, *don't push down on the knee!* C, for knee contractures, exert pressure near the knee, and *not as in D!* E, for an *equinus* deformity of the ankle, F, grasp it near the ankle, and G, dorsiflex it, *not as in H!* I, for a *varus* deformity of the foot, J, evert it and dorsiflex it for at least 5mins. K, manipulate an adduction deformity of the forefoot L, firmly and gently over a wedge.
Kindly contributed by Ronald Huckstep.

CAUTION! Don't try to release contractures of the knee too forcibly; you may injure the popliteal nerve, or damage the joint.

(iii) ANKLE
If there is an *equinus* deformity, support the ankle, and firmly dorsiflex the foot. If there is a *varus* deformity, or an adduction deformity of the forefoot, be especially firm and gentle. Don't push up the forefoot only; this may merely extend the mid-tarsal and tarso-metatarsal joints, without extending the ankle.

(4) CASTING FOR CONTRACTURES
Apply a well-padded plaster cast, close to but not at the extreme range of movement of the joint. If you do, pressure on its cartilage may cause necrosis and osteoarthritis later. So let it relax a little, before you apply the cast. A few weeks

later, if necessary, manipulate the joint again, and replace the cast with another one, in which the joint is nearer to the limit of its normal range of movement.

CAUTION!
(i) *Never put a joint, especially a knee, into a cast under tension*.
(ii) *Don't wedge a cast to correct a knee contracture*.
Both mistakes may cause an early painful osteoarthritis in what was previously a painless mobile joint. These are both very important rules. Fortunately, osteoarthritis and painful joints are rare in polio; it is tragic to create them unnecessarily.

OPERATIVE METHODS
You can release the soft tissues if there is a burns contracture or Dupuytren's (34.2). If there is polio, you can release the tendons of the ankle (32.9), the knee, or the hip (32.8). If the contracture is severe and longstanding, try to arrange a release combined with a myocutaneous flap, or by an osteotomy.

CONTRACTURES CAN FORM IN A FEW DAYS

32.2 Leprosy paralysis

The best way to deal with leprosy is to recognize it early, and treat it adequately. If this fails, surgery is necessary, because leprosy affects the nerves. Destruction of their sensory fibres makes the surface of the body anaesthetic, and thus liable to injuries that result in open wounds and ulcers. Destruction of their motor fibres causes paralysis, wasting, and sometimes contractures of the muscles. Most nerves are mixed, so that both things happen at the same time, with the result that the arms and legs become paralyzed and anaesthetic. Because there is little sensation of pain, injury is not noticed.

This results in neglect of the painless surface injuries, so that they become steadily progressive ulcers. The contractures, ulcers, and deformities that result are not an inevitable part of leprosy. In a well-conducted leprosy programme, there should be few such complications when patients first present and none later.

Leprosy most commonly involves the legs, but it can also involve the hands (32.18) and the eyes (28.17). Pyogenic organisms readily enter through the lesions that leprosy causes in the skin, so that you may need to drain abscesses (6.2), treat bone, joint, and tendon sheath infections (8.12), and enucleate the eye when its globe has become infected (28.14). Admit leprosy patients to the general ward. If your staff behave naturally towards them, other patients will do so too. Leprosy is not contagious and not particularly infectious.

Obtain a firm diagnosis with a split skin smear made from 6 sites (ear lobe, forehead, buttock, arm, knee and suspect lesion). Clean the skin fold with alcohol, pinch it to reduce blood flow, and incise it with a sterile blade. Turn the blade through 90º and scrape the skin, putting tissue on a slide and staining it by the Ziehl-Neelsen method. Examine using a x100 oil immersion lens and grade smears as paucibacillary or multibacillary.

Follow WHO treatment guidelines:

Type of leprosy	Monthly supervised dose	Daily dose	Duration
Paucibacillary (TT)	Rifampicin 600mg	Dapsone 100mg	6 months
Multibacillary (LL)	Rifampicin 600mg + Clofazimine 300mg	Clofazimine 50mg + Dapsone 100mg	12 months or more
Paucibacillary (TT) single lesion	Rifampicin 600mg + Minocycline 100mg + Ofloxacin 400mg		single dose

Surgically, your task is to:
(1) Care for the primary and secondary impairments.
(2) Set and record measurable objectives for preventing and limiting the disabilities, and plan how you are going to reach them.
(3) Provide protective footwear and aids.
(4) Teach self-care to prevent further disability.
(5) Teach the rest of the healthcare team how to do these things. Most leprosy work should be done by paramedical workers, and the present trend is for vertical programmes, with a specialized cadre of leprosy assistants, to be replaced by horizontal ones which manage many diseases. Much of what is described here can be done by paramedics, if you teach and encourage them.

If patients have good T-cell immunity, they get a milder disease, tuberculoid (TT) where the nerves are involved early as in borderline tuberculoid (BT) leprosy, and later and less severely in lepromatous (LL) leprosy. Here there is poor T-cell immunity and the presence of nodules and plaques. This involvement can be slow, progressive, and irreversible, or can occur suddenly in a Type I reaction.

Paralysis, whether slow or sudden, involves the nerves selectively:
(1) the facial nerve, so that the eye does not close (lagophthalmos).
(2) the ulnar nerve at the elbow or wrist, so that the hand becomes clawed.
(3) the median nerve at the wrist, so that the thumb cannot be opposed.
(4) the radial nerve, so that the wrist drops (in the arm, the ulnar nerve is most often affected, then the median, then the radial).
(5) the lateral popliteal nerve at the neck of the fibula, so that the foot cannot dorsiflex and so there is a 'foot drop'.
(6) the posterior tibial nerve behind the ankle, so that the intrinsic muscles of the foot become paralyzed, the toes clawed, and the sole anaesthetic.

Both kinds of lepra reaction can cause paralysis, but need different management:
(a) Type I lepra delayed hypersensitivity (reversal) **reactions** often cause sudden reversible paralysis in treated borderline forms, BT, BB, BL (immunologically unstable) leprosy. They make all the leprosy lesions in the skin and nerves swell acutely. The nerves become suddenly

paralyzed, and feel large and soft. They may be painless or tender. Skin lesions may ulcerate, and resulting fibrosis *may lead to contracture, unless you start physiotherapy.*

Suppress these reactions with prednisolone 40mg od (or 60mg od in severe cases) for 2wks, or as long as there is activity. Then as soon as the acute stage has subsided, reduce the dosage by 5mg monthly, even if there is no sign of nerve function returning. The total duration of treatment takes 6 months but may be more in BL patients.

(b) Type II 'erythema nodosum leprosum' (ENL) reactions occur in LL and BL patients. During 2-3h a crop of painful erythematous papules develop, typically on the extensor surfaces of the limbs, but in severe attacks over much of the body except the scalp. The skin may be thickened, especially over the backs of the hands and on the legs, where contractures may form. There is a high fever, malaise, headache, and anorexia with uveitis, swollen joints and testicles. Meanwhile, the nerves are painful, and become steadily paralyzed. Unfortunately, they are less likely to recover than after a Type I reaction. ENL frequently recurs, even up to 7yrs. Suppress a severe reaction with prednisolone 60mg od, reduced rapidly within 2-4wks. Treat mild reactions with aspirin. Use thalidomide 10mg/kg for recurrent reactions, reducing to 100mg od (but *remember this drug is teratogenic!*). Use clofazimine 300mg od for maintenance once the acute phase has settled, reducing by 100mg every 2 months.

Continue antileprotic drugs in both types of reaction. The nerves may start to recover within 3wks, or they may not improve for 3 months, or a year, or longer. Meanwhile, manage them as described below.

Physiotherapy in leprosy
A limb which is paralyzed by leprosy needs physiotherapy to strengthen its muscles and prevent contractures, especially if paralysis is recent, actively progressing, or possibly only temporary, as in either type of leprosy reaction. As long as there are signs of weakness, someone, preferably the patient himself, must put all the paralyzed joints through their full range of movement each day, even if they cannot be actively maintained in their positions of function.

Protect the paralyzed muscles by splinting the joints in their positions of function during sleep, and never allow a muscle to be overstretched. Make sure active exercises continue in order to retain mobility in all the joints. Even if all the intrinsic muscles of the hand are paralyzed, it will still be more functional if its joints are kept mobile with daily exercises. Start this protection the first day you diagnose a reaction.

When there are signs of recovery, as shown by pain decreasing, nerves becoming softer, and sensation and motor function returning, the patient must increase the range of active movement and strengthen the muscles with carefully graded active exercises, and practise any skilled coordinated movements that he will need later when he returns to normal life. Start exercises as soon as the acute symptoms of neuritis have subsided. Begin by doing each exercise 5 times, increasing to a maximum of 30 times, repeated 3-5 times daily.

Teach the exercises for the patient to do himself at home, but if you plan reconstructive surgery, he may need more intensive activity.

32.3 Bone and joint tuberculosis

When TB involves the skeleton, it is the involvement of the joints that matters most: the spine, hips, knees, feet, elbows, wrists, and shoulders, in this order of frequency, and occasionally other joints also. Bacilli reach the joints usually from some focus elsewhere. So look for lesions in the lungs, and for signs of TB in other parts of the body, especially lymph nodes (17.4). *These are frequently (in 70%) absent.*

The patient is often a young adult, or a child >6yrs, although children as young as 1yr and older people can also be infected, especially with HIV disease. One or more of the joints has become progressively painful and stiff during the previous few weeks. If the leg is involved, the first complaint is a limp. The infected joint fills with fluid, and the muscles round it waste. There is usually only mild to moderate pain, except on forced movement. Tuberculous arthritis is 'cold', which means that the skin over the infected joint is the same temperature as the normal skin. (The joint is not 'hot', as it is in septic arthritis.) Sometimes there are systemic symptoms, such as mild fever, night sweats, or loss of weight or appetite. Pain and fever may be quite marked. There may also be signs of tuberculosis in the chest, or a family history of it.

In a synovial joint (or diathrosis, where the fibrous joint capsule is continuous with the periosteum), the disease starts within the synovium and spreads slowly over the cartilage; it then extends through the cartilage into the underlying bone, which decalcifies. In the spine, disease starts in a disc. If you start treatment before the cartilage is destroyed, the joint will recover fully, or nearly so. Later on, the articular cartilage will be destroyed, so that even if the disease is arrested, the joint will develop a fibrous ankylosis (except in the spine, when ankylosis is always bony, 32.1). Sometimes, cold abscesses form, become secondarily infected, and may track for a considerable distance to produce a sinus far from the original lesion. If a tuberculous joint is secondarily infected, the ankylosis that results is always bony. The diseased limb develops a flexion contracture, and its joints may subluxate or dislocate, especially the hip, knee, shoulder, or elbow.

You can treat tuberculous joints successfully and cheaply if you make the diagnosis in the first few weeks. But even if treatment starts late, when joint surfaces have already been destroyed, you can expect a fairly good result, if you can prevent deformities and contractures.

There are no certain diagnostic signs, so the secret is always to be suspicious. Whenever you see any chronic bone or joint disease, ask yourself whether this could be TB. Taking an aspirate or biopsy of a node or the synovium will confirm the diagnosis in about 50% of cases. *Don't send specimens in formalin!* If you cannot send tissue for biopsy, you will probably have to rely on the characteristic radiographic changes.

(1) Use the drugs needed in adequate doses for an adequate period: *this is much the most important*.
(2) Rest the joint; if the arm is involved, you can usually treat it in a sling, but if there is tuberculosis affecting the leg, use POP; you will need to provide in-patient care.
(3) If there is disease of the hip or knee, apply traction. This will overcome spasm, prevent the softened bone from collapsing, and keep the inflamed joint surfaces apart, *e.g.* with an X-fix.
You may have to refer to experts to remove or drain a tuberculous lesion, or promote ankylosis.

(a) Radiographs. Look for:
(1) Generalized rarefaction: the whole joint is less dense than it should be. The earliest stage is a lack of definition; the joint is not as sharp as it should be.
(2) Localized areas of erosion or decreased density, caused by caseous lesions in the bone.
(3) Abnormally narrow or wide joint space.
(4) Irregular joint space, in late cases.
CAUTION! Joint destruction in TB is always more severe than the radiographic appearances suggest.
Remember to X-ray the lungs.

(b) Special tests
(1) A +ve tuberculin test is only of limited value (5.7).
(2) If a joint is swollen, aspirate it (7.17), and examine the fluid. With great patience, you may occasionally find AAFB in a stained film of the exudate.
(3) If there is any enlarged lymph node that might be tuberculous, aspirate (17.2) or biopsy one. Biopsy of synovial tissue is indicated in special cases only. Taking a biopsy from the spine is difficult, but you may be able to take one from the hip. Use the anterolateral approach, as for septic arthritis (7.18). Biopsy the knee (7.16). When you take a biopsy, use the opportunity to examine the articular cartilage. A biopsy is useful for distinguishing tuberculosis from late, imperfectly treated staphylococcal arthritis.
CAUTION! Biopsies are fallible, so accept a 'negative' biopsy result with caution. *About 50% of cases of tuberculous synovitis are reported as 'non-specific chronic inflammation'*.

(c) Differential diagnosis
Suggesting septic arthritis (7.18): a history of onset over hours or days, not weeks; a 'hot' joint which is acutely painful to move in any direction. There is a high fever with a leucocytosis. Aspiration produces frank pus, rather than slightly cloudy fluid. Bacteria (usually *staphylococci*) are visible in a Gram-stained film. If septic arthritis has been partly treated, diagnosing it may be difficult.

If the hip is involved, flex the knee to 90º and then flex the hip. If the leg moves into external rotation as you do this, (7-17), it is a sign that the upper femoral epiphysis is slipping. This is much more likely to happen in septic arthritis (or spontaneous slipping of the epiphysis) than in TB.

Both tuberculous and septic arthritis eventually involve pelvic bone. If it is already involved when you first see the patient, this suggests tuberculosis.

Suggesting trauma: a history of injury, a haemarthrosis: the radiograph may be normal, or showing widening of the joint.

Suggesting other forms of arthritis: a history of dysentery, brucellosis, or gonorrhoea.

Suggesting HIV disease or rheumatoid arthritis: presenting in a single joint; looks like tuberculosis, but tests are often non-specific. Other joints may flare up later. If possible, take a biopsy, aspirate fluid for a PCR test.
Remember that TB is commoner than mono-articular rheumatic disease in most parts of the world.

Suggesting Perthes disease (32.14) **or a slipped epiphysis:** the patient is a child and the hip is involved. Look for unavoidable passive external rotation of the hip when the patient tries to flex it (Drehmann's sign: 7-17).

Suggesting osteoarthrosis: in the old, or with a previous injury; look for osteophytes, areas of increased density (eburnation), and sometimes associated cysts (especially in the hip).

(d) Treatment
Admit the patient in order to:
(1) Confirm the diagnosis.
(2) Advise that the disease is curable, but that this needs long-term treatment.
(3) Apply traction to the lower limb, if this is needed.
(4) Start standard TB therapy (5.7) and screen for HIV disease. Do all you can to continue treatment to the end. Review regularly. When the course of treatment is completed, warn that the joint may flare up again at any time. If so, further treatment will be necessary.

(e) Position of function. The range of movement of the joint may be limited or absent, so make sure that it is kept in the position of function (7-16).

(f) Ankylosis. A fibrous ankylosis may be acceptable, even in the leg, especially in a child. It is also acceptable in the arm, provided it is near the optimum position of function. You can do this by compression with an X-fix.

UNTREATED BILATERAL HIP TUBERCULOSIS

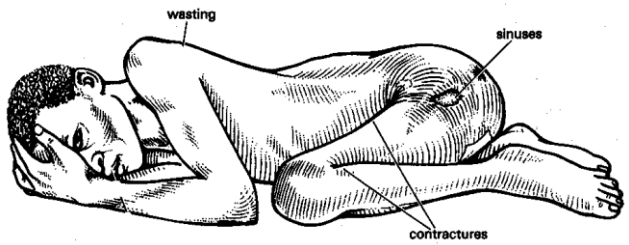


Fig. 32-3 NEGLECTED TUBERCULOSIS OF BOTH HIPS for 29yrs. This patient could not even crawl. He dragged himself along in a sitting position, with both knees and hips fully flexed.
Kindly contributed by Ronald Huckstep.

TUBERCULOUS OSTEITIS

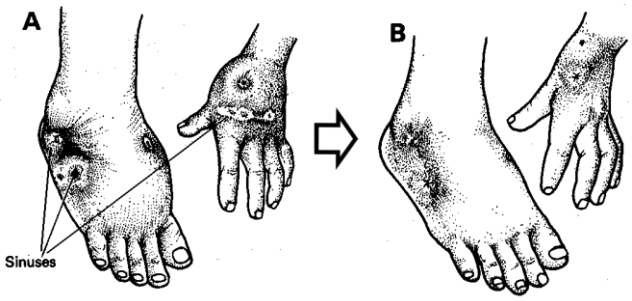


Fig. 32-4 TUBERCULOUS OSTEITIS in this patient improved rapidly on TB treatment, and the sinuses healed.
Kindly contributed by Gerald Hankins.

(g) Particular joints infected by tuberculosis

(1) SHOULDER. Aim for a loose fibrous ankylosis. Rest the arm in a sling, and then gradually encourage movements without it. If this is still painful at the end of treatment, refer him; an arthrodesis is necessary. This will not be a significant disability because of remaining scapulo-humeral movement.

(2) ELBOW. An elbow fixed in the position of function (7-16) is likely to be better than a stiff painful one. If non-operative treatment fails to produce a pain-free elbow, an excision/arthroplasty is necessary. This will provide a considerable range of movement, but little stability. Fusion is rarely necessary.

(3) HIP. In children presenting with a painful hip or a limp (32.14), symptoms may start slowly, but ultimately become serious with severe illness, and painful restriction of the movements of the hip. To begin with, it is flexed and abducted; later it is flexed and adducted, the leg is shortened, the thigh is wasted, and there may be abscesses in the buttock or groin. There is loss of joint space, and a characteristically severe rarefaction of the bone round the hip. If possible, aspirate or explore the hip, so as to confirm the diagnosis bacteriologically. Start TB treatment and rest the hip, at first in bed only, and then, when pain is a little less, apply skin traction.

If there is abscess formation, and the whole of the head of the femur is necrotic (uncommon in TB), its removal is necessary (7.19).

If the hip is in spasm (as diagnosed by rolling the leg), or the hip or knee shows any flexion deformity, apply extension traction for 6wks. This will control pain and prevent a flexion contracture.

If there is only narrowing of the joint space, and no bony destruction, mobilize, usually after 2 months, and allow cautious use of the leg, starting with partial weight bearing, using crutches and a patten (raised shoe) on the normal leg to keep the diseased one off the ground. Skin traction should have corrected any flexion contracture (if present) by this time.

If there is considerable bony destruction, especially of the head of the femur, there is still some hope of a reasonably functioning joint. Don't worry about whether the ankylosis is fibrous or bony. Apply skin traction for 3 months and then mobilize on crutches.

If, after 4-6 months, there is still a painful joint with very limited movement or no movement, except under GA (unusual), an arthrodesis of the knee or of the hip is necessary. Do both operations during the first 2yrs, while the patient is still on TB treatment.

(4) KNEE. Presenting with a limp, mild pain, a swollen knee, marked wasting of the quadriceps, limitation of movement (especially extension), and a flexion deformity. Rest the knee in a straight (Thomas) splint, or by extension skin traction, for at least 3 months, and then allow gentle weight-bearing on crutches. Gradually increase this until the patient is walking as well as he can. If the disease is advanced, or if the pain continues, fit him with a long leg plaster cylinder; otherwise avoid this.

If a child requires an arthrodesis of the knee, try to delay this until after adolescence, so as not to hinder growth.

(5) ANKLE. Apply a short leg walking cast.

(h) Tendon sheaths. If a chronic swelling of the tendon sheaths of the hand or bursae round the shoulder develop, don't forget that tuberculosis can also involve any of the synovial membranes.

(i) Difficulties with a tuberculous joint

If the symptoms are mild so that diagnosis is difficult, you can:

(1) Wait 4-6wks before committing yourself to long-term treatment, provided you are sure you are not missing acute untreated septic arthritis. During this time some diseases (transient synovitis and rheumatic fever) will settle, while others may reveal themselves (partly treated septic arthritis). Tuberculosis will not advance much during this time.

(2) Explore the joint, biopsy the synovial membrane, and remove a lymph node for biopsy. An ESR may also be useful.

Alternatively, and less satisfactorily, you can start a trial of treatment with streptomycin and isoniazid for a month.

If your diagnosis was correct, the spasm in the muscles round the tuberculous joint will become less, and the general symptoms will improve.

If you are not sure if there is septic arthritis or tuberculosis, even after opening the joint, treat for both, and review later when a histological report is available.

If deformity prevents satisfactory walking, corrective surgery is essential. If an arthrodesis is needed (more likely in the knee than the hip), it is usually best done 6-8wks after treatment starts.

If an old tuberculous joint is injured, observe closely. Fibrous ankylosis is always unsafe, and can flare up at any time. If pain and inflammation continue, and there is no bony injury and no ligament rupture, start another full course of TB treatment.

If a cold abscess develops, aspirate it repeatedly with a wide-bore needle, introducing the needle through a long oblique track, so that a sinus is less likely to form.

If the abscess is very large, explore it, clear out its contents, and close the wound to prevent secondary infection.

CAUTION! Don't leave a drain in a cold abscess, as you risk secondary infection.

If a sinus develops, it is the result of an abscess opening onto the skin, and occurs in immuno-compromised patients. Sinuses are rare once TB treatment has started, although an old sinus may re-open up temporarily. TB treatment will usually close it. A sinus may become secondarily infected, but does not require specific treatment. *A biopsy from the track is unlikely to confirm tuberculosis*, because non-specific granulation tissue lines it.

If a joint becomes warm and tender, with deteriorating radiographic signs, and there is fever and malaise, this is a flare-up. This is unlikely to happen if the course of treatment is completed, and is a sign that TB treatment has failed. Consider some other disease, such as septic arthritis, gonococcal arthritis, mono-articular rheumatism, gout or malignancy.

If the leg becomes significantly shortened, provide a shoe raise.

ANY CHRONIC JOINT INFLAMMATION IS TUBERCULOUS UNTIL PROVED OTHERWISE

32.4 Spinal tuberculosis

The spine is the most common and the most dangerous site for skeletal tuberculosis. Symptoms are mild. Infection usually starts in the anterior part of a disc (7.15), and spreads to the adjacent surface of the body of a vertebra, or to 2 adjacent ones. It seldom involves the lamina. The result is that, as the bodies of the vertebrae collapse, the spine angles forwards to produce a *kyphus* (an increase in the normal convex curve of the spine; a *scoliosis* is a lateral curvature). The shape of the spinal deformity depends on how many vertebrae are diseased. Commonly, as the deformity gets worse, a sharp angle (*gibbus*, 32-5B) appears. Uncommonly, the destruction is not symmetrical, so that the spine rotates. Symptoms are more severe; several of the vertebrae are involved widely in the spine (including perhaps some in the neck), and the disc spaces may not be narrowed.

The first symptom is pain in the back, and the first sign is increasing kyphosis. Later, pus from the diseased vertebrae may track along tissue planes to present as a cold abscess in unexpected places, particularly in the groin (psoas abscess). Paraplegia may develop (32.5). Persistent localized pain at a specific place not relieved by rest or NSAIDs after 4-6wks should arouse a suspicion.

SPINAL TUBERCULOSIS

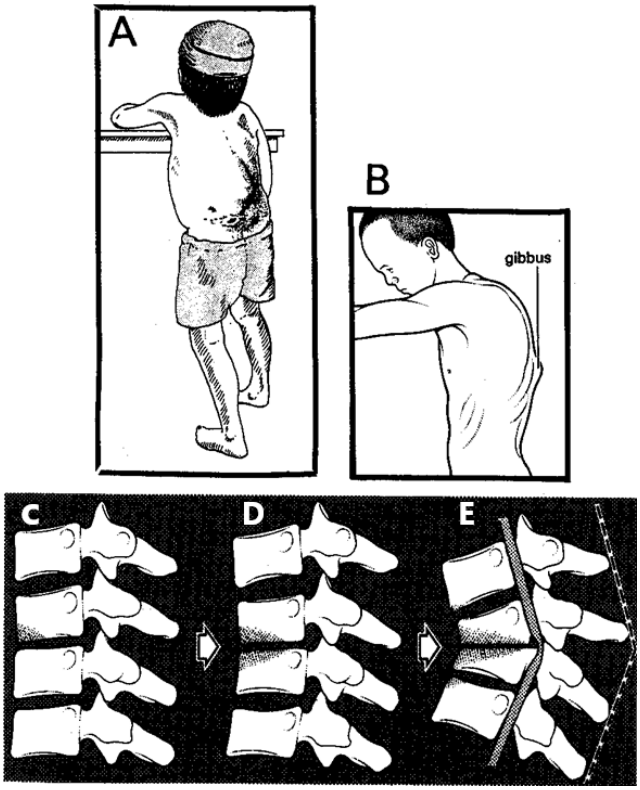


Fig. 32-5 SPINAL TUBERCULOSIS.
A, backache is the first symptom. B, *gibbus* is a late sign. Note that in both patients the lower thoracic region is involved (as is common). C, rarefaction of the anterior vertebral body, with early narrowing of the disc space. D, increased rarefaction and further narrowing of the disc space, E, collapsed vertebrae and obliterated disc space.
A, kindly contributed by David Nabarro.

In a child, spinal tuberculosis is an important cause of back pain, especially if associated with malaise and weight loss. There may be tenderness over the low thoracic or upper lumbar spine, and any of the signs seen in adults.

Start TB treatment as an outpatient, without applying a plaster jacket.

(a) Examination
Examine from the side; look and feel for:
(1) a *kyphus*,
(2) reduced movement of the lumbar spine,

(3) cold abscesses in the neck, paraspinal area, lumbar region and groin,
(4) sinuses.
Test the reflexes in the legs, and their tone, power, and sensation (32.1,6).
Look in the throat for a retropharyngeal abscess (6.8).

(b) Radiographs are critical (32-5). Remember to get chest films too. Look for:
(1) Narrowing or obliteration of a joint space, involving at least 2 vertebral bodies and the disc between them (this is the typical late picture). Sometimes several vertebrae disappear into the space normally occupied by only 1 or 2. So count the vertebral spines, because these may be all that is left when the vertebral bodies have been destroyed.
(2) Forward collapse of the spine.
(3) You may also see the shadow produced by a paravertebral abscess in the thoracic region (this strongly suggests tuberculosis, but it can be produced by staphylococcal and other forms of bacterial osteitis), and calcification in the psoas sheath, showing that a psoas abscess is forming. Evidence of a paravertebral abscess increases the probability of tuberculosis being the cause, but is not necessary for diagnosing it.
(4) Osteophytes and bridging (rare). If you do see bridging, it is more likely to be due to late staphylococcal infection.

(c) Ultrasound
Look for a fluid collection adjacent to the spine.

(d) Special tests
ESR may be very high. A falling ESR is a useful indication of response to treatment, but decreasing pain and tenderness is more important. Aspirate accessible abscesses with a 14G needle and look for AAFB.

(e) Differential diagnosis
Suggesting pyogenic osteitis or spondylodiscitis (7.15): a more rapid onset, less bone destruction, and a higher temperature. Confirming the diagnosis may have to depend on the aspiration and examination of pus from the spine, or on costo-transversectomy,

Suggesting poliomyelitis: weak, wasted, flaccid legs. If polio involves the spine, it is almost sure to involve the legs too. You will see scoliosis rather than kyphosis.

Suggesting idiopathic scoliosis: the curve is smooth, with no *gibbus* or muscle-wasting. Apart from the curved shape of the spine, there are no other signs; the radiographs are normal, and no vertebrae are destroyed. It is a disease of unknown cause, in which a child's spine slowly develops a curve. It may start as early as 3yrs, but more often starts at 7-8yrs; it progresses most rapidly from 12-14yrs, and gets worse until growth ceases.

If possible, fit a back (Milwaukee) brace, and if necessary get the spine fused at the appropriate time. If this is not possible, although the back will always be bent, spinal compression is rare, but a moderate or severe lesion will affect breathing by reducing the size of the thoracic cage.

Suggesting a congenital lateral hemivertebra causing scoliosis or **dorsal hemivertebra causing kyphosis** (usually mild): half of one of the vertebrae is missing. On a radiograph this is almost triangular, its edges are smooth and well formed, and there are no signs of disc destruction in the vertebra above or below. This kind of deformity does not progress with age, and needs no treatment.

Suggesting kyphoscoliosis due to lung disease: a previous history of empyema, other causes of lung fibrosis, or pneumonectomy. When the lung collapses, a collapsed thoracic cage may result. The spine itself needs no treatment.

Suggesting Burkitt's lymphoma: a child in endemic areas where this is the commonest cause of paraplegia (17.6).

Suggesting metastatic disease: involvement of the bodies of the vertebrae without involvement of their discs. The serum alkaline phosphatase is raised. If the primary is in the prostate, the acid phosphatase and PSA will be raised also.

Suggesting a senile kyphosis: an old woman with osteoporosis of all, or most, of the spine, and normal discs, which bulge into the softened vertebrae. The *kyphus* is gradual. Treatment is difficult (32.6).

(f) Treatment
Start TB treatment (5.7). Screen for HIV disease. If walking is possible, encourage this. Warn that treatment must continue, and that it will take some months to have much effect. During this time, the kyphoscoliosis may get worse, before it stabilizes. Do all you can to trace defaulters by keeping good records.

(g) Difficulties with a tuberculous spine
If the cervical spine is involved, treat with an orthopaedic collar or, failing this, a plaster corset and TB treatment. The spinal canal is larger in the neck than in the thoracic region, so the spinal cord is less likely to be compressed.

If there is clumsiness, weakness or incoordination, paraplegia is imminent (32.5).

32.5 Tuberculous paraplegia

If a patient with spinal tuberculosis complains of clumsiness, weakness, or incoordination of the legs, paraplegia is imminent. Later, the voluntary power of the legs is reduced, their muscle tone is increased, and the plantar responses become extensor. Later still, there are flexor spasms, and finally contractures.

Paraplegia is the major complication of spinal tuberculosis. In early cases it is due to an inflammatory oedema round a paraspinal abscess, and later to compression. Paraplegia may be the presenting symptom, and is usually treatable. In most cases it is motor only (unless it comes on

very rapidly), because the abscess presses on the anterior columns (grey matter, or motor neurones) of the cord rather than on the posterior columns (white matter, or sensory neurones).

Although tuberculous osteitis affects the various regions of the spine in the following order of decreasing frequency – low thoracic, lumbar, upper thoracic, and cervical – you will see tuberculous paraplegia most commonly in the thoracic region, sometimes in the cervical region, and seldom in the lumbar region. This is because the spinal canal is wide there, and the *cauda equina*, a loose bundle of nerves and nerve roots from L2-S5, is less affected by TB than the solid cord.

There are 2 types of tuberculous paraplegia:
(1) The common early type is due to inflammatory oedema which responds well to TB treatment, and surgery, if this is necessary.
(2) A less common later type, due to pressure and stretching from a bony deformity, when bony union has not occurred. It is the result of late, incomplete, or no treatment at all. Its prognosis is poor with TB treatment alone, and even with specialized surgery, it is not good. Obviously if there is untreated HIV disease, and maybe also HIV neuropathy, the prognosis is poor.

(a) Prognosis and management are different in the 2 forms of the disease. The bowels and bladder are sometimes involved in later stages; their improvement mirrors that of the limb muscles.

If the paralysis is fairly recent (<3 months) and the deformity is <60º (common), inflammatory oedema is the likely cause, and if the indications for surgery are followed, the prognosis is good. Even if there is >60º of deformity, this is worth managing as if oedema was the cause, but the prognosis will not be so good.

(1) If the muscle power (32.1) is fair (grade >3), it is almost sure to recover fully.
(2) If the power is significantly weak (grade <3) but without muscle spasms, it will probably recover.
(3) If the power is poor with extensor spasms, there is >50% chance of a full recovery, and if not, there will probably be a partial recovery.
(4) If the power is poor with flexor spasms, you can expect little or no recovery, and there is little chance of walking without special aids.

If the paraplegia is due to pressure or stretching from a bony deformity of the neural canal (uncommon in most areas and usually of late onset), the clinical picture is the same, except that the onset of paraplegia is late when kyphosis is marked.

However, even if there is marked bony deformity with no paraspinal abscess visible on radiographs, the paraplegia may possibly still be due to inflammatory oedema; surgery may still be useful but surgery of this kind of paraplegia is difficult. Otherwise give TB treatment alone.

(b) Treatment for tuberculous paraplegia

NON-OPERATIVE TREATMENT
If TB treatment (5.7) and bed rest don't cause neurological improvement in 6wks (unusual), review him. Screen him for HIV disease if you have not done so.
Your diagnosis may be wrong, but if you are sure that there is TB, consider costo-transversectomy.
NURSING CARE is the same as for traumatic paraplegia: so manage the patient's morale, the skin (pressure areas), the urine, and the bowels.
If you don't do this properly, there is no use in operating!

COSTO-TRANSVERSECTOMY

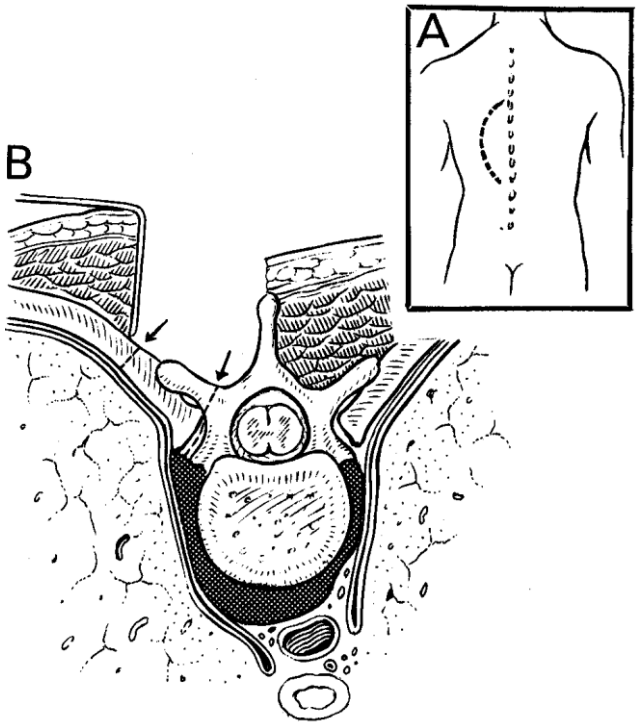


Fig. 32-6 COSTO-TRANSVERSECTOMY for a tuberculous or a pyogenic paraspinal abscess. A, incision. B, approach to the ribs and transverse processes.
After Campbell WD, Edmonson AS, Crenshaw AH (eds), Operative Orthopaedics. CV Mosby 6th ed 1980 Fig. 13.10, with kind permission.

COSTO-TRANSVERSECTOMY (GRADE 3.3)
INDICATIONS
(1) Osteomyelitis of the spine (7.15).
(2) Paraplegia due to osteitis (usually tuberculous), provided you can demonstrate a paraspinal abscess (tuberculous or pyogenic) on ultrasound or radiography.
(3) A large paraspinal abscess (tuberculous or pyogenic) when there is no paraplegia.
(4) To obtain tissue for histology, when the cause of an osteitis is still in doubt, after considering the clinical condition and the radiographs.

POSITION
Anaesthetize, intubate the patient and lay him prone with the chest supported on pillows, and mechanical ventilation, or lay him in the right lateral position.

PREPARATION
Have 2 units of blood in reserve. Diathermy is useful.
Find the vertebra or vertebrae most markedly involved, by noting the site of any *gibbus*, and examining the radiographs carefully. To make sure you choose the right level, get a radiograph done with a marker placed on the rib selected for removal: leave this marker in place.

INCISION
Make a 10cm incision centred on the affected vertebrae, curved to the left, and reaching the spinous processes at each end. Raise a flap of skin and subcutaneous tissues, and turn this medially, to expose the supraspinous ligament.

CAUTION!
(1) Approach the abscess from the left, so as to avoid the azygous vein (at some levels) and the vena cava. The aorta, being thicker, is much less easily damaged.
(2) Later, gentle dissection near the vertebral bodies will help you to avoid damaging the pleura and entering the pleural cavity.

With a knife, divide the posterior spinal muscles in the line of the skin incision, and strip the muscle mass with a stout periosteal elevator from the lamina of the vertebra and laterally from the back of the transverse process. When you have cleared the transverse process fully, divide it at its base with a sharp osteotome.

Clear the attached rib of muscle laterally to 8cm from the midline and then cut the periosteum of a rib longitudinally on its back, and strip it from the bone with a curved periosteal elevator all round, keeping close to the bone. This will help to separate it from the tissue covering the underlying pleura, and protect the intercostal vessels and nerve. Then cut the rib with rib cutters (or carefully with bone cutters), at the lateral extremity of the incision. *Avoid damaging the pleura*. Then grasp the outer end of the rib, and by a twisting movement remove it with the severed part of the transverse process at its base.

Now look for the paraspinal abscess. Insert your index finger along the side of the vertebral bodies, and separate the tissues gently. You may need some sharp dissection with scissors; if so, keep very close to the bone. This will lead you to the abscess, and *not the pleura!* Tuberculous pus is watery, with debris in it. Pus from osteitis is yellow and creamy. Drain and culture what you find. Pass your finger round the anterior surface of each vertebra, up and down to ensure thorough drainage.

N.B. Don't perform a laminectomy (*i.e.* dividing the posterior arch of the vertebra) as this may cause the spine to subluxate.

If you find no pus, check the radiograph; you may have chosen the wrong level. If so, re-examine the radiographs and remove a further transverse process and its related rib and feel again.

If you still find no abscess, take some tissue from the disc space for histology. The best place to biopsy is felt more easily than seen. Use cervical biopsy forceps, or dissecting forceps and a #15 blade mounted on a long handle.

CLOSURE. Preferably use suction drainage (4.9). There is no need for an intercostal drain, unless you damage the pleura. Approximate the muscles to the spine by sewing the spinal muscles to the supraspinous ligament with absorbable sutures. Close the skin with continuous 2/0 monofilament. Apply a dressing.

POST-OPERATIVE CARE
Insist on changing the patient's position 2hrly. He should be able to turn the upper part of the body by 48h, but he will still need 2hrly assistance with turning. He may show no improvement for up to 6wks.

If there is no improvement by this time, the outlook is poor. If improvement starts by 6wks, expect it to continue for 6 months. It will be hastened and improved by the drainage of a significant abscess.

If the paraplegia continues, treat with attention to possible complications.

CAUTION! Avoid an indwelling catheter.
Try using intermittent sterile catheterization if necessary!

DIFFICULTIES WITH COSTO-TRANSVERSECTOMY
If you damage the pleura, insert an intercostal drain (9.1).

32.6 Back pain and lumbar disc lesions

Backache is a very common symptom. Your task, as often, is to sort out those patients who need specific and sometimes urgent treatment, from those whom you can only help symptomatically.

The most common causes are:
(1) prolapsed vertebral disc
(2) osteoarthritis and senile osteoporosis in the very old.
Causes not to miss, particularly in children, are:
(3) pyomyositis (7.1)
(4) osteomyelitis (7.15)
(5) septic or tuberculous arthritis (7.16, 32.4).
Other causes include:
(6) malignant deposits in the spine
(7) back injuries including ligamentous sprains
(8) spondylosis, spondylolysthesis and ankylosing spondylitis.
Don't forget:
(9) pancreatitis (15.11)
(10) pyelonephritis
(11) retroperitoneal abscesses (6.15)
(12) a leaking aortic aneurysm (35.8)!

(a) Lumbar disc lesions are due to the protrusion of the *nucleus pulposus* of an intervertebral disc through a weakened area in its *annulus fibrosus*. This is the ring of firm fibrous tissue that holds the softer *nucleus pulposus* in place. Prolapsed tissue from the *nucleus pulposus* may press on a nerve root, and cause pain down the leg (32-7B,E).

Almost all lumbar disc lesions occur in the spaces L4/5 or L5/S1. Pressure on the S1 root causes pain down the back

of the thigh, calf, and outer side of the foot (32-7D). Pressure on the L5 root causes pain on the lateral aspect of the thigh and leg, and the dorsum of the foot (32-7C).

A protrusion from the L5/S1 disc usually presses on the S1 root (not on L5), but a protrusion from the L4/5 disc may press on the S1 or on the L5 root. Occasionally, when the protrusion is central, other roots are involved, and these may cause urinary problems.

Disc protrusions are more likely to occur in a back which has been weakened by a sedentary occupation, and may follow sudden flexion of the spine, or bumping in a sedentary position, as with truck drivers and their passengers in the back on bad roads.

The patient is usually a younger to middle-aged adult who presents with sudden severe pain in the front of the thigh, the back of the thigh (sciatic pain, which is present in most patients), the calf, or the foot. Movement, coughing, or sneezing all make the pain worse.

The dorsum of the foot may be numb, and its dorsiflexors weak, occasionally on both sides.

(b) Examination (32-8)
Remember to examine, for metastases, the prostate rectally in a man, and the breasts in a woman.

Measure the length of both legs: in many cases they are different, and a simple shoe raise will solve the problem.

In lumbar disc lesions, the lumbar spine is flattened, with loss of its normal lumbar lordosis, and slight scoliosis. There may be tenderness over the interspinous ligaments, at the site of the lesion, or on gently tapping the spinous processes. Movement of the lumbar spine is usually severely restricted. Forward flexion is always restricted, and is accompanied by spasm. Lateral flexion may be free, in one or both directions.

Lay the patient flat and raise the leg by the ankle. Straight leg raising is limited and flexing the ankle makes it worse. The ankle jerk (S1) may be diminished or absent. There may be also diminished sensation in the relevant dermatomes.

You can usually diagnose a disc lesion clinically. Most disc lesions are benign and self-limiting, and can be managed non-operatively, although they often recur.

N.B. Plain radiographs rarely show confirmatory signs. Look for mild scoliosis, loss of the normal lumbar lordosis and narrowing of the disc space. Radiographs must be well centred to show this. *If you take films obliquely, any disc space will look narrow*.

(c) A myelogram (38.1h) will show if sciatica is getting worse despite treatment, or if there is an acute cord compression, or deteriorating paraparesis. Obtain a chest radiograph especially in a smoker (looking for cancer) or if TB is likely.

(d) Differential diagnoses
Suggesting metastatic disease: an older patient with persistent spinal pain, both when active and at rest. The radiographs of the spine may be normal initially. Typically, there is a patchy osteoporosis of the bodies, and/or the arches, of the vertebrae (some metastases are sclerotic, especially those from the prostate). There may be a pathological fracture, especially of a vertebral body. The discs are spared. Look for the primary in the prostate (27.22), the breast (24.4), the bronchus (29.19), the thyroid (25.6), and the kidney (27.35), and for signs of multiple myeloma (32.20).

Suggesting an acute infective cause: pyomyositis (7.1)**, or osteitis of the spine** (7.15): an acute onset with fever in a child or young adult who is obviously very ill.

BACK PAIN

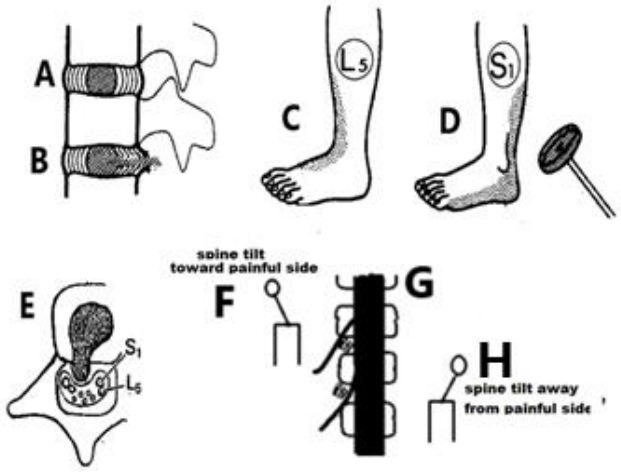


Fig. 32-7 BACK PAIN.
A, normal disc. B, ruptured annular ligament, with the *nucleus pulposus* protruding. C, L5 lesion causes loss of sensation on the dorsomedial aspect of the foot, and weakness of the dorsiflexors of the ankle, with sparing of the ankle jerk. D, S1 lesion causes loss of sensation on the lateral border of the foot, weakness of plantar flexors, and a diminished ankle jerk. E, prolapsed disc pressing on the large S1 nerve root. F, central protrusion risks involving S2, S3, S4 and S5 which control bladder function. G, if the disc protrudes medial to the nerve root, the patient tilts his spine towards the painful side to relieve pressure on the nerve root, whilst H, if the disc protrudes lateral to the nerve (at a lower level), he tilts away from the painful side.
After Apley AG, Solomon L. Apley's System of Orthopaedics and Fractures. Butterworth 6th ed 1982 Figs 18.20,21,23.

SPINAL EXAMINATION

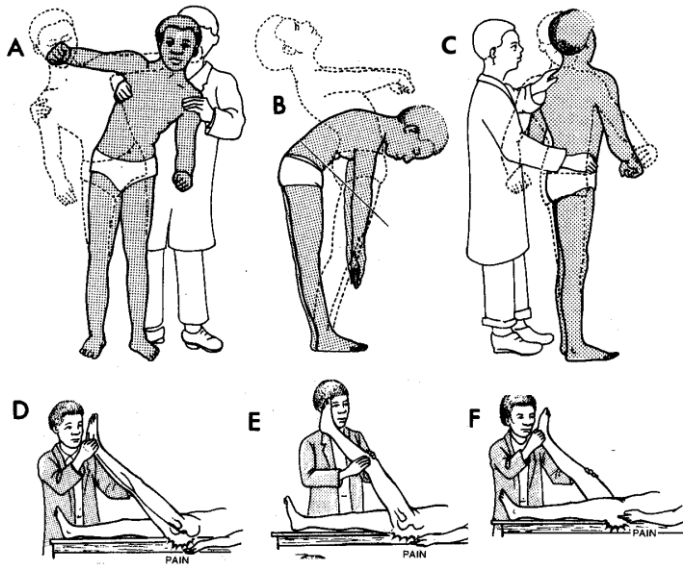


Fig. 32-8 EXAMINING THE SPINE.
A, flexion from side to side. B, flexion and extension forwards and backwards occur mostly in the lumbar spine. C, rotation occurs mainly in the thoracic spine. D, straight leg raising to test for sciatic irritation. E, confirm this by pressing in the popliteal fossa, or dorsiflexing the ankle, which cause sciatic pain through stretching. F, if you raise the opposite leg straight, this may also cause pain.

Suggesting tuberculosis (32.4): a slow onset with loss of weight, malaise, mild fever, and a *gibbus*.

CAUTION!

(1) Young children don't have disc lesions, so assume that all back pain in a young child is serious, until you have proved it is not.

(2) Back pain and fever are a serious combination.

So think of TB!

If a younger man has back pain and later stiffness, perhaps with inflammatory involvement of the other joints, consider the possibility of ANKYLOSING SPONDYLITIS (common in India, 32-9), a disease of unknown aetiology. Typically, the pain disturbs sleep in the early hours of the morning, is relieved by getting up and walking, and, unlike most other pains, is made worse by rest. The upper legs may ache, but radiating pain is unusual. There may be malaise. Test for bilateral sacroiliac tenderness, and pain over the sacroiliac joints on springing the pelvis (both early signs). All movements of the lumbar spine are restricted, sometimes with muscle spasm.

Chest expansion is also restricted (an objective early sign). Look for uveitis (28.5). The ESR is raised. The earliest radiographic sign is bony erosion of the lower ⅓ of both sacroiliac joints, followed later by secondary ossification and ankylosis of the whole joint. Early, the lumbar radiographs are normal; later there is a calcification of the intervertebral ligaments ('bamboo spine').

Teach exercises to help prevent severe curvature of the spine, and retain mobility. In the early painful stages, NSAIDs may help, *e.g.* indometacin 25mg tds.

Keep the major joints mobile, and if they do become fixed, try to ensure that this happens in the position of function (7-16).

(e) Treatment

Insist on bed rest in the most comfortable position, which is usually with the hips and knees flexed. Don't put pillows under the knees; they will immobilize the legs and promote deep vein thrombosis. Put boards under the mattress. Provide analgesia (if necessary, pethidine or morphine with NSAIDs for the first few days), and *don't let him get up, even to go to the toilet*. Make sure you turn a patient 2hrly if he cannot do so himself: do this with 3 assistants, rolling him and keeping the spine from bending. *There is no evidence that steroids help in the long term.*

Many patients improve without traction. If not, tie a band round the pelvis, and apply a total of 7-10kg to both sides for a maximum of 3-5 days. Raise the foot of the bed to apply counter traction. Alternatively, apply 4-5kg of traction to each leg with adhesive strapping.

Start active and passive movements of the legs as soon as the acute pain is over. When the pain is sufficiently improved, start back extension exercises. If symptoms improve, continue bed rest for 2-3wks. Then allow mobilization to the toilet, keeping the back straight. *Don't allow bending down*: insist on crouching with the hips and knees flexed, keeping the back straight, to pick something from the ground.

ANKYLOSING SPONDYLITIS

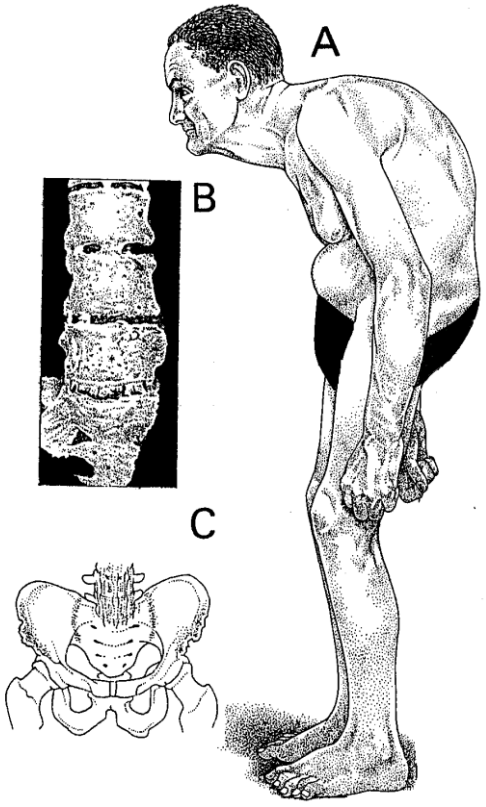


Fig. 32-9 ANKYLOSING SPONDYLITIS.
A, characteristic 'question-mark' posture of ankylosing spondylitis. B, bony bridges forming between the vertebrae. C, sacroiliac joints have fused and only the ghost of the old joint line is visible.
After Robin J in 'The Independent', permission requested.

(f) Indications for surgery
(1) Neurological deficit: if there is perineal numbness, loss of an anal reflex or incontinence of urine or faeces, surgery is urgent, otherwise incontinence may become permanent. Sensory loss in the S1, L5 or L4 regions usually means surgery is needed sooner rather than later.
(2) Significant weakness of the dorsiflexors (L5) or plantarflexors (S1).
(3) Failure of the pain to improve, despite 3-4wks in bed.

(g) Contraindications for surgery
(1) Be careful if the patient's neurological deficit does not correspond with the radiological findings; operation then, at the wrong site, obviously will fail to cure the problem!
(2) Make sure the neurological deficit is of spinal, not peripheral, origin, *e.g.* arising from a neuropathy (*e.g.* diabetic or HIV) or nerve injury (for example, from IM injections).

(h) Difficulties with back pain
If an older patient has pain on sitting or standing, or following manual activities, he is probably suffering from OSTEOARTHRITIS. Exclude other diseases. Keep the spine as mobile as possible with exercises. Encourage weight loss in obesity.

If an old patient, especially female, has a marked kyphosis, think of SENILE OSTEOPOROSIS, with a generalized loss of matrix and calcium, especially from the bodies of the vertebrae. The discs expand and compress the weak vertebral bodies. Painful pathological fractures are common, and there may be root pain. Treat symptomatically with analgesics, and encourage mobility, if necessary with the aid of a stick.

If an adult or older child has back pain, worse on standing, and often episodic, consider the possibility of SPONDYLOLITHESIS. In this condition the body of a vertebra (commonly L5) slips forwards on the vertebra below. It is often asymptomatic, and even if you find it, it may not be the cause of the pain, so exclude other causes. If you find it by chance, when there is no pain, do nothing. If there is pain, surgery may be necessary.

If a young man has leg weakness but has minimal back pain, consider HIV or schistosomal transverse myelitis, Guillain-Barré type neuropathy, or syphilis.

32.7 Chronic polio(myelitis)

Many children of the world have paid a high price for acquiring their natural immunity. Poliomyelitis is still found in Pakistan, Afghanistan, Madagascar, Myanmar and Central and West Africa, as well as war zones (Ukraine, Syria and Iraq). *In certain countries, there is an active effort to stop immunization, so this disease may soon acquire a rebirth.* Some cases of vaccine-derived polio have occurred in areas of inadequate vaccination and low immunity. The strategy to prevent this is to launch a high-quality, high-coverage immunization programme.

The polio virus destroys the anterior horn cells of the spinal cord. This causes a flaccid lower motor paralysis, without impairing sensation.

The flexor and extensor muscles of a normal limb are arranged so that they oppose one another's action. Polio can weaken both groups mildly or severely, equally or unequally. When, as is usual, the muscle groups are involved unequally, it is commonly the extensors which are most affected. When this happens, the stronger flexors pull the limb into a flexion contracture. If all the muscles of the limb are weak, there is a flail limb.

In a child, growth will cause further deformity. All this can happen in varying degrees to the hips, the knees, or the ankles, on one or both sides, to cause many patterns of paralysis. The arms are less commonly affected, and are usually less of a disability.

Make sure you have ruled out other causes of paralysis, especially HIV disease. Remember similar deformities may result from cerebral palsy. If you can correct physical deformities, mental deficiency will be much easier to handle.

CHRONIC POLIO

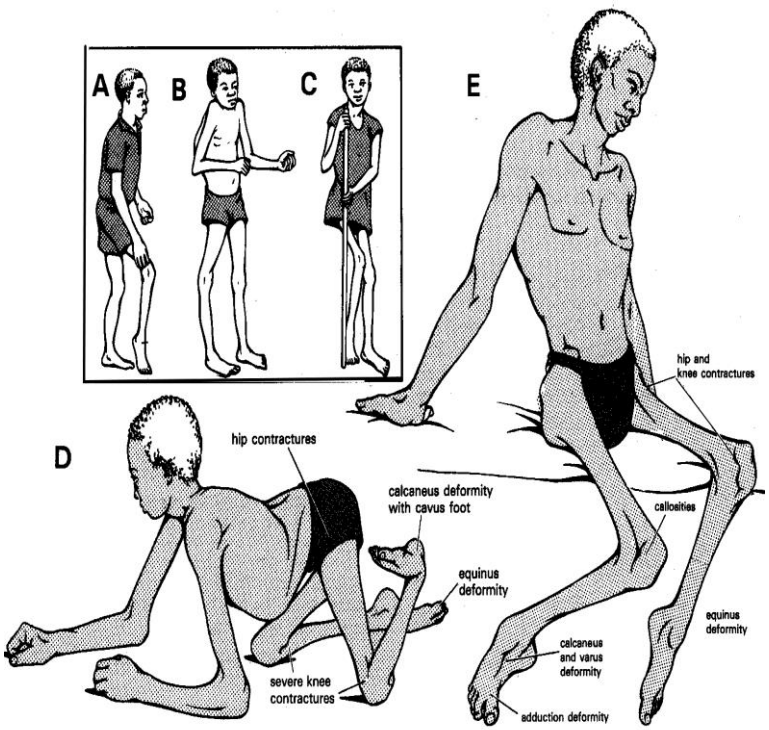


Fig. 32-10 CHRONIC POLIO.
A, a patient with a weak hip or knee may be able to walk by putting his hand on the thigh; *equinus* helps compensate the shortening, and B, by locking the knee in hyperextension, using the extensors of the hip. C, if the whole leg is weak, a stick will be necessary. D, if there are severe contractures of both knees, crawling on the ground may be the only way to get around. E, an adult 'crawler' seated. Note the large callosities on the knees.
Kindly contributed by Ronald Huckstep.

These should be your aims:
(1) Do all to promote the immunization campaigns in response to WHO's expanded programme of immunization (EPI).
(2) Try to prevent contractures developing immediately after the acute phase of the illness.
(3) If they do develop, use serial plasters, traction, or tenotomies to release some of the milder ones. More complex operations, such as osteotomies, arthrodeses, and tendon transfers, are tasks for an expert; so are all operations on the arms and spine, on the rare occasions when these are necessary.
(4) Provide patients with the necessary callipers (32-13A), crutches, and plaster splints.
(5) Follow them up for many years, if necessary, and help them to find places in schools, and to find jobs. As always in medicine, but particularly in polio, consider the total needs. Never treat a single joint without considering the other joints in the limb, the other limb, and the adaptations the patient has already made to the disability.

Your results should be good if:
(1) you operate carefully on the right indications,
(2) you use simple callipers (32-13A),
(3) you are able to provide the necessary physiotherapy and follow-up. (These last (2,3) are likely to be your main constraints.)

(a) The hip in chronic polio
If an adult or child has an isolated flexion contracture of the hip of <30º due to weakness of its extensors and adductors, he is probably walking adequately, and *needs no treatment, provided there are no other serious contractures*. The stability of the hip may even be improved and shortening compensated by a small adduction and flexion contracture.

If an adult or child has an isolated flexion contracture of >30º, consider releasing it.

If there is a flail hip due to paralysis of all its muscles, provide a pair of crutches.

If there is a hip dislocation or subluxation in a flail hip, reduce the dislocation, and apply an abduction plaster spica. Provide crutches. Occasionally, an osteotomy, an arthrodesis, or a psoas transfer is necessary. Reducing a dislocated hip can be difficult. It readily re-dislocates because there are no functioning muscles left around the hip to hold it in place. If the dislocation is recurrent, leave it alone.

(b) The knee in chronic polio
Not all weak knees need a complete calliper. Some patients may be able to manage with a knee splint.

If there is an isolated flexion contracture of the knee of <30º due to weakness of its extensors, apply serial casts for a child (32.1), and skin traction for an adult.

CAUTION! Never put a cast on a knee (or any other joint) while it is held under tension, or osteoarthritis will result (32.1).

If there is an isolated flexion deformity of the knee of >30º but <90º, release it surgically (32.8) by a very limited open tenotomy (32-15). Do this only if you need a little more extension in order to apply skin traction, when the *biceps femoris* tendon is tight, but not the *semitendinosus* and *semimembranosus*, which are attached medially (35-18). Feel the tendons when the knee is extended to its limit. If all the tendons are tight and need surgical release, more complex surgery is necessary.

If there is a flexion deformity of >90º, correction is going to be difficult, and a stiff painful knee may subsequently develop. If there is one contracted knee, either leave it alone, or consider an osteotomy or an arthrodesis. *An adult with 2 contracted knees is best left alone.*

If a small child has a severe *valgus* deformity of the knee, an osteotomy, or stapling of the medial epiphysis, by an expert, is necessary.

MORE POLIO DEFORMITIES

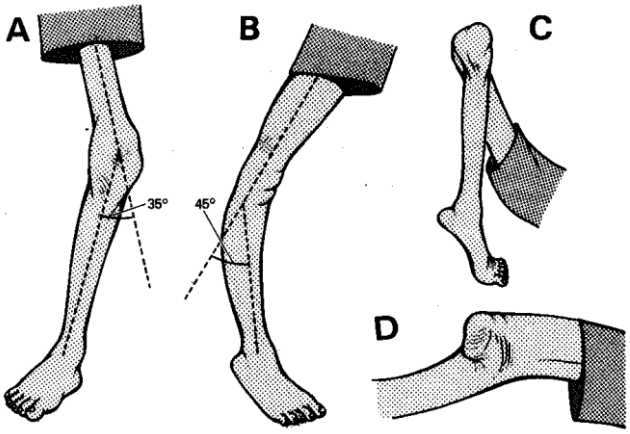


Fig. 32-11 MORE POLIO DEFORMITIES OF THE KNEE.
A, *valgus* deformity. B, hyperextension deformity (*genu recurvatum*). C, rotation deformity. D, posterior subluxation. All these are much less easily treated than flexion deformities.
Kindly contributed by Ronald Huckstep.

If there is a *valgus* deformity of the knee, usually associated with a flexion contracture, a surgical release and a calliper are necessary. If necessary, bend the calliper, or fit it with a *valgus* knee strap, to prevent it rubbing against the knee.

If there is lateral rotation of the tibia on the femur, or lateral subluxation of the knee, there is usually also a flexion contracture of the knee. Try to correct rotation and subluxation at the same time as the flexion contracture. More often, a late deformity is structural, and cannot be corrected by simple tenotomies. If rotation and subluxation are the only deformities, they are usually asymptomatic, and don't require specific treatment.

If a child has a hyperextended knee >10º (*genu recurvatum*), due to early weight-bearing on a weak knee, fit an above-knee calliper with a posterior strap.

If an adult has a hyperextended knee >30º, an osteotomy may be necessary.

(c) The ankle in chronic polio

If there is an *equinus* deformity of the ankle, owing to paralysis of its extensors, in a child, and flexing the knee allows you to bring the ankle up into the neutral position, correct the deformity by serial casting (32.1). If there is a greater degree of deformity than this, do a tenotomy.

In an adult, the decision as to whether an operation would be beneficial is difficult, and depends on:
(1) the degree of *equinus* of the ankle,
(2) the power in the knee and hip,
(3) the condition of the other leg,
(4) whether he can or cannot use crutches,
(5) whether he will need callipers after surgery and can get them.

If there is a *calcaneus* deformity of the ankle, due to weakness of the calf muscles, a lace-up boot may be all that is necessary. Otherwise, fit a below-knee calliper with a front stop.

If there is a *valgus* deformity of the ankle, usually associated with some degree of *equinus*, correcting the *equinus* deformity and fitting a calliper will probably be enough. Otherwise a transfer of the peroneal tendons, and perhaps a triple arthrodesis will be necessary (32-28D).

If there is a *varus* deformity of the ankle, due to weakness of the evertors of the foot, fit him with a below-knee calliper if the deformity is mild. Otherwise a soft tissue correction, or a triple arthrodesis will be necessary (32-28D).

If there is an adduction deformity of the forefoot, try several manipulations (32.1). Surgical correction will probably be necessary.

If there is a *cavus* foot (32-20A), a tenotomy, tendon transfer, or arthrodesis of the toes is necessary.

N.B. Apparent shortening is due to tilting of the pelvis, as the result of an adduction or abduction deformity of the hip. True shortening is a real shortening of the leg, and in polio is due to the failure of a paralyzed leg to grow. If necessary, correct an abduction contracture of the hip, a flexion contracture of the knee, or an *equinus* contracture of the ankle. If the shortening makes walking difficult (usually >4cm), raise the short leg with a clog or with boots. If necessary, fit callipers.

(d) Difficulties with chronic polio

If the femur or tibia fractures, fit a cast, and use the opportunity to correct any deformity, and maintain walking. The knee and ankle are unlikely to be functional, so stiffness will not be a problem. An internal fixation is indicated to maintain alignment.

(e) Appliances for polio

When you have released the contracture, the muscles of the leg will still be weak, so you will probably have to provide a brace, or a crutch, or both.

A weak hip needs crutches, a weak knee needs a long calliper (32-13A), and a weak ankle needs a short one. If you cannot provide callipers, *don't try to release the contractures!*

There are 4 types of orthopaedic appliances of increasing sophistication:
(1) Appliances of the traditional type, such as the pads, kneelers, sticks, peg legs (35-23B) and crutches that are used in traditional societies everywhere. Unfortunately, there are no traditional callipers.
(2) Appliances of the Huckstep type (32-13). These can be made in a hospital workshop using locally available iron, galvanized wire, wood, and leather, and can be repaired by a bicycle mechanic, a cobbler, or a blacksmith. A child will usually outgrow appliances properly made with hardwood, and need a larger size before they wear out. Those made of soft wood will wear out quickly.
(3) Appliances of an intermediate type are more durable. They are cheaper than appliances of type (4), and are technologically appropriate. An example is a modified Bata shoe with a metal tube to support the end of the iron bar (32-13B). If these shoes have an open toe, they will fit feet of various sizes, but are less durable in wet weather. Leprosy shoes (32-22) are of this kind.
(4) The expensive high-technology appliances that are standard in the industrial world. These need imported materials, particularly duralumin and special plastics, and can only be made and repaired by a skilled technician. Unfortunately, many prosthetists consider it a matter of professional pride to make only the most sophisticated appliances of this type, which patients cannot afford. Resist their efforts, and encourage them to make appropriate appliances in sufficient quantity.

If you cannot get ready-made appliances from an orthopaedic service, ask your hospital workshop to make those of types (2) or (3). All large or medium-sized hospitals, doing much surgery, need a workshop making a wide range of appliances of level (3). You will need above- and below-knee callipers, fitted when necessary with backstops or frontstops (32-13A). The callipers differ only in length, in the diameter of the ring, and in the presence of a knee piece in an above-knee calliper. Callipers of types (2) and (3) have irons each side of the leg. Although the single outside or inside irons of the callipers of type (4) look more elegant, they are weaker, more difficult to make and adjust, and usually less effective than double ones.

Use callipers to prevent deformities in a weak limb, and to straighten and support a child's leg after you have corrected the contracture. There are few indications for fitting callipers on an uncorrected contracture. Fit them as soon as walking starts, and replace them with a larger size with increasing growth. Encourage all children who have muscle weaknesses which might lead to deformities, to wear callipers until they have stopped growing, even if they can walk without them. The indications for fitting an adult with a crutch, or a calliper, are the same as in a child, except the deformities are static.

PARALLEL BARS FOR POLIO

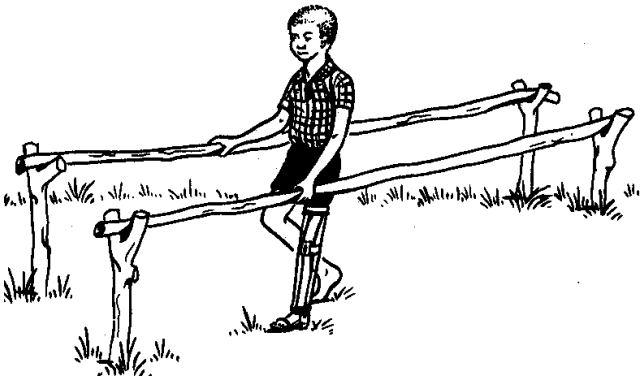


Fig. 32-12 PARALLEL BARS FOR POLIO are helpful when a child is learning to walk.
Kindly contributed by Ronald Huckstep.

(f) Above-knee calliper
If you are uncertain if a knee or ankle calliper is going to be useful or not, consider applying a suitable plaster cast of the same function for 4-6wks. If it is helpful, make a calliper. In this way, you will avoid making callipers that don't help.

INDICATIONS
Fit a child with an above-knee calliper if:
(1) The knee is so weak that lifting the leg against gravity is impossible (quadriceps power <3).
(2) He is likely to develop a contracture as the result of muscle imbalance.
(3) There is a mild knee contracture of <30º that a calliper could correct, if it is worn during the day or at night.
(4) He is developing a hyperextended knee, as the result of trying to lock and stand on it.
(5) There is weak quadriceps (35-18C), and at the same time too much *equinus* to let him swing the leg and lock the knee.

CONTRAINDICATIONS
Don't fit a calliper, or crutches, if:
(1) Walking is reasonably good with a flail ankle: walking may be easier without them.
(2) Walking is reasonably good with a weak knee, using the hamstrings (35-18C) to extend the thigh and lock the knee.
(3) There is enough power in the triceps, shoulders, or trunk to use crutches (needed because of weak hips).
(4) You have not corrected the deformity (unless it is very mild, and the calliper is designed to correct it).

FITTING
Choose a calliper which reaches about 2cm below the groin on standing, make the straps fairly tight, and make sure that the knee piece gives the knee adequate support anteriorly. Make the posterior strap slightly loose, unless the knee is abnormally hyperextended (32-13A).

If there is a mild flexion deformity of the knee, fit an ordinary calliper with a loose posterior strap, and a tight knee piece, which may need to be padded.

If the knee is hyperextended (*genu recurvatum*), you can correct this easily, so apply only slight tension to the posterior strap.

If there is a *valgus* knee, bend the calliper to avoid the bony points, and fit an inner knee pad tied to the outer side of the callipers, to prevent the knee from rubbing against the inside iron, especially on weight-bearing. Fit it so that it presses on the medial side of the knee, and corrects the deformity on walking.

CAUTION! A long calliper keeps a knee straight, and allows walking. But, because the knee does not bend, it may become fixed in extension, and be a nuisance on sitting. So make quite sure that on removing the calliper, the knee is put through a full range of passive flexion. This should not be a problem if the calliper comes off each night.

(g) Below-knee calliper
INDICATIONS
Provided there are no complications, fit a below-knee calliper if the foot is flail or drooping, or is tending to go into *varus* or *valgus*, provided the quadriceps power is ≥3. If it is <3, an above-knee calliper is necessary.

FITTING
Choose a calliper that will allow the knee to flex fully, with a socket which will fit firmly, and not allow too much movement. Always fit a supporting ankle strap.
If the calf muscles are so weak that the foot dorsiflexes excessively, fit a frontstop.
If there is little power in the dorsiflexors, so that the foot tends to *equinus*, fit a backstop (32-13).
If the ankle is inverted or everted, fit the appropriate inner or outer T-strap.

FOLLOW-UP
Try to review at least every 6 months. Replace the calliper with a larger one as the child grows. A long calliper is no use if it ends just above the knee! Make sure that the family understands that the child will need a calliper for life. Do all you can to help with education.

(h) Crutches
You will need a variety of sizes. If possible, make them to measure. If necessary, you can use any straight stick with a handle and a bar for the axilla. A crutch will be useful if polio has weakened the hips. Allow a suitable trial period. The grip must be strong enough to hold it, the triceps must be strong enough to propel the patient forwards, and the spine must be strong enough to allow sitting without help.

A crutch is likely to help if:
(1) Both legs are in callipers.
(2) One leg is in callipers, and the opposite leg or the spine is weak.
(3) One leg is in a calliper and is very weak, and the hip on the same side is weak.

Fit crutches. The length and the position of the hand grip must be right. Many patients who are given crutches could manage equally well with a stick. It you try a stick, teach him to hold it on the opposite side to the weak or weaker leg. If his hands are too weak to hold ordinary crutches, forearm crutches may be useful.

CAUTION! Make sure that he does not lean on the crutches while they are in the axilla. This may paralyze the radial nerve, or even all the nerves to the forearm and hand, and they may take 6 months to recover.

A TOE SPRING may be of great help if there is foot drop. Fix a suitable spring or a piece of bicycle tubing to the toe of the shoe and to a strap below the knee (32-16). Alternatively, attach a backstop, which is easier to fit.

AN ANKLE SPLINT will be useful if there is danger of foot drop while in bed. Make a suitable splint from plaster, or padded boards, to keep the foot at 90º to the leg.

(i) A raised shoe will help if one leg is >4cm shorter than the other. Any good cobbler can make a shoe raise. Start with a shoe raise of ½ the deficit in length. *If there is <4cm of shortening, don't supply a shoe raise*.

APPLIANCES FOR POLIO

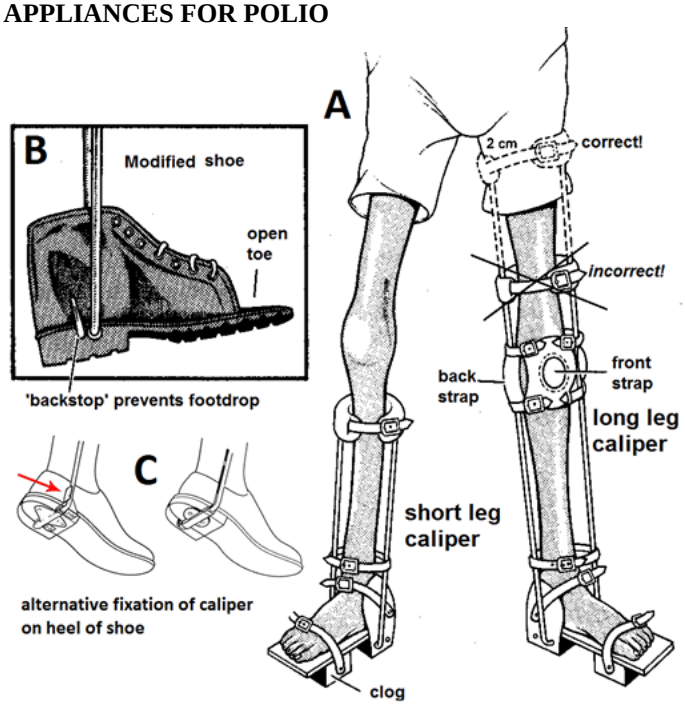


Fig. 32-13 APPLIANCES FOR POLIO.
A, simple callipers. *Note that long leg callipers should reach to 2cm below the groin.* They will be useless if they only reach to just above the knee. B, modified shoe, as an example of an appliance of type (3). C, alternative calliper fixation to the heel of the shoe.
Kindly contributed by Ronald Huckstep.

32.8 Hip and knee contractures

Before you decide on any operative treatment, assess the function of the limbs in detail (32.1), and what the particular social needs are. There may already be remarkable mobility, and although a straight leg may look better, it may not work better, especially if it needs callipers. However, if a child's legs are so weak and contracted that he crawls along the ground, even if it be at some speed, you must try to get him walking, because the psychological effect of doing so will be tremendous.

But if he is an adult, consider the whole future carefully first. He may be able to crawl fast and cultivate the fields on the hands and knees, but if he can only walk slowly and stiffly in callipers, he may die of starvation. So a cultivator may be better crawling, especially if his arms are too weak to use crutches, whereas an office worker, for example, may benefit from callipers.

N.B. Sometimes an operation is an obvious disservice, *e.g.* lengthening the Achilles tendon for someone walking on the ball of an *equinus* foot.

A severe contracture can:
(1) cause the skin over a joint to shrink,
(2) shorten the muscles, intermuscular septa, nerves, and vessels,
(3) contract the joint capsule,
(4) deform the epiphyses.
Undoing all this is difficult, and may be impossible; so only try to relieve milder contractures, and follow the indications carefully, or you may damage important structures, or cause infection or skin loss. A child's contractures are easier to correct than an adult's; he probably only needs a tenotomy, whereas an adult may need an osteotomy, or an arthrodesis.

Contractures of the hip and knee are often associated. You may have to release or lengthen:
(1) The iliotibial band in several places down the thigh. In a young child, 1-2 incisions may be enough, and you may not need the complete set of 4 incisions described (32-14).
(2) The tight structures on the front of the hip, particularly the *iliopsoas*.
(3) The tendon of the *biceps femoris* in the popliteal fossa.
(4) The medial hamstrings (occasionally).

You can cut tight bands and tendons in 2 ways:
(1) Push a long thin tenotomy knife through a small skin incision, and cut the bands by palpation. This is satisfactory for the less severe contractures, provided you do it correctly, and as extensively as necessary.
(2) Cut tight structures under direct vision. In the thigh, this is the best method, especially for severe contractures, but it needs more skill and the wound may break down, so use the closed method. Behind the knee, the common peroneal nerve is very superficial, so the only safe way to divide the tendons there is by open operation.

Lengthen a tendon by a Z-plasty incision. Slit the tendon longitudinally according to the length required, and extend this cut laterally proximally and distally in the form of a Z; then suture the two parts of the tendon together side-to-side to provide the extra length.

N.B. Don't try to treat a contracted hip by manipulation and serial casting in a spica; surgery is better.

The knee is the most difficult of the three joints on which you may have to operate, especially in an adult, whose tibia can be rotated backwards and laterally, as well as being flexed. Be safe, and *don't try to release a contracture of >90º*.

If you try, the tight popliteal vessels and nerves may be stretched, and pain, paralysis, and even gangrene may follow. Even a contracture of <90º may be difficult.

After you have released it as much as you can by tenotomy, you can obtain the final correction by daily increasing buckle correction. Insert a Steinmann pin through the upper tibia and incorporate this in a long leg cast, with a slit in the popliteal area. Fit a buckle ratchet attachment at the back of the knee. Apply traction to the Steinmann pin to avoid posterior subluxation of the knee, and adjust the buckle to give an extra 1mm extension per day (32-16).

ANATOMY. The common peroneal (lateral popliteal nerve) descends obliquely along the lateral side of the popliteal fossa to the head of the fibula, close to the medial margin of *biceps femoris*. It lies between the tendon of *biceps femoris* and the lateral head of *gastrocnemius*, and winds round the lateral surface of the neck of the fibula deep to *peroneus longus*.

(a) Tenotomy

INDICATIONS

(1) An isolated hip contracture of >30º at any age.
(2) A child with an isolated flexion contracture of the knee of >30º.
(3) An adult with an isolated flexion contracture of the knee of <90º.
(4) Combined hip and knee contractures of >30º, provided there are no contraindications.

If you have many patients, start by operating on the younger ones with lesser deformities first.

CONTRAINDICATIONS to any release operation are:

(1) Weak arms. The patient will need crutches, so he must have 2 arms, especially if both legs and the trunk are weak. There are exceptions to this rule, and a really determined adult, or child, sometimes manages surprisingly well with limited weakness in one or both arms, provided the trunk is strong.
(2) A patient with mild contractures, who is walking reasonably well, is probably best left as he is. This includes:
(i) a contracture of the hip alone of ≤30º, especially if it also has a mild abduction deformity, which may increase its stability and compensate for shortening.
(ii) an isolated knee contracture in a child: treat with manipulations under GA every 2wks.
(3) An adult who is earning his living as a 'crawler', and is happy to go on doing so.
(4) *Don't operate on any patient unless you can provide him with callipers* (32-13).

CAUTION! Scar tissue is not stable for at least 6 months, so when you correct contractures, you must find some way of maintaining the position of the limb for at least 6 months, or longer, if there is still muscle imbalance, or much scar tissue.

(1) ILIOTIBIAL BAND TENOTOMY (GRADE 1.3)

Use a narrow tenotomy knife, or an old cataract knife, or a #11 scalpel blade altered (32-17) so that only its tip is sharp.

Operate under full sterile precautions, and prepare both legs, even if you are only going to operate on one of them. Squeeze all blood out of the incisions periodically during the operation, and at the end. The hip incision may bleed considerably.

Release the hip and knee before you release the ankle. The structures you are going to divide must be tense, as you divide them, so keep the hip in as much extension and adduction as you can, while you cut. Feel the tight structures through the skin, to make sure that you have left no tight bands undivided.

CUTTING THE ILIOTIBIAL BAND

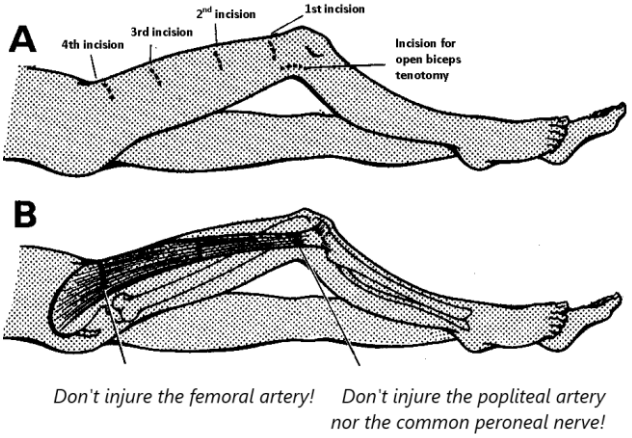


Fig. 32-14 ILIOTIBIAL BAND TENOTOMY.
A, position of the incisions. B, structures to be cut.
Kindly contributed by Ronald Huckstep.

1ST INCISION

Make this on the outer side of the thigh, about 2cm above the knee. You can usually feel the *tensor fascia lata* as a tight band. Push the knife horizontally into the outer side of the thigh, just behind the tight band, until it just touches the lateral side of the femur.

Rotate its blade, so that its cutting edge is upwards; then cut all the subcutaneous structures anterior to the blade, and lateral to the femur. Provided you make the incision in the right place, and only cut anteriorly, you will not cut anything important. *Don't cut posteriorly*, or you may cut the popliteal artery or the lateral popliteal nerve.

2ND AND 3RD INCISIONS

Make these ⅓ and ⅔ of the way down the outer side of the thigh. Feel for the *tensor fascia lata*, and push the knife in along its posterior border down to the bone, exactly as for the first incision.

Then rotate the knife 90º and cut anteriorly and laterally to the outer side of the shaft of the femur.

If you cannot feel the *tensor fascia lata*, insert the knife where you think it should be and cut exactly the same way. There are other tight structures to be cut, including the *vastus lateralis*.

The 4TH INCISION is the one which releases the hip. Make it 2cm below the anterior superior iliac spine.

CAUTION!

(1) *Don't damage the femoral vessels and nerve. Don't push the knife further medially than a point 2 cm lateral to the mid-inguinal point* (where you can feel the artery).

(2) Feel for the inguinal ligament, and take care not to divide that.

Push the knife in subcutaneously, below the anterior superior iliac spine, from a lateral to a medial direction, so that its flat surface is in the plane of the skin just caudal to the anterior superior iliac spine. Stop 2cm lateral to the mid-inguinal point. Then rotate it 90º, so that its edge faces backwards, and cut all the tight subcutaneous structures.

If the contracture is severe, cut all tight structures lateral to the branches of the femoral nerve. Cut right down to the front of the trochanter of the femur.
When you have cut the tight structures anteriorly, twist the knife so that it cuts laterally, and cut all the tight structures on the anterolateral side of the hip.

CAUTION!
(1) When the tip of the knife is deep, angle it caudally, so that its blade is parallel with the inguinal ligament, and will not cut it.
(2) *Don't cut further back than the coronal plane of the anterior part of the hip joint.* Leave the abductors posterior to this, to give lateral stability to the hip.
(3) Keep the hip in as much adduction and extension as you can, while you divide the tight structures. Feel them through the skin during the operation, and *don't leave any tight deforming bands behind*.

(2) OPEN *BICEPS FEMORIS* TENOTOMY
(GRADE 1.3)

INDICATIONS. This is only indicated if the knee contracture is >30º but <90º. The method which follows is a very limited open tenotomy suitable for a patient:
(1) who needs a bit more extension, so that he can be put into skin traction,
(2) whose *biceps femoris* tendon is tight, but not the *semitendinosus* and *semimembranosus* tendons, which are attached medially. Feel the tendons when the knee is extended to its limit. If all the tendons are tight and need surgical release, get expert help.

OPEN *BICEPS FEMORIS* TENOTOMY

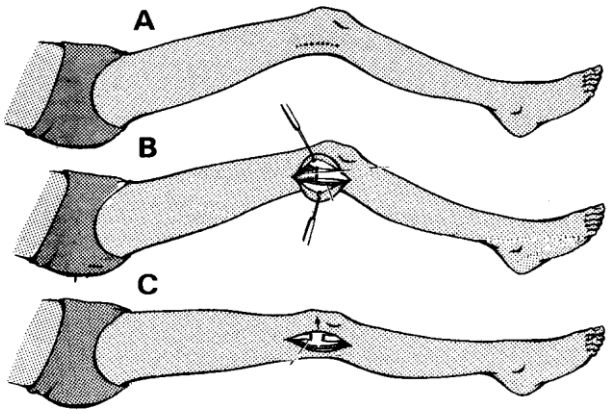


Fig. 32-15 OPEN *BICEPS* TENOTOMY to release a contracture of the knee.
A, site of the incision. B, be quite sure you find the lateral popliteal nerve, before you cut anything. C, divide the *biceps femoris* tendon.
Kindly contributed by Ronald Huckstep.

METHOD. Make an incision on the lateral side of the knee (32-15). Feel for and find the *biceps* tendon under direct vision, hook it out of the wound, and cut it.

CAUTION! Be sure that it is the *biceps* tendon, and only the *biceps* tendon. *Be careful you have not caught the lateral popliteal nerve with it*: they both look very similar.

Look for muscle fibres being inserted into the tendon before you cut it. *Be sure the nerve has not stuck to the back of the tendon*.

Put your finger into the wound, and feel for any other tight structures which need cutting. You may need to cut the posterior part of the iliotibial band, and the lateral intermuscular septum. Sometimes, the anterior part of the deep fascia lata also needs cutting.

Apply skin traction, or a well-padded cast.

(b) Casting and manipulation. If the contracture of the knee is <45º, apply a well-padded cast with the knee just short of the full extension to which it is capable. It must not be under any tension, or it will be painful. If the contracture is >45º, apply a pulley support keeping the knee elevated, with skin traction to the distal leg (Russell traction).

CAUTION! NEVER put a knee into a cast under tension, or wedge a cast to correct a knee contracture, or its articular cartilage may necrose, and early painful osteoarthritis may follow, in what was previously a painless mobile joint.

Check the hip incision again (if you have released the hip at the same time), as soon as you have applied the cast, and squeeze out any clot which has formed, under full sterile precautions. Pad the incision and apply light adhesive strapping.

Every 2wks, remove the cast, and manipulate the hip and knee, until the knee is in at least 5º of hyperextension, and there is <10º of flexion deformity ('fixed flexion') in the hip. Manipulate it (32.1), and spend 5-10mins on each joint. Be sure to flex the knee fully, and to rotate the tibia medially and laterally, to maintain these very important movements. Correct or avoid backward subluxation of the knee. If necessary, correct the lateral rotation of the tibia on the femur.

After each manipulation, apply a well-padded above-knee cast, with the ankle firmly dorsiflexed. As always, *don't put the knee under tension!*

As soon as the flexion deformity in the knee is <40º, fix a walking piece on the bottom of the cast, and encourage walking with crutches.

Leave the final cast on for 2wks, and then replace it with an above-knee calliper, with its posterior strap loose. Advise wearing it day and night for 2-3wks, until the risk of recurrence of a flexion contracture of the knee is less.

Later, daytime wear only, for up to 6 months, is necessary when the tendency for the contracture to recur will have passed, or indefinitely if the knee needs stabilization.

If possible, provide physiotherapy, or assisted exercises, after you have removed the cast. If the post-operative care is not properly done, you may end up with a stiff, painful knee, in which the tibia is subluxated posteriorly on the femur.

MOBILIZE and get the patient into a chair as soon as possible. For a child, you can usually do this in a few days. In an adult, full mobilization may take a month or two. Prop him up gradually in bed or a chair, before he tries to get up. He may need crutches or a calliper; crutches must fit properly.

(c) Difficulties releasing contractures of the knee
If the knee is painful and stiff (which, so it is said, should never happen with proper treatment), reassure that it will slowly recover some, or all, of its movement in a few weeks or months. The pain will probably go, even if the knee does not regain its full movement. If pain and stiffness persist, try intensive physiotherapy or Russell traction. An arthrodesis is necessary very occasionally.

BUCKLE KNEE EXTENSION DEVICE

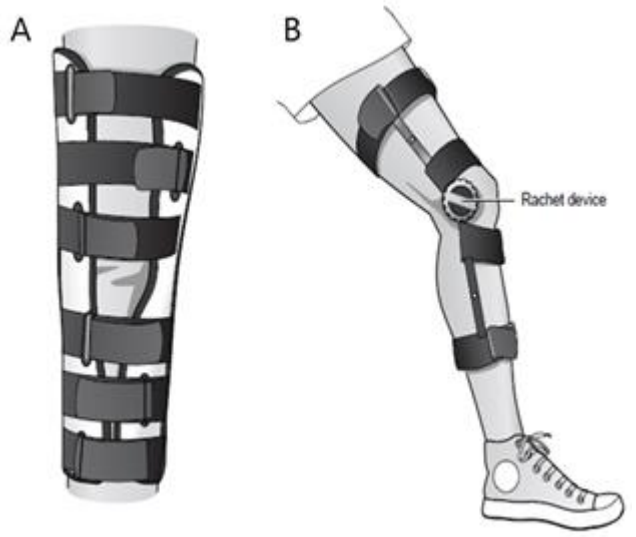


Fig. 32-16 BUCKLE CORRECTION FOR KNEE FLEXION DEFORMITY.
A, fixed splint. B, adjustable splint.

32.9 *Equinus* ankle deformity

This is the most common deformity in a child, and is fortunately the easiest to correct. Provided there is no severe *valgus* or *varus* deformity (32.1), you can correct a milder *equinus* deformity with serial casts, each of which will release the deformity a little more. If you are inexperienced, you will find serial casts very useful.

If a child has a more severe deformity, he needs the Achilles tendon lengthened. You can perform this by an open or closed method. The closed operation is simpler, and there is less risk of infection or keloid formation. The advantage of the open method is that it is possible to divide the posterior capsule of the ankle joint if this is necessary, as it may be in polio. The risk in both methods is that you may cut the posterior tibial nerve and vessels, and cause gangrene of the toes, but this should never happen if you are careful.

(a) Serial casting
INDICATIONS
Mild degrees of *equinus* deformity: make sure that when you flex the knee as far as possible, the calf muscles relax enough so that the ankle comes into a neutral position (90º).
If both legs are involved, correct them one at a time, to avoid a long confinement to bed.

METHOD
Don't use GA. Apply a below-knee cast, while the knee is flexed to 90º, to allow you to achieve more dorsiflexion of the ankle. You may not be able to get the foot into the neutral position on the first occasion. When the cast is dry, apply a walking heel, and allow mobilization. Encourage progressive knee extension. Once the patient is walking with the knee fully extended, repeat the procedure and apply a further cast, until the foot will reach the neutral position with the leg extended.

(b) Closed Achilles tenotomy (GRADE 1.3)
INDICATIONS
Where serial casting is inadequate, with also a minor *varus* or *valgus* deformity, provided it is not so severe that it will prevent you fitting callipers.

CONTRAINDICATIONS
In a child, tenotomy is contraindicated if:
(1) The *equinus* ankle is helping to compensate for a short leg, or to stabilize an unstable knee, and so enabling satisfactory walking.
(2) He will never walk because his arms are weak.
(3) The deformity is minimal, and he is managing well with a shoe or boot, with or without a calliper.
(4) The feet are infected; if so, delay the operation.
(5) There is a severe *valgus* or *varus* deformity which will make fitting a calliper impossible.

METHOD
N.B. For club foot, use the method described in 32.10.

Use a small tenotomy knife, or the improvised one (32-17). Use full sterile precautions, scrub up, gown yourself, use gloves, and apply a tourniquet (3.4).

1ST INCISION: cut through the posterior ⅔ of the Achilles tendon, above the level of the malleoli, by pushing the knife into the tendon from the medial side, in the line of its fibres, at the junction of the anterior ⅓ and the posterior ⅔. Rotate the knife 90º, and then cut posteriorly, until you feel the knife cutting very easily, which shows that you have now cut the posterior part of the tendon.

2ND INCISION: push the knife into the tendon in the line of its fibres, at the junction of its lateral ⅓ and medial ⅔,

just above its insertion on the calcaneum. Then, rotate the knife through 90º, and cut medially.

CAUTION!
(1) Use full sterile precautions, and drape with sterile towels.
(2) Use a gentle sawing motion, and *don't break the blade.*
(3) *Don't cut the posterior tibial vessels and nerves*, which lie anteromedial to the Achilles tendon (32-18).
(4) *Don't try to divide the tight posterior capsule of the ankle joint in this method*. This is not tightened in poliomyelitis, unless there is an associated *varus* deformity, which must be corrected at open operation.

To manipulate the ankle, flex it dorsally. Apply force as close to the joint as possible. *Don't apply force to the distal tibia*; you can easily break it. In a young child with polio, you should be able to achieve 20º of dorsiflexion (calcaneus); in an adult or older child, you may get less. If necessary, manipulate it again 2wks later.

CLOSED ACHILLES TENOTOMY

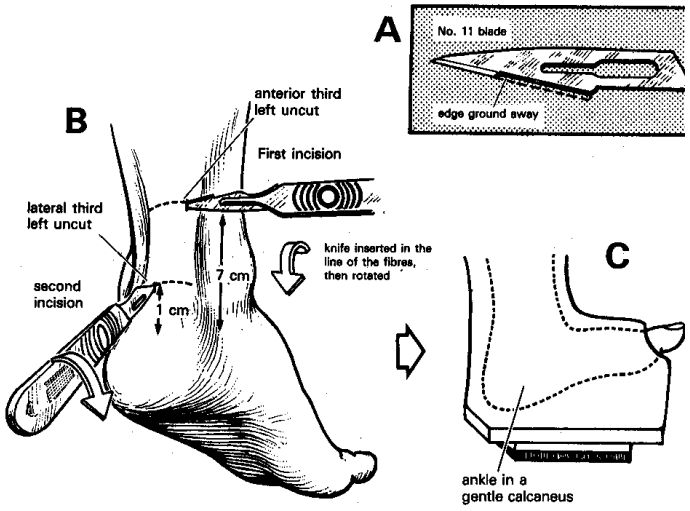


Fig. 32-17 CLOSED ACHILLES TENOTOMY.
A, if you don't have a tenotomy knife, make one by grinding off the proximal part of a #11 blade. B, the 2 incisions: make only tiny incisions in the skin. Insert the knife with its blade in the plane of the fibres, and then twist it before you cut. Make the 1st cut from the medial side above the malleolus, and the 2nd posteriorly cutting medially. C, put the foot into a cast in gentle dorsiflexion. *N.B. The fibres of the Achilles tendon twist from medial to lateral as they descend.*
Kindly contributed by John Stewart.

If the ankle does not reach the neutral position (90º), check that the tendon has been divided properly, by reinserting the tenotomy knife in the same 2 tenotomy sites. If the ankle is still not fully corrected to 90º, wait and plan to increase correction by applying serial casts every 2-3wks.

N.B. If necessary, release the ankle on the same occasion as the knee and the hip.

POST-OPERATIVELY, squeeze out all subcutaneous clot. Bleeding is usually slight. Apply a small dressing.

If the knee is stable, apply a well-padded below-knee walking cast, with the foot near the maximum correction, but not at the extreme limit of extension. Elevate the leg, and encourage walking. Review a young child in 3wks, and an older child or adult in 6wks. Remove the cast and apply a below-knee calliper with a backstop (32-13A).

If the knee is unstable and has no contracture, apply an above-knee cast, and later an above-knee calliper instead of a below-knee.

DIFFICULTIES WITH CLOSED TENOTOMY
If you have cut the whole Achilles tendon, *don't be alarmed*. It will almost always heal satisfactorily in the lengthened position. *Don't try to repair it at this stage.*

If the deformity recurs, it probably did so because the patient did not wear a calliper, or wore one without a backstop (32-13A). *If he fails to wear one initially, the deformity is sure to recur*. He may be able to do without a calliper 6-12 months later. Follow up carefully, so that you can decide about this.

If you fracture the lower tibia because you have manipulated it too vigorously, fit a cast.

(c) Open Achilles tenotomy (GRADE 1.3)
INDICATIONS
Equinus contractures of the ankle in which there is a contracture of the posterior capsule of the ankle joint that requires release. This is an alternative to the closed method, especially when that has not achieved normal dorsiflexion.

METHOD
Using GA, apply an exsanguinating tourniquet to the thigh (3.4). Use the lateral position, with the leg to be operated on uppermost.

Make a longitudinal incision over the lower ⅓ of the leg, extending proximally from the attachment of the Achilles tendon to the calcaneus. Dissect out the Achilles tendon. You may see the small tendon of the *plantaris* (35-22I) on its anteromedial side.

Make 2 incisions halfway across the Achilles tendon: the lower one 1cm above its insertion, either from the medial to the lateral side, or vice versa. If there is any *varus* deformity of the foot, leave the lateral side intact. If there is any *valgus* deformity, leave the medial side intact.

The aim of doing this is to help the distal attachment of the tendon to correct the deformity. At a suitable level, c.4-10cm proximally (depending on the size and the degree of plantar flexion to be corrected), make a small incision opposite the first one (32-18A,B). Then push up the foot into normal dorsiflexion, without too much force: if the fibres of the Achilles tendon pull out, stop there.

If you have divided the Achilles tendon completely, bring its ends together with a special figure-of-8 suture. Close the skin with 2/0 monofilament. Pad the leg, apply a below-knee cast with the knee flexed to 90º and release the tourniquet.

If you fail to put the foot into satisfactory dorsiflexion, make a longitudinal incision down the middle of the tendon joining the two cuts. If this still does not correct the position of the foot, dissect down to the posterior aspect of the ankle joint, under direct vision. Divide the posterior capsule of the ankle joint transversely (32-18D), from lateral to medial, and open up the ankle, by dorsiflexing the posterior part of the foot.

CAUTION! Be careful not to cut:
(1) the *flexor hallucis longus* tendon (35-22I),
(2) the posterior tibial nerve and vessels, which lie on the medial side of the posterior aspect of the ankle joint.
These structures are only in danger if you divide the posterior capsule.

OPEN ACHILLES TENOTOMY

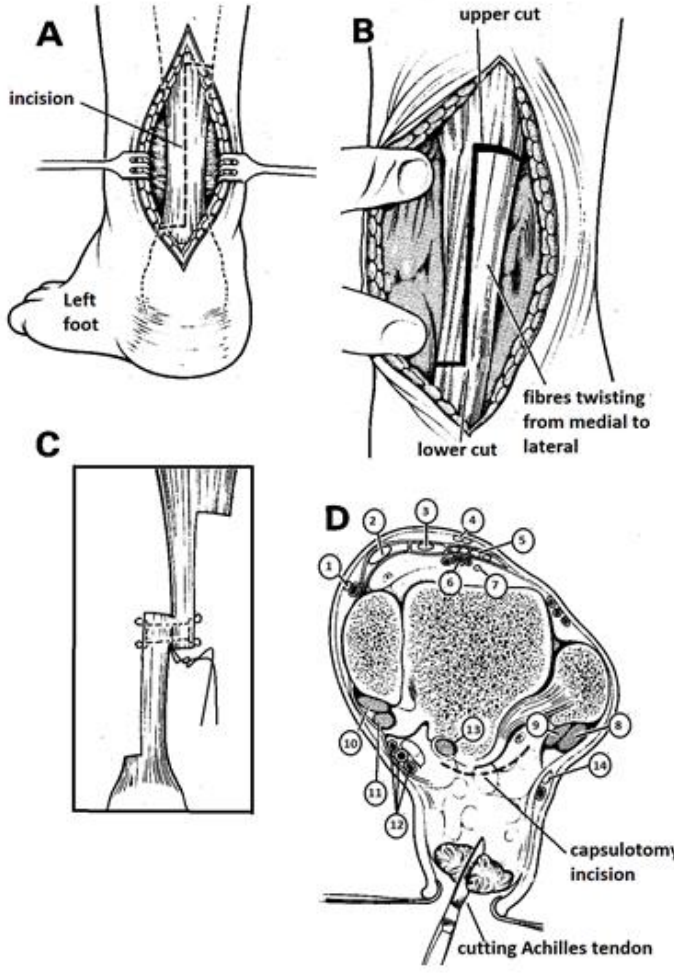


Fig. 32-18 OPEN ACHILLES TENOTOMY.
A, expose the Achilles tendon. B, divide the tendon, initially at the lower level, either on the medial side if there is *varus* deformity, or lateral if *valgus*; then vertically if necessary. C, repairing the tendon with a variation of the figure-of-8 suture. D, cross-section at level of mid-malleoli: (1) saphenous nerve and vein. (2) *tibialis anterior*. (3) *extensor hallucis longus*. (4) superficial peroneal nerve. (5) *extensor digitorum longus* and *peroneus tertius* in the inferior extensor retinaculum. (6) dorsalis pedis vessels. (7) deep peroneal nerve. (8) *peroneus longus*. (9) *peroneus brevis*. (10) *tibialis posterior*. (11) *flexor digitorum longus*. (12) posterior tibial artery and vein and tibial nerve. (13) *flexor hallucis longus*. (14) sural nerve. *N.B. The fibres of the tendon twist from medial to lateral as they descend.*

POST-OPERATIVELY, raise the foot of the bed, expose the leg on a pillow, and check the circulation in the toes hourly. Next day, add a 'shoe' to the cast. Provide crutches, and check the cast in 3wks. Remove it or change it at 6wks.

REHABILITATION is essential for chidren. Imaginative play areas are crucial.

REHABILITATION

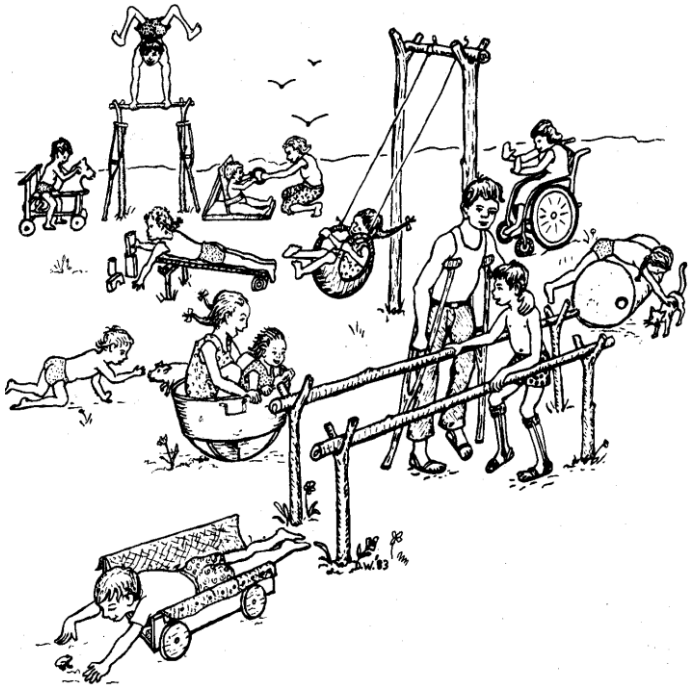


Fig. 32-19 REHABILITATING DISABLED CHILDREN.
With the kind permission of David Werner.

32.10 Club foot (Neonatal *talipes equinovarus*)

A child is sometimes born with shortening of the soft tissues of the flexor aspect of the leg, and the medial side of the foot, which causes the talus to point downwards (*equinus*) and inwards (*varus*). At the same time, the forefoot is adducted at its tarso-metatarsal joints, and the 1st metatarsal is plantarflexed to a greater degree than the 5th metatarsal (*cavus*). This may happen to one or both of the feet and is known as idiopathic club foot. The deformity is common and may arise in 1 in 500 live births.

If the deformity is left to progress without correction, the navicular bone may be pulled medially, and sometimes even away from the front of the talus. An extreme result is a plantarflexed calcaneus and vertical talus, with dislocation of the navicular causing a 'rocker bottom foot'.

Manipulation and casting by the Ponseti method when properly done will correct this disability permanently. It is best if you can organize for your physiotherapy team to attend a course to learn to do this correctly; although the Ponseti method is not difficult, it is important to understand the anatomy, and avoid the pitfalls.

Occasionally, a child is born with the feet pointing in other directions or with other deformities: these may result in secondary, as opposed to idiopathic, club foot. Some cases of *talipes* are due to paralysis, for example those associated with MENINGOMYELOCOELE (33.10). These may be helped but are often resistant to conservative manipulation treatment.

However, the feet of a child with **arthrogryposis** can be corrected by the Ponseti method, though recurrence is a problem. You need to use the Ponseti technique properly and carefully, but it has probably the *best cost-benefit ratio* of any surgical procedure. It is most effective if you start before 9 months of age, but may still correct 85% of deformities if you begin even up to 12yrs.

(a) The Ponseti method

You have the best chance of correcting club feet permanently, so that a child will be able to walk normally in normal shoes, if you start treatment in the first days after birth. Manipulation weekly and applying plaster casts for 5-6wks is normally successful. A tenotomy completes the correction, which is maintained with a foot abduction brace.

You should avoid other operations which tend to produce scarring and a chronically painful foot, until at least 2yrs. You should aim to correct the *cavus* and *adductus* components of the clubfoot simultaneously, and *varus* when the entire foot is fully abducted. Lastly correct *equinus.*

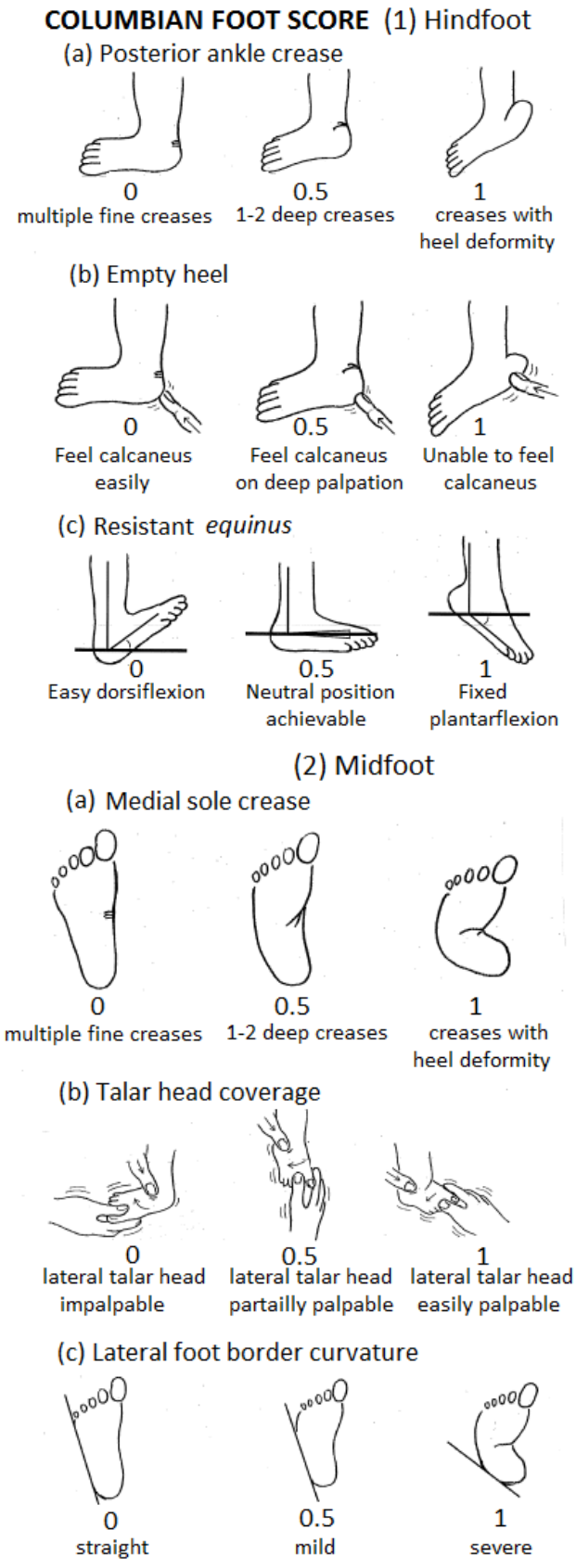
N.B. Distinguish between inversion and eversion at the ankle, and pronation and supination at the forefoot!

You should record the deformity objectively using the COLUMBIAN (Pirani) CLUBFOOT SCORE, giving 0 for no deformity, 0·5 mild deformity and 1 severe deformity:

	(a)	**(b)**	**(c)**
Hindfoot	Posterior ankle crease	Empty heel	Resistant *equinus*
Midfoot	Medial sole crease	Talar head coverage	Lateral foot border curvature

So, a normal foot scores 0 and a severe deformity 6.

N.B. The 'adducted metatarsus' has only a midfoot and no hindfoot contracture and is not a club foot.



PONSETI METHOD FOR CORRECTING CLUB FOOT

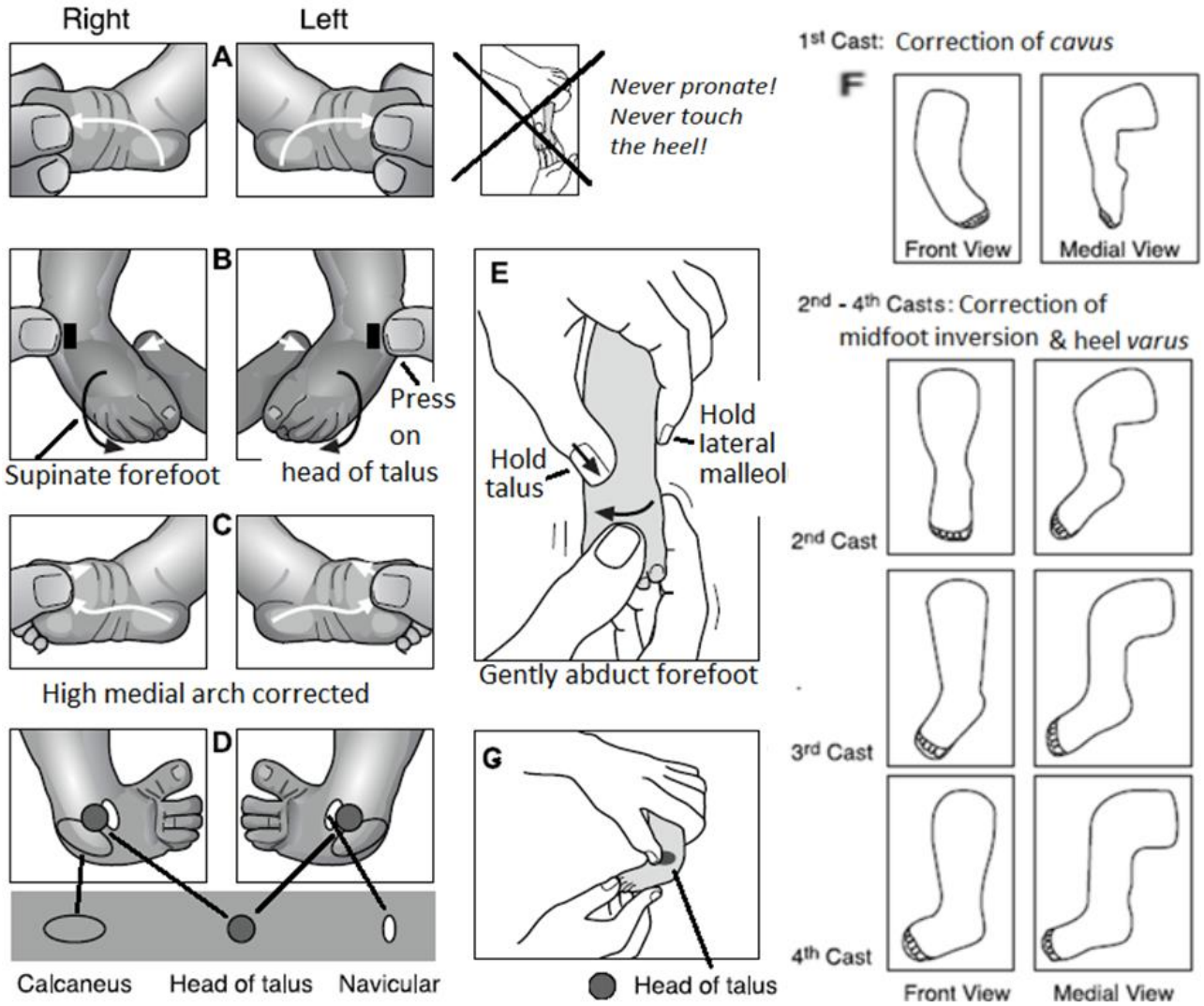


Fig. 32-20 CORRECTING CLUB FOOT.
Get the forefoot in line with the midfoot in terms of supination. A, the arrows show *cavus*, the high medial arch, due to pronation of the forefoot in relation to the hindfoot. B, correct *cavus* by supinating the forefoot with pressure against the head of the talus. C, *cavus* (the forefoot adducted at its tarso-metatarsal joints) corrected. D, locate the head of the talus. E, correct *adductus* by gently abducting the forefoot whilst stabilizing the talus with your thumb and holding the lateral malleolus with your index finger. F, appearance of casts and the foot at each stage. G, apply padding while holding the maximum corrected position.

Correct *cavus* by supinating the forefoot and making a normal longitudinal arch (32-20A, 32-26J) and correct *adductus* by abducting the forefoot in proper alignment with the hindfoot. With the arch well moulded and the foot in slight supination, gently and gradually abduct the entire foot under the talus (32-26J) securing it against rotation in the ankle mortice by applying counter-pressure with the thumb against the lateral part of the talus head, using this as the pivot or fulcrum (32-20B,C), *not on the cuboid*.

So, make sure you can locate the head of the talus by first feeling for the lateral malleolus, and moving your thumb forward in front of the ankle mortice. The navicular (32-26J) is displaced medially to a position in front of the head of the talus, almost touching the medial malleolus. The anterior part of the calcaneus is beneath the head of the talus (32-20D). *Don't touch the heel at this stage.*

Gently abduct the forefoot, whilst stabilizing the head of the talus and holding the lateral malleolus, as far as you can without causing discomfort to the child. Hold this position with gentle pressure for 1min (32-20E) so that the big toe is almost straight, and apply a cast for 1wk. Continue further abduction, holding the position in the 2nd and 3rd casts, each for 1wk. Correct heel *varus* when you have corrected *adductus*, keeping the position in the 3rd and 4th casts (32-20F), again for 1wk.

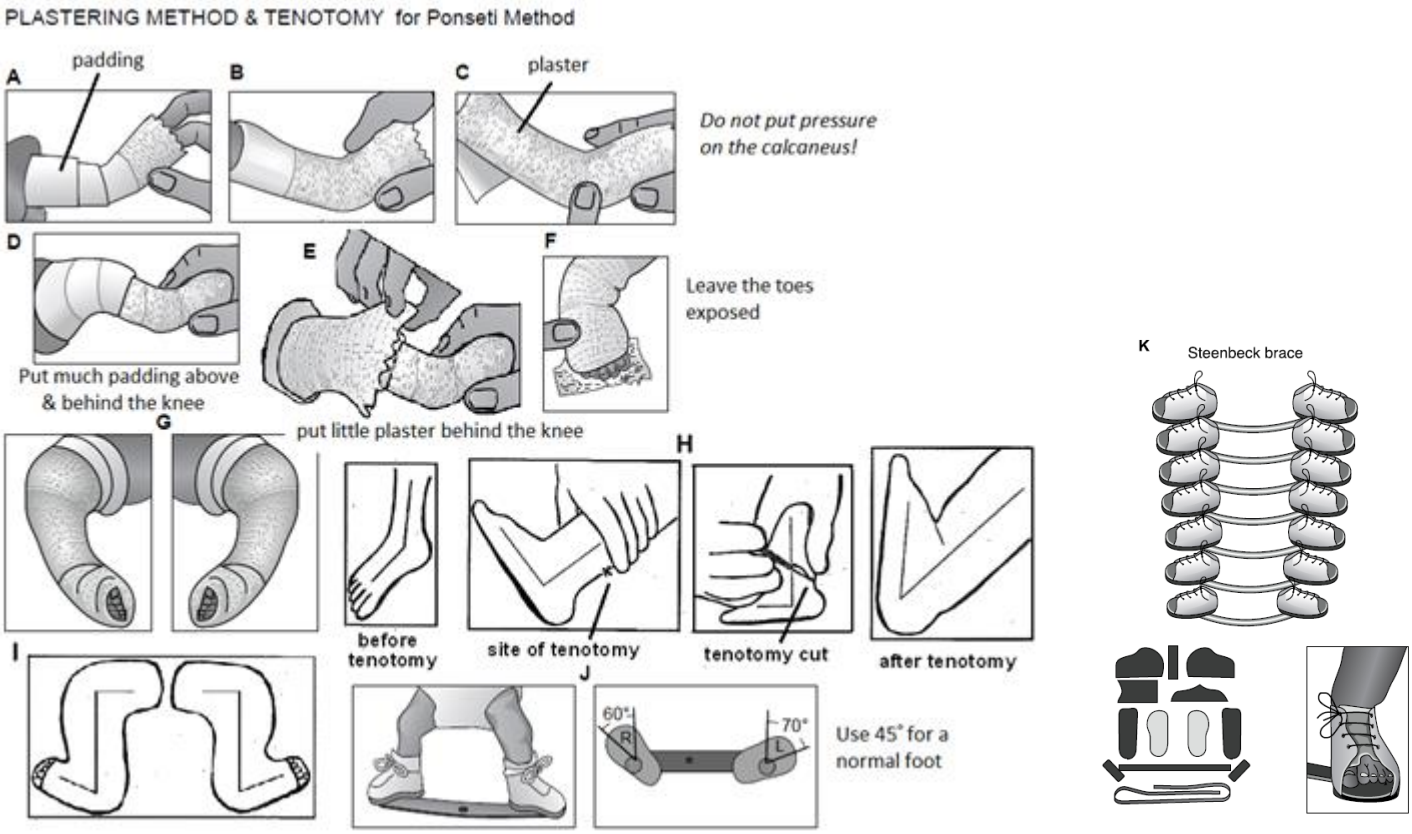


Fig. 32-21 PLASTERING TECHNIQUE AND TENOTOMY FOR THE PONSETI METHOD.
A, apply plaster in 3-4 turns first around the toes. B, continue up the leg applying some tension above the heel. C, mould the head of the talus *but do not apply pressure on the calcaneus.* D, apply copious padding above the knee, but E, little plaster behind it. F, leave the toes exposed removing plaster to the mtp joints dorsally, but leaving the plantar side as a support. G, final appearance. H, infiltrate 0·5ml LA 1cm above the *calcaneus*, and make a tenotomy here. I, apply a 5th cast with the foot abducted 60-70° with respect to the front of the tibia. Use 45° for a normal foot. *Never pronate the foot.* J, degree of abduction with the brace with different angles. K, the Steenbeek brace: different-sized boots, the materials needed to make the boot, and the final result. Use this to prevent the deformity from recurring.

Apply well-moulded, long-leg plaster casts after completion of each manipulation. The purpose of the casting is to immobilize the contracted ligaments at the maximum stretch obtained after each manipulation. Apply padding whilst maintaining the maximum corrected position (32-20G). Apply the cast with plaster of Paris in 3-4 turns first around the toes (32-21A), and continue up the leg, adding a little tension above the heel (32-21B). Keep some space around the toes by wrapping the cast around your assistant's holding fingers (32-21A)!

N.B. Don't force the correction with the plaster, and *don't press continuously on the head of the talus,* but rather, mould the plaster over the head of the talus and under the arch to avoid flatfoot. *Don't put pressure or try to mould the calcaneus* (32-21C).

Continue plastering above the knee using copious padding (32-21D) but *avoid large amounts of plaster behind the knee* (32-21E). Trim the plaster dorsally up to the mtp joints, leaving the plantar surface intact to support the toes (32-21F). Remove each cast just before you are ready to apply a new one. Soak the plaster in water for 20mins before removal. Start removing it at the thigh. *Do not allow a long interval between re-casting* because you may lose considerable correction thereby.

Finally, correct *equinus* by dorsiflexing the foot. You will usually need to do a simple percutaneous tenotomy of the Achilles tendon, unless the Pirani score is <1 for hindfoot and midfoot deformity and the talar head is easily palpable.

Don't perform a tenotomy if the heel is in varus, because this means you have not achieved adequate correction!

ACHILLES TENDON TENOTOMY (GRADE 1.3)
Place the child in a supine position with an assistant holding the pelvis in one hand and the leg in the other. Flex the knee slightly, and abduct and externally rotate the hip so that the heel faces the surgeon. Palpate the Achilles tendon and infiltrate 0.5m LA (32-21H) with an insulin syringe after cleansing the skin (*don't use too much LA* as you will then find the Achilles tendon difficult to palpate). Dorsiflex the foot to stress the Achilles tendon. Insert a 22G needle medial to the tendon, and by multiple cuts, divide the tendon across 1cm above the calcaneus; you will feel a sudden 'pop' which will allow 10-15° extra dorsiflexion. Apply a 5th cast with the forefoot abducted 60-70° with respect to the front of the tibia (32-21I). *Never pronate the foot.* Keep this cast in place for 3wks.

When you remove the cast, 30º of dorsiflexion should be possible in a well-corrected foot. The tenotomy scar is minute. Now apply an abduction brace for 23h/day at 3 months (*i.e. all the time except when bathing*). You may have to adjust this brace as the child grows, and should review him monthly. Make sure the brace is fitted to open-toe high-ankle straight-laced shoes, with 75º external rotation and 10-15º dorsiflexion for the affected foot, and 45º for the normal foot (32-21J).

The bar is slightly curved convexly forwards. The knees are free so that the child can stretch the *gastrosoleus* tendon, and the bend in the brace helps to stretch the *gastrocnemius* (35-20B) and Achilles tendon. You can get a skilled cobbler to make the Steenbeek brace (32-21K) with readily obtained materials.

If the deformity recurs after bracing, you need to start again with serial casting, with possibly another Achilles tenotomy. Then maintain outward rotation in open shoes attached to a bar 12h at night and 2-4h in the middle of the day for 3-4yrs to prevent recurrence. The results, although anatomically not always perfect, are almost perfect functionally till late adult life. Bracing is essential: *if you don't follow the programme diligently, you will get an 80% recurrence rate*. Teach parents how to put on and take off the brace, and encourage the child to move both knees simultaneously. There should be no 'negotiations' about wearing the brace with the child. Compliance is as necessary as with TB treatment.

To prevent the child kicking the shoe off, make sure the laces are well tied. Review the child at 2wks, 12wks, then every 4 months till age 3, every 6 months till age 4, then every year till skeletal maturity.

(b) Difficulties with the Ponseti method

Avoid these errors:
(1) *Don't pronate or evert the foot* because this increases the *cavus* and does nothing to unlock the calcaneus locked under the talus, and will result in a 'bean-shaped' foot.
(2) *Don't abduct the foot at the mid-tarsal joints by pressing on the cuboid with the thumb*, because this will make correction of *varus* of the heel impossible.
(3) *Don't externally rotate the foot while the calcaneus remains in* varus, because this produces posterior displacement of the lateral malleolus.
(4) *Don't forget to immobilize the foot* after each manipulation, with ligaments at maximal stretch.
(5) *Don't apply below-knee casts*, because these don't hold the forefoot abducted and tend to slip.
(6) *Don't correct* equinus *before correcting heel* varus *and supination*, because this results in a 'rocker-bottom' deformity.
(7) *Don't perform an incomplete tenotomy*, because it will not give enough release and the tendon anyway heals rapidly in infants.
(8) *Don't attempt to obtain a perfect anatomical correction*, because it is a functional correction that you want! *The radiographic appearances have no correlation with long-term function!*

If there is an *adductus* or *varus* relapse, recognized by supination of the forefoot (with the child walking towards you), and heel *varus* (with the child walking away), go back to manipulating and casting as from infancy.

If there is an *equinus* relapse at 1-2yrs, apply casts to get the calcaneus at least into a neutral position. You may have to repeat an Achilles tenotomy and follow this by 4wks of foot abduction in a long-leg flexed knee cast. Then go back to using the brace at night. If there is a late relapse at 3-5yrs, check if the foot dorsiflexes to 10º and perform a tenotomy as before. Otherwise more complex surgery is necessary.

If there is persistent *varus* and supination during walking, usually because of non-compliance, characterized by thickened lateral plantar skin, correct any fixed deformity with 2-3 casts and then refer for a *tibialis anterior* transfer. It is best to do this between 3-5yrs of age, but *always* after ossification of the lateral cuneiform (usually at 2½yrs).

If no treatment has been started at all, you should start the Ponseti method as for a newborn, but results are not as good, and depend on the delay starting treatment and the severity of the deformity.

If other non-surgical treatment has already been started elsewhere before 28 months, you should start the Ponseti method as for a newborn: results are just as good.

If treatment fails, check for a neurological cause; the options are a talectomy (Lambrinudi procedure), further casts, lengthening the Achilles tendon (32-18) or a *tibialis anterior* transfer operation.

32.11 Neuropathic foot care

The feet are often just as important as the hands. Someone is able to work with a paralyzed hand, but if he cannot walk, he will probably be unable to undertake the essential activities of daily life unaided. Many diabetics who are being adequately treated medically are being allowed to walk about on ulcerated feet. The dressings that cover their ulcers don't prevent them from deepening, and widening, and involving the bones underneath. The quiet progressive destruction of these feet is not inevitable, and can be minimized. It may be a losing game, so play it as cleverly as you can, and try to retain the usefulness of the foot as long as possible.

Ulcers can be caused by:
(1) Constant mild pressure, which causes necrosis by impairing the blood supply to the tissues, as in neuropathic ulcers. In a normal person ischaemia soon causes pain, so that the ischaemic part is moved, and its blood supply restored. In an insensitive foot there is diminished pain sensation (though some sensation to touch remains), so that the ischaemic tissue is allowed to become necrotic

and ulcerates. Also, *an unnoticed fracture will produce deformity because the bone fragments are not immobilized*.
(2) A strong force which cuts, shears or tears the tissues. In the foot, the strength of the force is less important than the small area over which it is applied.
(3) The frequent repetition of moderate forces, which causes inflammation and so weakens the tissues. This is an important cause of ulcers, so try to minimize the pressure on a foot.
(4) Forces which spread infection to soft tissues and bone. An infected foot is so painful to a normal person that it must be rested: *a patient with a neuropathy does not do this spontaneously.*
(5) A previous ulcer. This is the commonest cause. If a patient has never had an ulcer, he may escape without one, if he is careful. If however he has already had many ulcers, he will probably not notice getting one more.

The key to preventing ulcers is:
(1) to teach a patient how to prevent injuries to himself in the first place,
(2) to teach him 'self-care' for any injuries he does receive, in their earliest stages. All primary care workers should be able to teach this. When the tissues have been damaged, they will usually heal if he rests them completely.

Surgery is much less important than rest, at the right time, and for the right length of time. Antibiotics without rest will not heal ulcers.

Ulcers commonly start in the deeper tissues, and develop slowly over several days, so teach him to recognize an ulcer as a 'hot spot' in its 'pre-ulcer' stage, before the skin over it has been broken. Get a radiograph to exclude underlying osteomyelitis.

A hot spot is a warm area of skin, usually with swelling, that occurs after activity, and persists during at least 2h of rest. In an anaesthetic foot, a hot spot may be the only indication of some underlying pathology, such as a fracture, disintegrating bone, a strain, or an abscess. Any of these may break through to the surface, and form an ulcer.

The patient, or a family member, must learn to look for hot spots, because they mean, “Stop!” He must take them seriously, and rest the foot until all signs of inflammation have gone. Rest at the hot-spot stage is the only way to avoid the serious damage that starts the downhill road to amputation.

The risk of an anaesthetic foot developing an ulcer depends partly on the shoe (if there is one), and partly on how much it is injured by walking. Perhaps he can ride a donkey, or a bicycle? The kind of shoe he needs depends on the state of the foot, as defined by the 'degree of risk'. Many patients with moderate- or even high-risk feet can remain free from ulcers without moulded shoes (32-22) if:
(1) they practise self-care,
(2) they have microcellular rubber insoles in their sandals or shoes (32-22A,B,C),
(3) they limit their walking, and
(4) they take small steps.

Moulded shoes are more difficult to make, and many hospitals manage without them. With a little instruction, a local cobbler should be able to make a suitable unmoulded shoe in the local style, with the necessary insoles and straps, and using only the local materials. If you want him to make a moulded shoe, he will need these special materials:

MICROCELLULAR RUBBER. This has a closed bubble structure, and is much more resilient than ordinary 'foam rubber'. Some shoe factories can provide it. It is not the same as the foam plastic used for cheap sandals, which is less resilient. Car tyres make good soles, and inner tubes can make uppers.
FOAMED POLYETHYLENE, 1cm thick. This is a light thermoplastic which a skilful cobbler can use to make a moulded shoe, for a moderate- or severe-risk foot. It resists wetting and is easily cleaned, but it does need an oven. Its main disadvantage is that it wears away in <6 months. Heat a piece of sheet to exactly 140ºC in an oven and hold it at that temperature for five minutes: place it on a 10cm polyurethane foam pillow; and then ask the patient to stand still on it until it is cool, or let him sit while you force the foot down on it (32-24C). It will not burn him, and will set in the shape of the sole (32-24D,E). Be sure to support this moulded material with microcellular rubber, or cork and latex, built up to produce a flat sole; foamed polyethylene is not resilient enough to make an insole by itself.

'SELF-CARE'. Teach a neuropathic patient to:
(1) Recognize that his anaesthesia is abnormal.
(2) Care for his anaesthetic limbs, and prevent injury.
(3) Inspect the limbs daily, so that he can remove any thorns, and recognize and care for any wound, either open or closed, while it is still small, and before it gets worse.
(4) Rest the limbs when they are injured.
(5) Recognize and understand the seriousness of 'hot spots' and corn formation.
(6) Treat the 1st ulcer as the calamity that it really is.

LOOK FOR SWELLING AND REDNESS
FEEL FOR 'HOT SPOTS'

CAUTION!
(1) Explain that it is injury to anaesthetic feet and not the disease itself which leads to ulceration and loss of tissue.
(2) Walking must be limited if there is a hot spot, or an area of deep tenderness.

(a) Examining an insensitive foot
Look for swellings, injuries and callosities. Are any of the toes pushed apart (with oedema from an injury)? Examine the arches of the feet on standing, and look for flattening. Feel the whole foot. Warmth or swelling suggests active pathology, and the need for extra care.

Press deeply over the common sites of ulceration (32-26A,D,G). There may still be deep pain when all ordinary sensation is lost. Assess the circulation, feel the peripheral pulses and the temperature of the skin. Time the capillary return.

FOOTPRINT MAT, also called a rubber Harris mat. This is a mat with little rubber ridges which you ink. Place a piece of paper on the inked mat and ask the patient to walk on it. The greater the pressure, the blacker the ink impression. If you are really interested in the care of leprosy feet, get a footprint mat.

FOOTWARE FOR FEET AT RISK

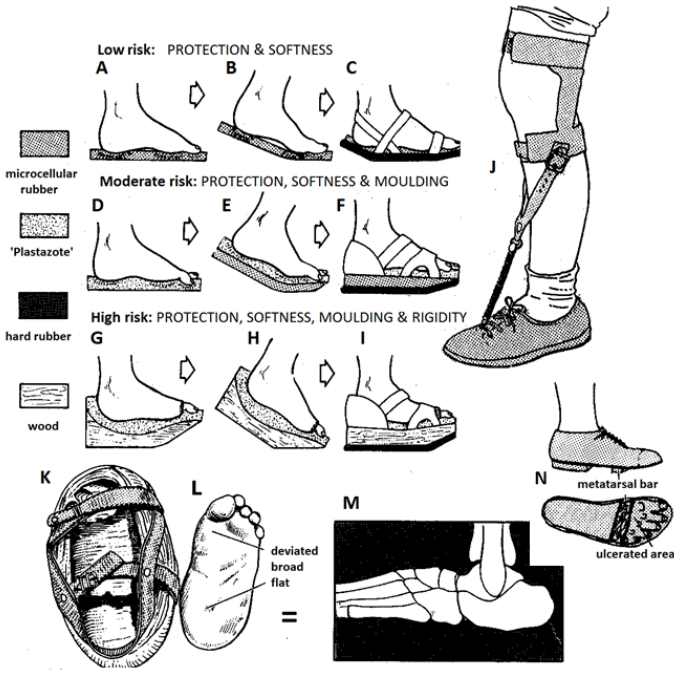


Fig. 32-22 FOOTWARE FOR FEET AT RISK can be made by any cobbler if you are prepared to teach him.
LOW-RISK FEET. A, microcellular rubber distributes the pressure. B, dark areas indicate where there is increased pressure on walking. C, car tyre sole applied.
MODERATE-RISK FEET. D, first layer of 'plastazote'. E, layer of microcellular rubber. F, layer of car tyre.
HIGH-RISK FEET. G, layer of 'plastazote' in a carved wooden clog distributes the weight evenly. H, when the patient walks, neither foot nor ankle flexes, and weight continues to be spread evenly. I, completed sandal. J, toe-raising strap for footdrop. *This is a very helpful device, so don't fail to fit one when it is needed.* If necessary, use canvas or plastic straps and make the 'spring' from a car inner tube. K, properly made shoe can protect a badly damaged foot. L, note that it has no ulcers, even though it has lost its arches, and its toes are clawed and deviated. It has remained free from ulcers because the patient limited his walking, and because the shoe has a layer of microcellular rubber built up under a moulded 'plastazote' insole. M, radiological view of a flattened arch and calcaneum. N, the simplest modification for an ulcerated foot is a metatarsal bar, stuck or sutured to the outside of the sole, just behind the metatarsal heads, proximal to the ulcerated area.
After Bryceson A, Pfaltzgraff RE. Leprosy. Churchill Livingstone 2nd ed 1979 Figs 11.1-3 with kind permission.

Watch the patient walking barefoot. You can easily miss a dropped foot if it is also short. Ask him to walk on his heels. He cannot do this if the *anterior tibial* or *peroneal* muscles are weak. Finally, *don't forget to look at his shoes!*

(b) Differential diagnosis of neuropathic foot

(1) Diabetes mellitus,
(2) HIV disease,
(3) Peripheral vascular disease,
(4) Syphilis,
(5) Leprosy,
(6) Spinal pathology,
(7) Inherited neuropathies,
(8) Peripheral nerve damage, *e.g.* from badly located IM injections.

(c) Testing for neuropathy

To chart sensation carefully needs time. Check this in the territory of the medial and lateral plantar and calcaneal nerves (32-23). Make your checks out of 5 attempts: a positive result is ≥3. Place a 5/0 monofilament on the skin with just enough pressure to bend it: check if the patient perceives touch. Test temperature with a test-tube filled with water at 50^0C, and with an ice bar. Test pressure sensation with the blunt end of an office pin pressed just hard enough to blanch the skin. Test pain sensation likewise with a sterile safety pin pressed just hard enough to deform the skin. Test 2-point discrimination with a specially home-made paper clip discriminator (32-23B) pressing its points applied just enough to blanch the skin, measuring the discrimination (in mm) sensed by the patient.

SENSORY AREAS OF THE SOLE

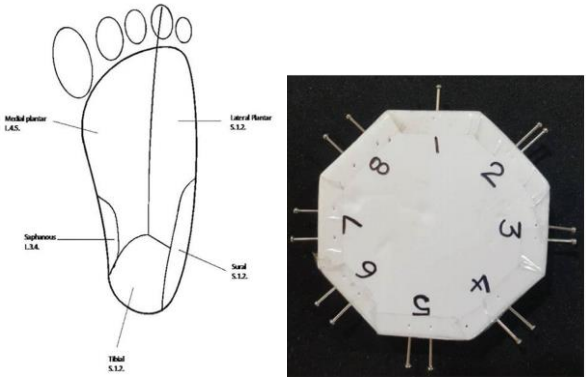


Fig. 32-23 TESTING SENSATION ON THE SOLE.
A, sensory supply areas. B, home-made discrimination tester.
After Agarwal P, Mukati P, Kukrede R, Sharma D. Neurologyindia.com DOI:10.4103/0028-3886.310095

(d) Skin care

Denervation of the skin reduces its natural secretions and makes it dry, so that it more easily cracks, fissures, and becomes infected. Softening dry skin reduces these dangers, and may allow any fissures that have formed to heal. So ask the patient himself to get plain water, without detergents, into the dry feet (or hands) by soaking them for 15-20mins at least twice a day. *Don't use warm water in case it is too hot!* If a fungal infection is present, add potassium permanganate to the soaks. Then ask him to cover the skin with petroleum jelly, or any kind of grease or oil (including car oil).

It is the water that is important, not the grease which keeps it in. *Beware of cockroaches which like the oil:* advise a patient whose living conditions are poor to get a cat, or use insect repellents.

Pare away thick corn with a surgical blade, or ask the patient himself to rub it away with a pumice or other stone. Remove rough corns regularly, because it may split and crack, or cause ulcers by pressure.

(e) Other ways to prevent ulcers
When necessary, remember to:
(1) Correct deformities. If there is a foot drop, fit a toe-raising strap (32-22J).
(2) Advise taking short steps, which will reduce the pressure on the front of the foot and the heel.
(3) Advise avoiding any hard edges or knots in the shoes or socks.
(4) *Beware of newly healed ulcers*. The scar will not have had time to become supple, and is in danger from any shearing force applied to it.
(5) *Avoid tight bandaging, and especially trying to compress a swollen or bandaged foot into a tight shoe*. Insist on wearing a thick sock. The best dressing substance is magnesium sulphate and glycerine paste, or simple syrup.
(6) *Avoid hot water bottles on ischaemic cold feet, as burns are easily caused.*

(f) 'Pre-ulcers'
Try to recognize a 'pre-ulcer foot', because from 3 days to 3wks of immediate bed rest at this stage may prevent a serious ulcer forming. Look for:
(1) swelling of the sole,
(2) separation of the toes (32-26C),
(3) necrosis blisters at the side of the foot, caused by fluid which has tracked from the necrotic area above the plantar fascia (32-26E,F),
(4) 'hot spots' (32.11),
(5) redness,
(6) pain (if there still is any sensation), especially pain on deep pressure.

(g) Neuropathic feet at risk
(1) LOW-RISK FOOT is anaesthetic but has little or no scarring. It needs protection and a resilient sole. You can use a resilient insole in a shoe which is one size larger than one usually worn. This may be enough. *Don't make the insole too thick*, and make sure the shoe is well fastened, so that it does not slip and produce blisters. Alternatively, use a car-tyre sandal with an insole of microcellular rubber.

(2) MODERATE-RISK FOOT is anaesthetic, has multiple scars, and has lost some of the subcutaneous fat pad on its sole.

A shoe for a foot like this needs to be moulded, to take the weight off the metatarsal heads and spread it evenly over the entire sole. Such a foot will however do fairly well in a simple car-tyre and microcellular rubber sandal, if the corn is kept well pared down. Or, make a piece of moulded 'plastazote' as described above.

When the base has set firm, build microcellular rubber up underneath it, and then fit this to a car-tyre sole. If it is made as a sandal, it will need a retainer for the heel moulded into it. A shoe with a moulded sole is better than a sandal at preventing the foot slipping out, but it must have a well-fitting upper with buckles, laces, or straps, so that it remains in its correct relationship to the foot.

(3) HIGH-RISK FOOT has, in addition, a mild deformity, such as flattening of the arches, and shortening, or loss, of toes. It needs a shoe which is moulded to conform to it completely, and has a rigid sole. Build microcellular rubber up under a sole of moulded 'plastazote', and carve a wooden rocker clog to fit it; then fit this with a hard rubber sole. A clog is rigid, so its front end must be boat-shaped (32-22G). Some of these feet do well in microcellular rubber sandals, if their owner looks after them carefully.

(4) DISINTEGRATED FOOT has a major bony deformity such as fragmentation of its tarsal bones, or is 'boat-shaped', (32-26K) or has a dislocated ankle. Rehabilitation is difficult; reconstructive surgery and an adapted orthopaedic boot (32-13B) may be necessary.

(h) Protective footwear
Instruct a cobbler how to make the footwear described above, and to follow the local styles as much as possible. Make the straps broad, and adjustable with buckles or laces, so as to allow for swelling or bandages. The simplest protection for an ulcerated foot is a metatarsal bar, stuck or sutured to the outside of the sole, just behind the metatarsal heads (32-22N).

CAUTION!
(1) *Never use nails or wire to make or repair shoes:* glue and sew them.
(2) If the foot is significantly supinated or pronated, only major surgery will allow satisfactory walking.
(3) New shoes need special care. Warn the patient to walk short distances only until the leather has become adjusted to the foot; meanwhile he should use the old ones most of the time.

(i) Paralysis of the feet
If there is a posterior tibial nerve palsy, either:
(1) Apply a firm bandage to limit friction at the back of the ankle. Combine this with a heel retainer, to minimize the use of the small muscles of the foot, and trauma to the anaesthetic sole; or
(2) Apply a padded plaster boot.

If there is an acute common peroneal nerve palsy, producing a flapping gait and foot drop, passive exercises will help to stretch the Achilles tendon and prevent a contracture:
(1) Advise squatting with the heels flat on the ground.
(2) Advise standing erect about 70cm from a wall, keeping the feet flat on the ground, and, with the palms of the hands flat on the wall, doing 'press-ups' in the vertical position.

Some protective device is also necessary:
(1) During the day, fit a toe-raising spring (32-22J). This will allow some work. By night, apply a posterior slab to hold the ankle in neutral position.
(2) Or, apply a complete plaster cast, including the foot and leg up to the middle of the thigh, with 15º of flexion of the knee, and with the ankle in neutral position, taking care that the cast does not press on the nerve. Leave this on for 6wks.

PROTECTIVE FOOTWEAR FOR NEUROPATHY

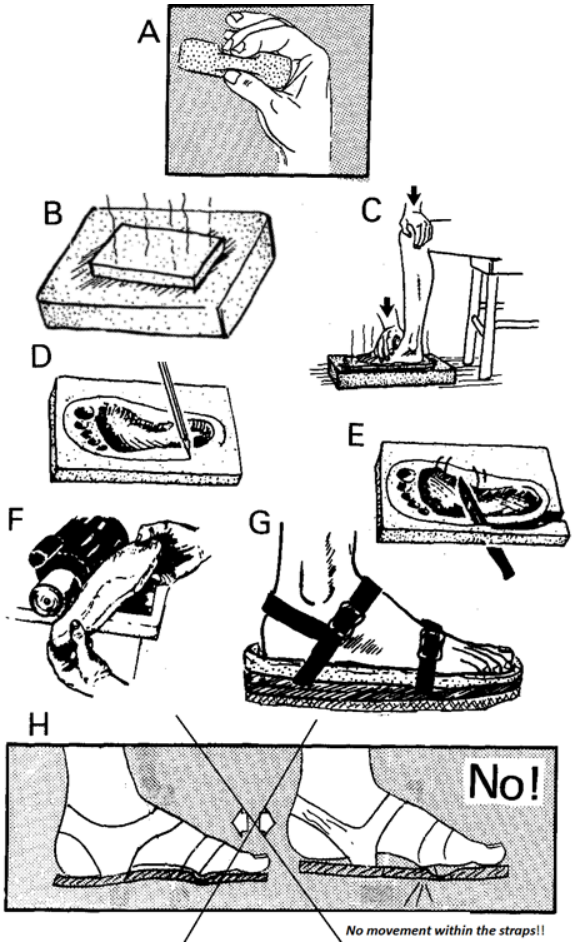


Fig. 32-24 PROTECTIVE FOOTWEAR IN NEUROPATHY.
A, the right kind of microcellular rubber can be squeezed to half its thickness; if it is flatter than this it is too soft, if it is thicker it is too hard. B, sheet of hot 'plastazote' laid on soft foam. C, take the mould by applying even pressure and holding it for 3mins. Mark it out (D), and cut it (E), so as to project 0·5-1cm in front of the toes and behind the heel. Shape it (F), smooth it on a polishing disc, and support it with a layer of microcellular rubber and stick it to a hard rubber sole. G, completed shoe made from moulded 'plastazote' supported by layers of microcellular rubber, and soled with car tyre. H, *the moulded shoe must be anchored to the foot and must not be allowed to move about*.
After Brand P. Insensitive Feet: A Practical Handbook on Foot Problems in Leprosy. Leprosy Mission International, with kind permission.

If there is also plantar ulceration with the foot drop, be sure to use a posterior slab or a cast. If the ankle is not supported, the Achilles tendon is likely to contract on bed rest. Provide crutches while the ulcer heals, so that there is never any pressure applied on the ulcer.

If there are clawed toes, transfer the *flexor longus* tendon to the extensor expansion on each toe (32-29G).

FIND OR TRAIN A CAPABLE SHOEMAKER TO HELP YOUR PATIENTS

32.12 Renervation of the foot

If you can restore sensation to the foot, the life of a patient will be radically transformed, and the risk of ulceration very much diminished.

(a) Saphenous nerve transfer (GRADE 2.5)
Make sure you have carefully documented the sensory loss (32.11).

Apply an arterial tourniquet at the thigh. Make a V-shaped incision on the medial side of the ankle (32-25A), starting anterior to the medial malleolus extending proximally for 7cm and then down to between the medial malleolus and the Achilles tendon. Identify the saphenous nerve running on the medial surface of the tibia with the greater saphenous vein (32-25B).

Dissect the saphenous nerve free of its fascia for 7cm. In the distal $^1/_5$ of the leg, it splits into dorsomedial and supramalleolar branches. Here both branches contain 4-5 fascicles. Identify the posterior tibial nerve in its groove between the medial malleolus and the Achilles tendon after dividing flexor retinaculum for 5cm. Make an epineural incision on the posterior tibial nerve, and identify 4-5 sensory fascicles using an intra-operative electric stimulator. Those fascicles which do not evoke motor contractions of the foot muscles are considered as sensory fascicles.

Cut these proximally and reroute them towards the saphenous nerve. Cut this distally and join its sensory fascicles to those of the posterior tibial nerve using 9/0 nylon sutures and epineural repair (32-25C, 48-1). Close the wound and apply a below-knee plaster slab for 3wks. Then allow walking with a microcellular rubber insole. Sensation should return by 6 months.

SAPHENOUS NERVE TRANSFER

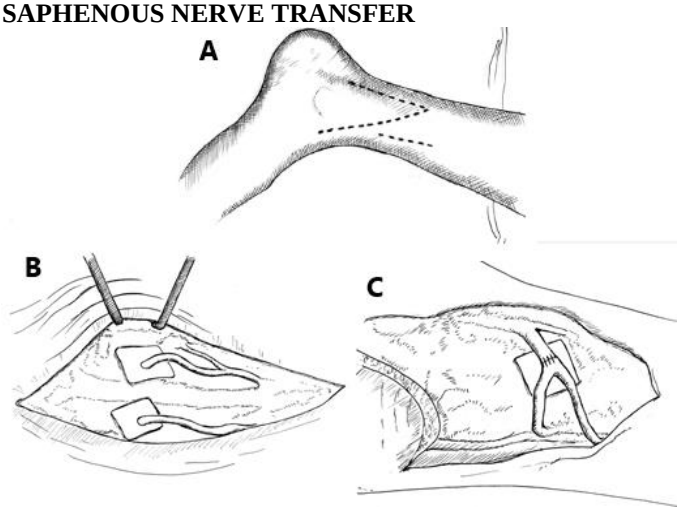


Fig. 32-25 SAPHENOUS NERVE TRANSFER.
A, V-shaped incision on the medial side of the ankle. The other line shows the surface marking of the saphenous nerve. B, saphenous nerve dissected from its fascia, the lower the anterior branch, and the upper the posterior branch. C, fascicles of the posterior tibial and saphenous nerves joined.
After Agarwal P, Shukla P, Sharma D. Saphenous nerve transfer: A new approach to restore sensation to the sole. J. Plas Reconstr Aesth Surg 2018; 71:1704-10.

32.13 Foot ulcers

An uncomplicated ulcer is only skin-deep, does not involve bone or deeper structures, and usually heals easily if the patient rests the leg. A complicated ulcer has involved the bone underneath it. It has a deep sinus or marked infection, and healing is much more difficult.

The Wagner classification describes different stages:

I	simple ulcer
II	with cellulitis
III	with bone involvement
IV	with spreading infection
V	with gangrene

Simple plantar warts or corns from ill-fitting shoes can result in ulceration, especially if the patient cannot feel the discomfort properly. *Don't use LA to cut out plantar warts and shave corns;* you may go too deep and the injection is anyway usually unhelpful and more painful than the procedure!

Ulcers in a neuropathy patient are almost inevitable. Help the patient himself to find the cause of the injury. Never let him accept that the cause was the disease. Was it caused by repeated stress, or by a blow, a puncture, or a burn?

Concentrate all your educational energies on him. You can do much more for a patient with the 1st ulcer, than for one whose foot is already mostly destroyed.

If you can find some way of resting an ulcer, it will usually heal. This means that the patient must 'not take one step' on it, until it has finally closed over and all the scabs have fallen off. If it is uncomplicated, this takes 4-6wks.

You can:
(1) Try bed rest. Unfortunately, this is rarely successful because staff often don't understand fully the need for rest and neither explain nor encourage this, and because there is no pain and thus apparently little incentive to stay in bed.
(2) Allow the use of a splint and crutches continuously until the ulcer has healed. This is unlikely to be successful unless you educate and supervise the patient carefully.
(3) Make a special curative shoe, with a rigid rocker bottom and a specially moulded surface for the sole of the foot. This needs much skill, and is not described here.
(4) Put the foot in a cast.
N.B. Antibiotics are of no use.

A plaster cast is one of the most practical ways of resting an ulcerated foot. It immobilizes the foot, it spreads the strain of weight-bearing, it is quick to apply, and it is easy and effective. You can apply one in a remote clinic and send the patient home, provided you tell him that he must absolutely avoid walking on the foot. If you apply a cast on the indications listed below, it will usually allow an ulcer to heal in 6wks.

Unfortunately, although resting a foot in a cast may heal an ulcer, even if walking on it, the bones and ligaments weaken. Bones only retain their normal strength if they are regularly used. Rest causes bones to lose their minerals, and ligaments their strength.

The result is that when the cast is finally removed, the patient may be delighted to find that the ulcer is healed, but he may not realize that anaesthesia is preventing him from experiencing the stiffness and pain that protect a normal foot. Consequently, he may be tempted to use the anaesthetic foot too vigorously, with the result that it dislocates, or its tarsal bones fracture, and he ends up with a worse neuropathic foot. So use casts cautiously and remember their risks.

When you remove one:
(a) warn of the sad consequences of energetic early exercise,
(b) start a programme of 'walking training' which aims at a slow return to full activity in 7-10 days,
(c) be sure to teach 'self-care',
(d) be sure also to watch carefully for 'hot spots' (32.11),
(e) *most importantly*, supervise the patient for at least 1wk after you remove the plaster.

Bone damage is common, and serious, and may be the result of:
(1) Sepsis spreading from an ulcer, particularly if the patient walks on it.
(2) Mechanical strain, which is particularly likely to occur when the protective mechanism of pain is absent.
(3) Disuse atrophy in bed, or in a plaster cast.
(4) Osteoporosis, which may predispose to fractures.

Get a radiograph of the foot early.
The best way to minimize bone damage is to treat ulcers carefully, so that bone is not damaged in the first place. There are however also some additional principles:
(1) Keep the weight-bearing surface of the sole as large as you can.
(2) When you remove bone surgically, *don't do so unnecessarily*. Make sure it really is dead or infected.

Dead bone is usually grey or black; it has no periosteum, and so feels rough to a probe. When you nibble it with forceps, its fresh surface is pale, and not pink. Ideally, you should allow a *sequestrum* to separate before you remove it, but this takes 8-12wks, during which time the ulcer will not heal. You can shorten this time by removing dead bone.

When bone has been damaged, clean up the mess it has caused. For example, if there is a deep sinus under an ulcer with bone involvement, rest the leg for a few days to localize the infection. Then remove the dead soft tissue and bone, perhaps one or more metatarsal heads, leaving the toes if you can.

The short *equinus* foot of leprosy is one of its end results, and is due to the absorption of bone, which may be due to:
(1) Neglected ulcers and infections.
(2) Paralysis of the extensor muscles.
(3) Unduly radical surgery. Muscle imbalance may pull the heel up too much, or push the forefoot down too much, so that it increases the pressure on the metatarsal heads, and so causes worse ulceration and more shortening.

A boat-shaped foot is another of the late effects of neglected leprosy. The arch is destroyed, and instead of being concave, it becomes convex, often with ulcers and bony spurs on the convexity.

PLANTAR ULCERS IN NEUROPATHY

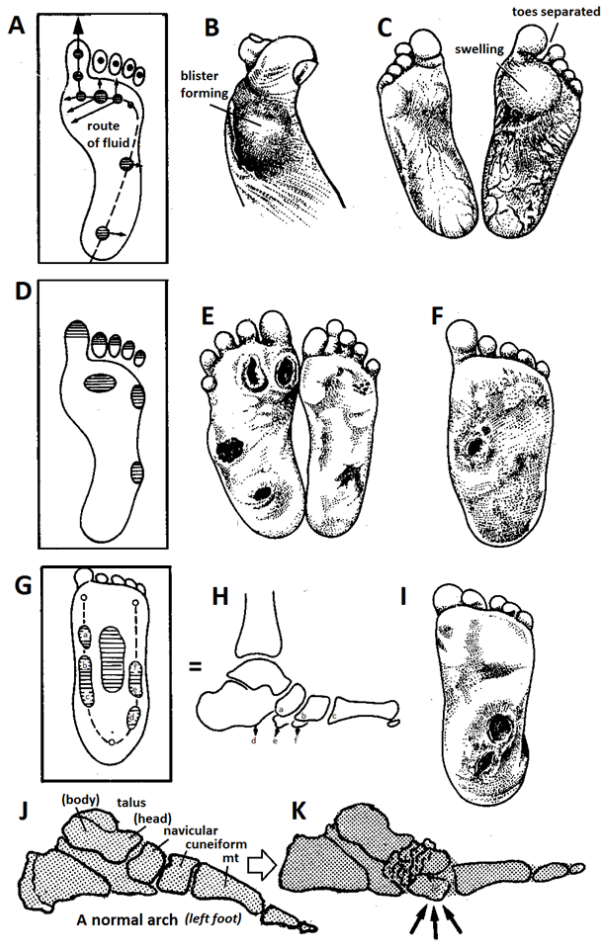


Fig. 32-26 PLANTAR ULCERS WITH NEUROPATHY.
A, where ulcers form in a flexible anaesthetic foot with intact muscles; the arrows show where fluid may track and B, where blisters form. C, fluid is forcing the toes apart and collecting under the ball of the foot. D, sites where ulcers form in a paralyzed foot. E, if there is a peroneal nerve palsy, the lateral arch collapses; ulcers develop at the lateral side of the foot; if there is a complete foot drop, the ulcers are anterior on the ball of the foot, under the metatarsal heads, or on the toes. F, if the medial arch collapses, the ulcers develop on the medial side. G, sites of bony prominences (a-f) in a collapsed foot where ulcers form. H, the flattened radiographic appearance. Each of its bony prominences (a) to (f) has produced an ulcer. I, 2 ulcers in just such a foot. J, radiology of a normal arch in the left foot. K, boat-shaped foot: the arch is reversed and ulcers form under the 'keel' of the boat.
After McDowell F, Enna CD, Surgical Rehabilitation in Leprosy. Williams & Wilkins 1973 Figs. 40-1,2,9, with kind permission. J,K, kindly contributed by Grace Warren.

(a) Management of acute ulcers
Insist on bed rest. Splint the foot and raise it to encourage drainage and prevent oedema. This is much the best treatment. Ambulant treatment seldom works. Forbid all walking except with crutches to reach the toilet. If necessary, fix a piece of wood to the dressings (32-28A) to prevent walking!

Don't apply a walking cast!

Local applications to an ulcer make little difference, so there is no need to change dressings at frequent intervals. Dress the ulcer 2-3 times/wk with hypochlorite ('Eusol'), hypertonic magnesium sulphate, sugar (which is best used daily), honey, ghee or some mild antiseptic. Or, soak it, scrape it regularly to remove excess corn, oil it, and dress it daily. When the discharge stops, you can apply a cast, leave the dressing unchanged for 6wks, and send the patient home, making sure he has learnt self-care.

If there is fever and other signs of generalized infection, such as a profuse discharge, or tender groin glands, use an appropriate antibiotic and elevate the leg.

CAUTION! Antibiotics have no place in treating uncomplicated ulcers: what they need most is rest!

When the acute stage is subsiding, and there is no sign of spreading infection, explore the ulcer with a sterile blunt probe to find out if there is exposed bone in its base.

If bone is exposed, feel if there are any loose pieces or sequestra, and remove them. Pack the ulcer with hypochlorite until it is healing well, and continue to rest the leg.

If bone is not exposed and infection is controlled, continue bed rest with a splint and crutches until the ulcer heals. Probe ulcers: if you reach bone, the chances of osteomyelitis being present are c.70%. *Pus swabs are, however, no help.*

(b) A short leg walking cast
INDICATIONS
A chronic *non-inflamed* ulcer whose base is visible without any necrotic bone, tendon, or other dead or infected tissue, which you must remove before applying the cast.

CONTRAINDICATIONS
(1) Signs of inflammation or infection: heat or oedema of the dorsum opposite the ulcer, excessive discharge, or regional adenitis.
(2) Involvement of a joint or synovial sheath (synovial discharge).
(3) Dead bone or tendon or capsular sloughs in the base of the ulcer.
(4) A long deep sinus with small openings whose base you cannot see.

METHOD
Normally leave the toes open, unless you have to keep out stones and sand.

Measure the feet for shoes before applying the cast: when you finally remove it, allow no single step without these shoes being worn. Shape the Böhler stirrup (32-27D, walking iron) to the leg before you apply the plaster.

Dress the ulcer with dry gauze or a simple ointment. Cover, but *don't pack the wound*; discharge must be able to escape easily. Apply stockinette, a nylon stocking, or an evenly applied gauze bandage.

N.B. Don't apply excessive padding to bony prominences. Use strips of adhesive tape to fix 3 strips of padding (32-27A), but *don't apply the tape directly to the skin*. If you don't have padding, use many layers of bandage instead.

Use the prone position with the knee at 90º and the leg vertical. Apply a thick layer of plaster to the leg without pressure. End the cast 5cm below the head of the fibula, to avoid pressure on the common peroneal nerve, and leave the toes open.

Apply a back slab and circular reinforcing layers. Then fit a Böhler iron or a walking board (32-27B, wood with a piece of car tyre). Let the cast get completely dry before weight is placed on it.

Alternatively, a thin, well-moulded layer of plaster, covered by a layer of fibreglass, will make a longer-lasting cast. Preferably, use fibreglass tape rather than sheet, because it lasts longer.

CAUTION!
(1) *Don't mould the cast under pressure to obtain the required position*, or you may cause ulcers and gangrene.
(2) Ask an assistant to hold the ankle at exactly 90º or slightly dorsiflexed (32-27E), until the plaster has set; *it must not be plantar-flexed or inverted or everted*.
(3) *Don't press into the plaster with your fingers*, because you may produce pressure points where more ulcers will develop.
(4) Remember that no pain is felt. A wrongly applied cast may cause ulcers! *So don't apply excessive pressure over a tight bandage*.

A PLASTER CAST FOR NEUROPATHIC ULCERS

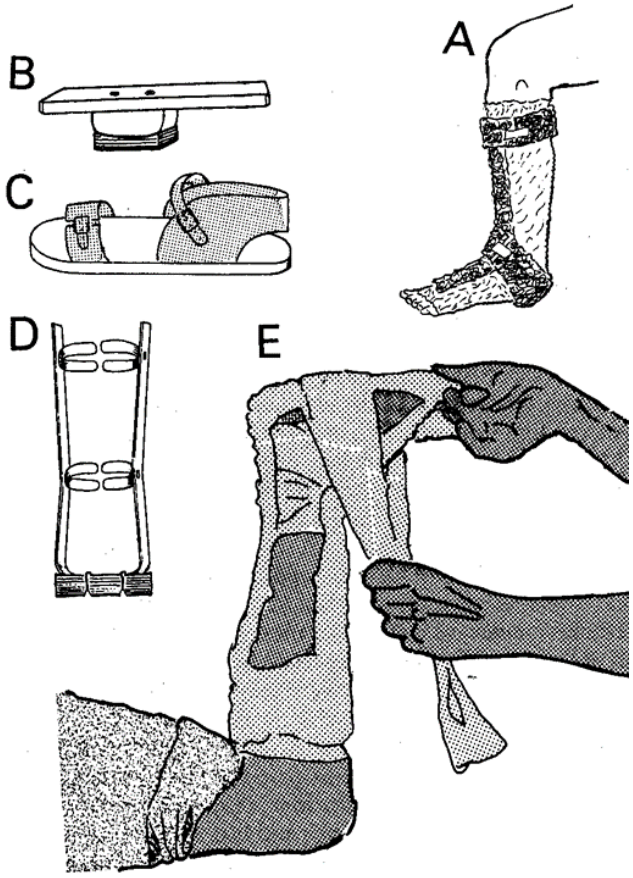


Fig. 32-27 A PLASTER CAST FOR NEUROPATHIC ULCERS.
A, put extra padding as shown under a plaster cast. B, wooden rocker shod with car tyre. This has a single bar. If a patient has casts on both legs, double bars on the rockers will enable him to walk more easily. C, rubber-soled sandal with plastic straps to wear over a plaster cast. D, locally made Böhler walking iron shod with car tyre. E, apply the cast in the prone position. Ask your assistant to hold the toes up and to pull downwards on a loop of bandage placed as shown. This will flex the ankle, and help to form a better arch if one is needed. Apply the plaster over the bandage.
Kindly contributed by Grace Warren.

If there are ulcers on both feet, a wheelchair may be necessary. If the patient has to crawl, provide 'hand sandals' to protect the hands. Make these with a piece of microcellular rubber, and give them a single strap. Incorporate a rocker bar in the plaster to take pressure off the ulcer site. If there are casts on both feet, double bars on walking boards will allow walking. Alternatively, make cushioned wooden rockers on a flat board and sandals to go over them.

Leave the cast on for 6wks. Remove it earlier if there are signs of infection (smell, discharge, pain). When you remove it, the shoe that you measured earlier should be ready. Make sure that there is a period of 'walking training' before resuming full activity. Apply a firm bandage, and start walking in a carefully graduated way. Check the foot for swelling or an increase of temperature. Rest it again if signs of inflammation return. Advise walking as little as possible, taking short steps, and avoiding uneven ground, sudden strains, and long walks.

If the ulcer has not completely healed in one cast, apply another.

(c) Bone involvement
INDICATIONS FOR REMOVING BONE
(1) There is osteitis.
(2) It is loose.
(3) It is projecting into a septic cavity with no obvious blood supply around it.
(4) It is projecting after an ulcer has healed, so that it forms a pressure point; if so, cut it horizontally.
(5) One metatarsal is obviously longer than the others, and the skin over it is ulcerating. Apart from the first metatarsal, which may usefully be longer, the others should all be on the same line across the foot, so that walking is possible without one sticking out prominently and taking extra stress.

METHOD
Use appropriate antibiotics pre- and post-operatively.
Apply a tourniquet (3.4), try to loosen the bone, and cut it off at the line of separation. If this line has not yet formed, nibble it at the point where you see the periosteum is adherent again.

CAUTION!
(1) *Don't remove bone from the base of an ulcer unnecessarily, especially in the heel.*
(2) Probing an ulcer will tell you if bone is exposed, but *not if it is dead*. Exposed bone may be healthy, but the soft tissues will take time to grow over it.
(3) *Never strip periosteum unnecessarily*, because this may kill the bone under it.

METHODS FOR NEUROPATHIC ULCERS

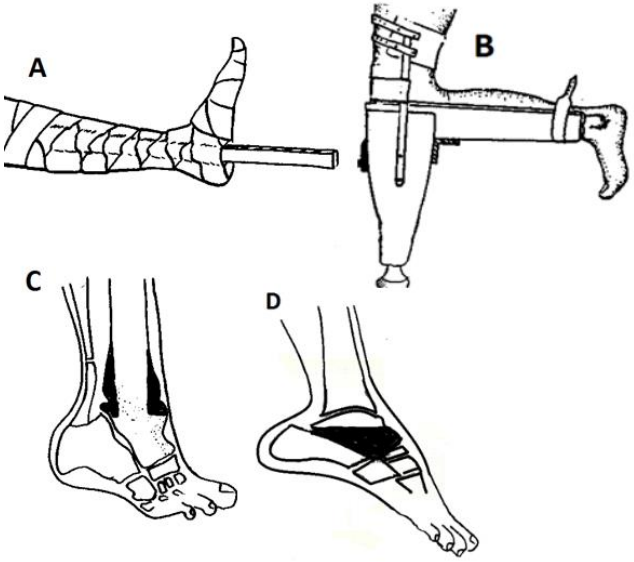


Fig. 32-28 METHODS FOR NEUROPATHIC ULCERS.
A, one way to prevent walking on an ulcer while it heals is to bandage a wooden bar to the leg. This is only suitable for short periods. B, an alternative to bed rest is to provide a walking prosthesis like this. C, if all else fails, for an *equinus* foot, try to arrange an ankle arthrodesis: the malleolar parts of the fibula and tibia and parts of the talus are sawn off and used as bone grafts to fix to the talus internally. D, for an *equinovarus* foot, arrange a subtalar triple arthrodesis where a wedge of bone from the talus, calcaneus and navicular is removed to get the foot into dorsiflexion.
A,B, after Brand, P. Insensitive Feet, A Practical Handbook on Foot Problems in Leprosy, Leprosy Mission International with kind permission. C,D, after Fritschi, EP. Reconstructive surgery in leprosy, Wright 1971.

(d) Difficulties with neuropathic feet
FOOT DIFFICULTIES

If bed rest is impractical:
(1) Provide a splint and crutches, and avoid weight-bearing on the foot with the ulcer. The splint may be plaster (expensive and short-lasting), wood, plastic or wire (mesh fencing wire) but should last 6wks; or
(2) Attach a projecting bar to the foot (32-28A) and provide crutches.

If a plaster cast for an ulcer is impractical, you can:
(1) Fit the kneeling leg prosthesis (32-28B), which is suitable for limited activity only.
(2) Fit a 'healing shoe' which is less cumbersome than a cast, but also less effective. It must have (32-22I):
(a) a rigid sole with a central rocker,
(b) an insole (ideally 'plastazote') moulded exactly to the shape of the foot,
(c) an upper strapped round the foot and ankle, so that they cannot move in relation to the shoe.

If the ulcer recurs, check the way the patient cares for his feet. Does he inspect them and soak them daily and remove rough corn? Look at the shoes:
(1) Is there increased pressure in some area which has caused necrosis?
(2) Are the straps so loose that they allow movement of the foot inside the shoe, or so tight that they cut into skin?
(3) Can the contour or fit of the shoes be improved?
(4) Does the patient always wear the shoes?
(5) How far does he walk without resting? Can he walk less, or walk with less pressure on the ulcer, or more slowly?

There are 2 possibilities:
(1) You may be able to excise the ulcer, and all the scar tissue under it, and then graft it with split skin. This may provide a more suitable bed for the regrowth of subcutaneous tissue than the original scar tissue.
(2) You may rarely be able to excise the scarred area, and close the gap you have made with monofilament sutures. This means an initial relieving incision on the dorsum or side of the foot, and packing the cavity till healing occurs from the base of the wound. Use honey or other suitable dressings (34.9).

If the metatarsal heads protrude, shave them off.

If the feet are well cared for and the right shoes are worn, the ulcer should not break down again. If it does, there is some underlying abnormality, such as:
(1) Chronic osteomyelitis of the bone under the ulcer. Remove sequestra surgically (7.5).
(2) A protruding bone spur which needs excising.
(3) A thick scar which splits under tension with walking.
(4) Inadequate subcutaneous tissue over the metatarsal heads.
(5) Malignant change in the ulcer (34.5).
(6) Claw toes which repeatedly ulcerate.

CAUTION! When you treat ulcers, avoid cutting into living bleeding tissue unless it is to:
(1) Open an abscess.
(2) Improve drainage from a deep sinus.
(3) Remove necrotic tendon, muscle, or bone.
(4) Remove a free-lying sequestrum (7.5).
(5) Remove bone that is so placed that healing and normal function are mechanically impossible.

If there is an ulcer on the lateral border of the foot (32-26E), it is likely to be associated with peroneal nerve paralysis (32.11). Treat it by bed rest and splints or casts. When it has healed, a toe-raising strap attached to the area of the 5th metatarsal head may help to prevent recurrence.

If you suspect infection under an ulcer in the middle of the lateral border, surgically pare down the cuboid or the base of the 5th metatarsal, and remove any infected tissue. Make a dorsolateral incision which leaves a sufficient bridge of tissue between the incision and the ulcer. Turn back the infected tissues by subperiosteal dissection, trim the bone, remove necrotic tissue, excise the ulcer with an elliptical incision on the sole.
When the wound is clean, try primary closure with monofilament to achieve healing of the plantar wound without a large scar which might ulcerate again.
You can allow the dorsolateral wound to close by granulation if you cannot easily close this. *Don't let it close if the depth of the cavity is not clean*. A toe-raising spring (32-22J) may help to prevent recurrence.

If a terminal phalanx becomes visible in an ulcer at the tip of a toe (or finger), nibble it away with a bone nibbler. If it is badly infected, disarticulate it. If necessary, use a fish mouth incision over the top and down the sides, which will leave the pulp intact.

If you remove part or all of the middle or proximal phalanges, approach them through incisions at the sides of a toe (or finger). If the remaining toe is stiff, awkward or painful, amputate it (35.7).

If bone is exposed under a heel ulcer, be very careful about removing it from the calcaneus: you can easily remove too much, and a foot without a heel can be a problem. Patients can however walk on very little calcaneus or even none, if you provide them with a rubber heel pad. Try conservative management with special footwear and daily skin care. A normal calcaneus has a spur which projects forwards along the line of the plantar ligaments parallel to the ground; this is harmless.

EXCISION OF A CALCANEAL SPUR (OR OTHER BONY PROJECTION) (GRADE 2.1)

If there is an abnormal residual bony spur on the undersurface of the calcaneus or elsewhere, associated with an ulcer and which projects vertically downwards, remove it. Spurs may form under any prominent bone in a boat-shaped foot. Irregular bone may also develop because of a fracture or an infection. *Don't remove these bony projections through the ulcer*, because this will make the plantar scar bigger. Instead, paint the ulcer edge with gentian violet. Then make an incision round the back or lateral side of the heel (32-29B), so as to avoid the medial calcaneal vessels. Deepen the incision to the bone (32-29C), and lift the heel pad off the bone by clean sharp dissection. Continue the incision, so as to raise a flap of heel and plantar fascia, and mobilize the ulcer. Then excise and suture it as described above (32-29D). Trim the calcaneus with an osteotome to leave a flattened surface. *Don't remove bone unnecessarily*, or leave new sharp edges or corners to form new ulcers.

If there is a short foot, examine the patient carefully to see if the heel is taking its proper share of weight. *You can easily miss foot drop in a short foot.* Ask him to walk on his heels; if he cannot do so, some of the muscles are weak. Lengthening the Achilles tendon may help, even to the point of making the calf muscles useless, because this will make him walk mainly on the heel, and less on the front of the foot. If there is definite paralysis of the dorsiflexors, he will be better off with a tendon transfer (32.13). If this is not practical, fit him with a toe-raising spring (32-22J).

If where there is muscle weakness, the tarsal bones disintegrate, you may find this in any of 3 stages.
(a) 1st stage: the foot is hot, it may be swollen, but its shape is unchanged. Raise the foot to allow swelling to subside.
(b) 2nd stage: the foot is still hot with active bone disintegration; its shape becomes abnormal, and it may be hypermobile. Raise it in a splint for 3 days to reduce swelling. Then use the prone position (32-27E) and mould the foot into as functional a position as you can, accentuate its arch as much as possible, and apply a cast. Leave this on for 6-12 months, and then mobilize with care.
(c) 3rd stage: the foot is no longer hot, showing that the bone lesions are no longer active. If there are rough bones, which will be likely to cause ulcers, trim them. A high-risk shoe (32-22I) may keep the foot ulcer-free. An arthrodesis (32-28C,D) may then be needed, after which a walking cast for 6-9 months is necessary. Hopefully, the foot will revert to the 'moderate risk' class; if it does not, a special prosthetic shoe or brace will be necessary long-term. Many of these patients can manage to live well in a simple sandal, with daily skin care.

If there is an old fixed deformity which cannot be altered, supply a special moulded high-risk shoe till you can arrange reconstructive surgery.

If there is no practical way to establish a good arch, at least try to get its bones healed and sclerosed. If the arch of the foot becomes completely flat, it should remain ulcer-free, but if the bottom of the foot becomes convex and boat-shaped, it will be more likely to ulcerate. If the talus and calcaneus are totally destroyed, perform an amputation (35.7).

If clawed toes develop, stage them, as ulcers are often associated, and treat as follows:

GRADE 1	**GRADE 2**	**GRADE 3**
Weak intrinsic muscles, mobile toes	Limited movements of toes	Fixed toes ± dislocation of mtp joints
No contractures	Moderate contractures	Severe contractures
Flexor tendon transfer	+ Excision metatarsal heads	+ ≥1phalanx amputation, or transmetatarsal amputation

N.B. For seriously clawed toes, you may have to remove at least one phalanx, or the metatarsal head, or both to straighten them.

If the remainder of the toes will not bear weight because they are so badly scarred, perform a transmetatarsal amputation (35-23).

If squamous carcinoma develops in a longstanding (Marjolin's) **ulcer**, the alternatives are local excision and amputation. You may be able to excise smaller lesions that don't involve bone and are distal to the midfoot (34.5).

ANKLE DIFFICULTIES

If there is an irreducible dislocated ankle or a fixed ankle deformity, a correction is necessary.

If there is an *equinus* or *equinovarus* foot, walking may be satisfactory, but elaborate footwear is necessary to prevent ulcers recurring. If walking is difficult, and particularly if there is fixed plantar flexion or inversion, an ankle arthrodesis (32-28C,D) is necessary. Afterwards, an ordinary high moulded shoe, or a sandal of microcellular rubber, may be adequate to prevent ulcers.

If the foot is pronated, a simple canvas tennis shoe may be enough. If not, a surgical correction is necessary. Afterwards, an ordinary high moulded shoe (32-22I) or a sandal of microcellular rubber (32-22F) may be enough to prevent ulcers.

SEPTIC COMPLICATIONS

If there are signs of an acute infection with lymphadenitis, treat with rest, cloxacillin or another suitable antibiotic, and if necessary, drainage. *There may not be a neuropathy*, and the foot may merely have a soft tissue infection.

If heat and swelling rapidly return and persist, there is active pathology, so apply a cast for 6wks, repeat the radiograph and plan treatment accordingly. After this interval, stress fractures and other bony lesions will have caused enough osteoporosis to be seen radiologically. If you are in doubt, or have no radiographs, suggest another trial of walking.

If in a case of leprosy, heat and swelling return a 2nd time, bone disintegration is highly likely; so reapply a well-fitting walking cast for 6-12 months, depending on its site and severity.

If there is septic arthritis in a toe ip joint, excise it through a dorsal incision, remove the remains of its ligaments and cartilage, pack the cavity, and keep the toe straight at its ip and mtp joints: this will produce a fixed toe.

If septic tenosynovitis complicates an ulcer, draining the tendon sheath may assist healing. Drain it through an incision along the arch of the foot. Clean out all the infection, as far back as is necessary to find and remove the infected tendon stump. Close the skin with monofilament, so as to leave the smallest possible scar on the weight-bearing area. Leave both ends open, so that you can irrigate the lesion until it is clean. Allow it to heal by secondary intention.

If a septic toe requires amputation, use a racquet incision on the dorsum (35-24), leave the metatarsal head, and only resect the surface cartilage if there is septic arthritis of the mtp joint. Drain or pack the wound dorsally.

If plantar ulceration results in osteitis of a metatarsal head, you may need to excise it (32-29I). This will move the weight-bearing area proximally, so that more ulceration is likely. If you can save a toe in good position, it will help to protect the area of the new 'metatarsal head'. If you can save the distal part of the first toe, it will help to protect the second metatarsal head, which may otherwise soon ulcerate. Sometimes, you may have to remove several metatarsal heads. Do this through dorsal longitudinal incisions between them. If there are plantar ulcers over the metatarsal heads, excise them, and close the incisions in the sole with monofilament. Leave drains or packs in dorsally.

(e) Operations on the feet in leprosy

Most leprosy ulcers don't need an operation, but there are some simple operations which you should be able to do. Try to correct clawed toes, because they predispose to ulcers at the tip of a toe, on the knuckle, and under the metatarsal head. Apart from the correction of clawed toes, most other tendon transfers are work for an expert. The only other possible exception is a *posterior tibialis* transfer for foot drop (32.13).

(1) EXCISING AN ULCER OR SCAR ON THE SOLE

You may be able to close the gap you have made by primary closure. To do this, excise the ulcer with an elliptical incision, and close the wound with deep mattress sutures (4-8) of '0' monofilament to eliminate dead space. Keep the wound dry, and leave the sutures in for 2wks.

If you can only close an ulcer under excessive tension, perform a Z-plasty (34-4). Loosely pack the dorsal incision, and leave it to granulate as described below. Make sure the bridge of skin, between the ulcer and the relieving incision, is adequate to maintain the circulation.

CAUTION! Only close clean surgical incisions by primary suture. Even some of these need drains to minimize haematoma formation. Remove the drains after 48h.

If there is deep infection, pack the wound and use honey or similar hygroscopic substance (34.9), till the wound is clean.

If there is a deep ulcer with a sinus track, outline this with gentian violet. Cut away all the violet-stained tissue, so that you remove all the infected areas.

If there is osteitis, excise or curette the sinus tracks and insert a pack.

(2) EXCISING AN ULCER ON THE HEEL

If osteitis is already draining through the centre of the heel, curette and pack the lesion, without trying to excise the ulcer. Stop all weight-bearing until the ulcer is healed. Provide a splint.

When the infection is controlled, trim any rough bone. As soon as the osteitis is controlled, excise the ulcer scar and pack the lesion laterally till it is clean. *Don't allow walking on trimmed bone* for 6wks, or until the wound is fully healed, and the scabs have fallen off.

Heel skin is specialized, so try to obtain primary closure. Make a 'fish mouth' relieving incision around the back or lateral margin of the heel (32-29B), then dissect to lift the heel pad off the calcaneus (32-29C), in order to be able to close the plantar defect.

SPECIAL OPERATIONS FOR LEPROSY

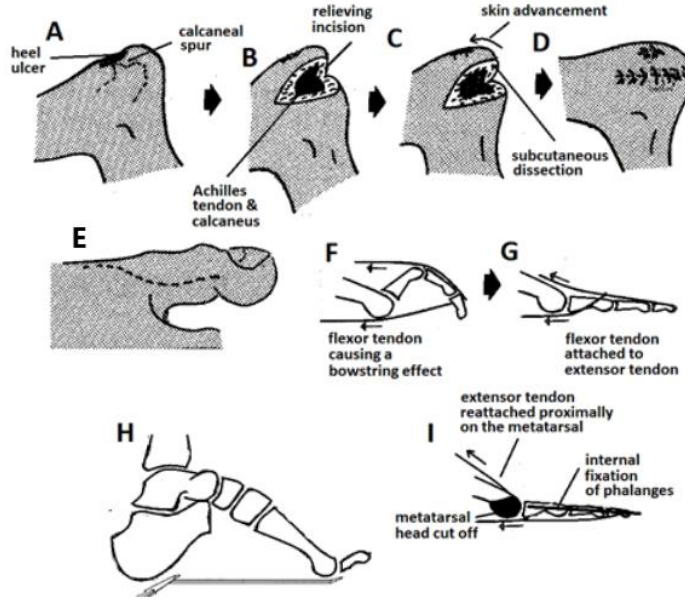


Fig. 32-29 SPECIAL LEPROSY OPERATIONS.
A, heel ulcer (with calcaneal spur shown). B, relieving incision in posterior heel skin taking care not to cut the Achilles tendon. C, dissection of the heel pad off the calcaneus to allow primary closure of the heel ulcer. D, finished result. E, incision in the middle of the medial side of a clawed toe curving dorsally towards the metatarsal head. F, the flexor tendon divided distally, and G, re-attached proximal to the pip joint onto the extensor tendon. H, if there is severe *cavus*, make a small incision over the attachment of the plantar fascia to the calcaneus and divide the tissue until you can flatten the foot. I, if the metatarsal head protrudes, and the clawed toes are immobile, remove the head and divide the extensor tendon and re-attach it proximally on the dorsum of the metatarsal. *Take care not to damage the plantar skin if it is still viable.* Fix the phalanges with a K-wire for 6wks to keep them straight. You may transfer the flexor tendon as in G if it makes a bowstring.
After Brand P. Insensitive Feet, A Practical Handbook on Foot Problems in Leprosy, Leprosy Mission International. F,G,H,I, after Apley AG, Solomon S. System of Orthopaedics & Fractures Butterworth 6th ed 1982 p.315. E, kindly contributed by Grace Warren.

(3) TENDON TRANSFERS FOR CLAW TOES (GRADE 2.4)

This operation allows the toes to take more part in weight-bearing, and so protects the metatarsal heads. *It is not applicable where toe joints are fixed.*

INDICATIONS
Mobile clawed toes.

METHOD
Using a tourniquet, incise along the midline of the medial side of the middle and proximal phalanges of the toe whose tendon you want to transfer. Proximally, curve the incision dorsally to reach the dorsum of the foot at the distal end of the web (32-29E). Find the long flexor at the dip joint. Hold it in forceps, and cut it distally (32-29F). Cut the flexor sheath back to the middle of the proximal phalanx.

Lift the skin and soft tissue off the dorsum of the proximal phalanx and pip joint, and transfer the long flexor tendon so that it runs diagonally across the proximal phalanx, and reaches the long extensor tendon of that toe, and attach it there onto the long extensor tendon, proximal to the pip joint (32-29G). (Transferring the *flexor digitorum longus* at this level will keep it as a flexor of the mtp joint, but makes it an extensor of the pip and dip joints). Close the skin with monofilament. Splint the foot on a flat board for 6wks, and prevent walking.

If there is severe *cavus*, make an incision where the plantar fascia attaches the calcaneum, and divide the tissues at this point (the Steindler operation), so that you can get the foot flat. Close the incision with a few sutures. *Make sure the patient does not walk on the wound till it is well healed.*

(4) METATARSAL OSTEOTOMY, INTERNAL FIXATION AND TENDON TRANSFER FOR CLAW TOES (GRADE 2.4)

INDICATIONS
(i) Stiff, clawed toes (especially >1)
(ii) Ulcers under the metatarsal heads.

Aim to reduce the scarred area, by shortening the metatarsals of one or all of the toes, so bringing the toes down to take some weight. Keep all incisions dorsal, and aim for a mobile pseudarthrosis, not an ankylosis. *Sepsis is not a contraindication*, if you leave the dorsal wound open and pack it, but try to get the operation site as clean as you can.

METHOD
Over every stiff toe make a dorsal incision which is long enough for you to see the mtp joint, and 2cm of the metatarsal. Elevate the periosteum, and remove the metatarsal head with bone nibblers or cutters. *Take care to preserve all the viable plantar skin*. Smooth the remaining shaft with a small bone file or nibbler. You should now be able to straighten the toe; if it is still dorsiflexed, remove a little more metatarsal. If there is much scarring under the metatarsal heads, consider removing all of them.

Don't leave one metatarsal obviously longer than the others. Excise any ulcers on the sole, as above, and close them when they are clean.

Avoid damaging the proximal phalanges. If you can find any *flexor digitorum longus* tendons, release them distally and anchor them over the proximal phalanges, as in the claw-toe method above.

For each toe, cut the branches of *extensor digitorum longus* and *brevis* and re-attach the proximal cut end to the metatarsal. If the *flexor digitorum longus* tendons cause a bowstring effect, release them distally and anchor them over the proximal phalanges, as above.

Splint the toes straight by inserting a K-wire through the distal toe pulp for 6wks.

If there is the slightest hint of infection, keep the wounds open and pack them daily till they are clean (34.9). *Make sure the patient does not walk on the wound till it is well healed.* After all the above procedures, try to prevent walking for at least 6wks. If absolutely necessary, use a walking cast, with the ankle in good dorsiflexion, and with sufficient plantar protection to stop trauma to the healing area. Leave the end of the granulating foot protruding for dressings.

If there is marked osteoporosis, apply a walking cast for 2-5 months to allow the damaged bones to recalcify, as they will do when infection is controlled. The bone may still look osteoporotic on a radiograph, but, provided walking resumes gradually, it should recalcify without breaking.

If you are operating on the head of the 1st or 5th metatarsal, do it in the same way. Make an incision on the medial or lateral side of the foot, but make sure there is enough width in the skin bridge to prevent it necrosing.

If the soft tissue under the metatarsal heads has become so scarred that it constantly re-ulcerates, remove all the metatarsal heads through dorsal incisions.

If the foot has become shortened, the toes may remain projecting, and make it difficult to fit a shoe, or they may be subject to excessive pressure. If so, amputate them (35-24).

If the dip joints of the toes only are fixed, or they have repeated ulceration, amputate them (35-24).

If the foot is chronically scarred and ulcerated, and parts of all the toes are lost, but there is good sole tissue proximally, perform a transmetatarsal amputation (35-23).

CAUTION! Foot operations leaving shorter stumps are prone to develop complications.

If the heel pad has some sensation and a good prosthetist is available, consider a Syme's amputation (35-22): this is, however, too short and too small to be used for weight-bearing unless you can provide a good elephant boot.

If there is a foot drop which is a great disability, it is also a treatable one, whatever its cause:
(i) If there is a strong *tibialis posterior* and *gastrocnemius*, and a mobile ankle, it is possible to transfer the *tibialis posterior* tendon.
(ii) If surgery is impractical, you can fit a toe-raising spring (32-22J), if necessary made with canvas or plastic straps, and using the rubber from an inner tube as the 'spring' or callipers (32-13), which will need careful fitting on an anaesthetic limb, if they are not to cause friction burns.

When the lateral popliteal nerve is paralyzed, dorsiflexion of the ankle is impossible, so that walking is liable to injure the lateral side of the foot, the toes, and the ball of the foot. Severe ulcers and marked deformity may follow.

Before referring a patient for reconstructive surgery, make sure you can provide the necessary physiotherapy. *Tibialis posterior* and *gastrocnemius* are normally used together in walking. An important part of physiotherapy is education to separate these actions.

N.B. Tibialis posterior transfer is quite different from *tibialis anterior* transfer for relapsed club foot (32.10).

Also, check the power of:
(i) The *tibialis posterior*. Test inversion of the foot against resistance (move it medially). The only other inverter is *tibialis anterior*, which is usually powerless or very weak in patients needing this transfer.
(ii) The peroneal muscles. Test eversion of the foot, and feel the peroneal tendons contracting behind the lateral malleolus (if they are strong, you should not sacrifice them).
For surgery to succeed, there needs to be a strong *tibialis posterior* and *gastrocnemius*, and a mobile ankle.

In leprosy *all* these conditions must apply:
(i) the leprosy must have been controlled, and there must have been no reaction for at least 6 months,
(ii) the lateral popliteal nerve should have shown no sign of improving after 6 months of treatment and the use of a toe-raising spring,
(iii) the power of the *tibialis posterior* must be 4 at least and preferably 4+ or 5 (32.1),
(iv) the patient must have no ulcers or infections,
(v) preferably, he should be skin-smear -ve,
(vi) you must be able passively to dorsiflex the ankle to 15º (with the knee flexed at 90º),
(vii) the ankle must be suitably mobile, so test it like this: flex the knee to 90º. If you cannot passively dorsiflex the ankle beyond 0º, *tendon transfer alone is not enough.* Straighten the knee. If you can passively dorsiflex the ankle to 15º (unusual), a tendon transfer alone is enough. Otherwise, an Achilles tendon lengthening is needed at the same time.

If the ankle is too stiff to dorsiflex without inverting, you will not achieve a good gait. Try to refer for a wedge osteotomy, perhaps with a tendon transfer later.

(5) TENDON TRANSFERS FOR THE TOES

If a foot cannot dorsiflex normally, its toe flexors shorten. If you correct the foot drop, the toes will remain abnormally flexed, unless they are corrected. So the clawed toes also need tendon transfers (32-29F,G), either at the same time as a *tibialis posterior* transfer, or later. *If you fail to do this, walking may continue with the toenails turned under the toes*, which will cause them to ulcerate.

Record the progress of the foot pre-operatively and again after removing the plaster cast, and at regular intervals afterwards. Record the angles of rest, active dorsiflexion, and active plantar flexion with the knee straight, and passive dorsiflexion with the knee at 90º.

SOME CRITICAL DETAILS

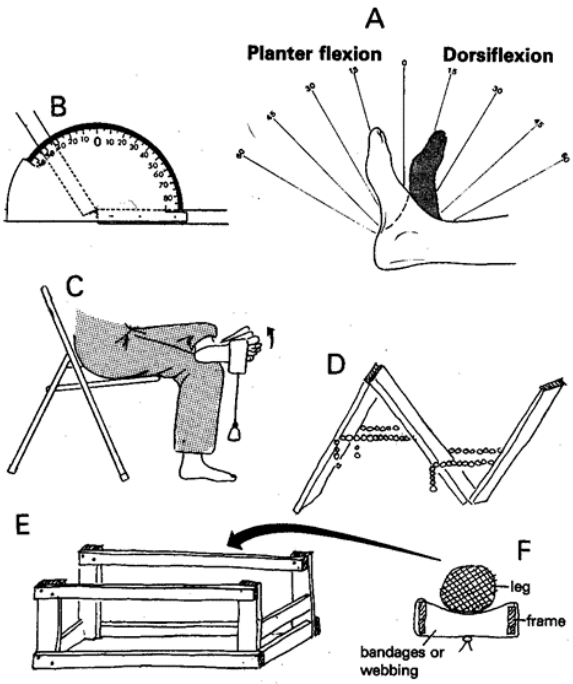


Fig. 32-30 SOME CRITICAL DETAILS.
A, measure the movement of the ankle like this. B, locally made goniometer. Hinge 2 boards together and nail a protractor to one edge. Mark the angles of dorsi- and plantar-flexion on it. C, exercises for *tibialis posterior*. D, locally made foot-drop-positioning frame made in three parts, hinged together, and adjusted by chains. E, frame for a leg rest (24x24x36cm). F, how the leg rests on webbing, cloth, or bandage stretched across the frame.
Kindly contributed by Grace Warren.

32.14 Painful hip or a limp in a child

(a) Congenital dislocation of the hip (CDH)

Congenital dislocation of the hip causes no symptoms at birth, so it has to be diagnosed by screening all newborn babies. The danger is that it may cause premature osteoarthritis in later life. If however you can recognize a baby's dislocated hip at birth, reduce it, and hold it in place with a simple splint; you can usually prevent later complications. If it is not diagnosed at birth, the child may present with a limp (often very mild) when he starts to walk. The leg may then be shortened and the hip unstable. If however the dislocation is bilateral, you will not be able to diagnose shortening, and he may appear to walk normally, although careful observation should show a slight waddle. Baby girls are more likely to dislocate their hips than baby boys.

DIAGNOSIS
In a baby, do Ortolani's test. The child must be relaxed, preferably after a feed. Flex the knees and hold them so that your thumbs are along the medial sides of the thighs, and your fingers are over the trochanters (32-31A). Flex the hips to 90º. Starting from a position in which your thumbs are touching, abduct the hips smoothly and gently (32-31B). If a hip is dislocated or subluxed, you will feel the head of the femur slipping into the acetabulum as you approach full abduction (32-31C).

You may hear a 'clunk', but this is not essential for the test to be +ve. *Restriction of abduction may indicate an irreducible dislocation*. If the test is +ve, *you must not ignore the abnormality*.

CONGENITAL HIP DISLOCATION

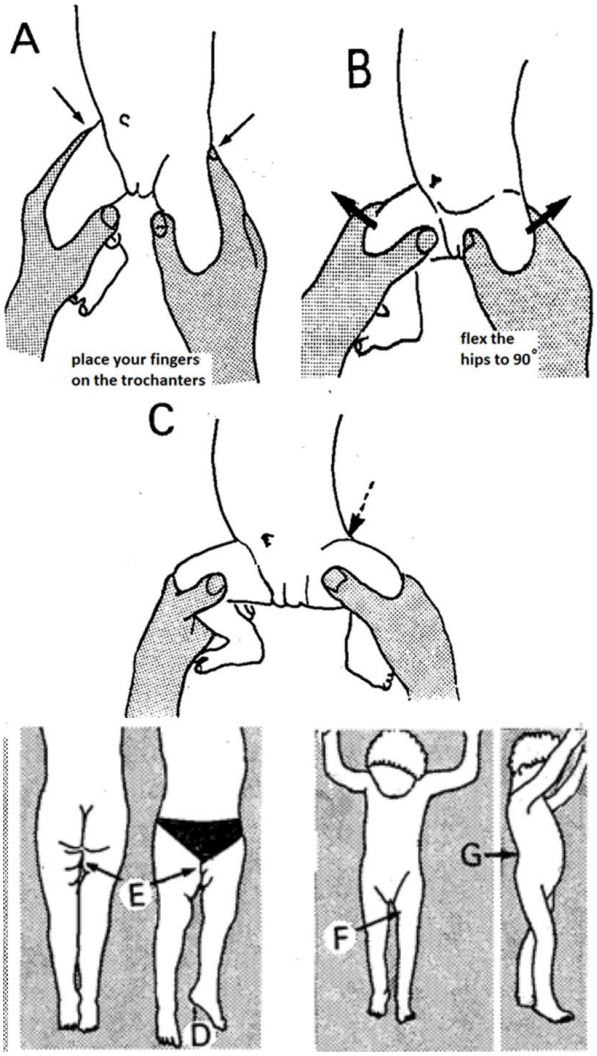


Fig. 32-31 CONGENITAL DISLOCATION OF THE HIP.
Ortolani's test: A, place your fingers on the trochanters. B, flex the hips to 90^0. C, abduct the hip: feel the femoral head slipping into the acetabulum. D, if the child is older, the leg may be slightly shorter, and the hip externally rotated. E, the skin folds of the thigh may be asymmetrical, but this sign is not very reliable. F, if both hips are involved, the perineum is usually widened owing to displacement of the hips. G, if the child has been walking, lumbar lordosis may be increased.
After McRae R, Clinical Orthopaedic Examination, Churchill Livingstone 1988, permission requested.

If the child is older, one leg may be slightly shorter, and the hip externally rotated (32-31D). The skin folds of the thigh may be asymmetrical (32-31E), but this sign is not very reliable. If both hips are involved, the perineum is usually widened due to their displacement (32-31F). If walking has started, the lumbar lordosis may be increased (32-31G).

DIFFERENTIAL DIAGNOSIS OF LATE WALKING includes cerebral impairment and neurological deficits. Confirm CDH radiologically (32-32).

RADIOGRAPHS OF CONGENITAL HIP DISLOCATION

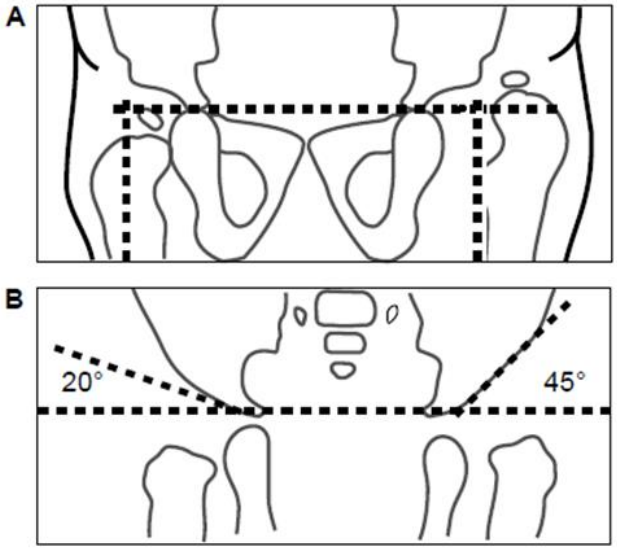


Fig. 32-32 RADIOLOGICAL APPEARANCES OF CDH.
A, draw horizontal (Perkin's) lines through the junction of sacrum, ilium and ischium and vertical lines down from the outer edges of the acetabula: the abnormal femoral head lies lateral to the vertical and above the horizontal line. B, where the acetabular roof is defective, the acetabular angle is increased.
After Apley AG, Solomon L. System of Orthopaedics and Fractures. Butterworth, 1982, p.248 Fig.19.8.

TREATMENT

For a neonate, treat CDH with double nappies which will hold the hips in flexion and abduction. Examine again at 1wk. If the displaced hip has become stable, apply double nappies for a further 3wks, and examine again. If it is still stable, one nappy only is necessary.

If instability persists, the child needs a more substantial splint. Ideally use the von Rosen splint (32-33B). Alternatively, improvise a simple splint with a sheet of stiff polythene, padded round the edges, which passes between the abducted legs over the nappy. The edges of the sheet are held together at either side by 2 pieces of 'velcro' strapping. Apply the splint for 3 months. Then examine the hip again and X-ray it. If the hip is still dislocated, the child may need a subtrochanteric (Salter) osteotomy.

N.B. Over the age of 6yrs, reduction of a dislocated hip needs too much force and will damage it! Don't try to reduce bilateral dislocations after 4yrs because of the risk of asymmetry.

DIFFICULTIES WITH CDH

If you diagnose CDH late (>3 months), skilled surgery is needed and this will be difficult >8yrs. If a good range of movement is particularly important, as in societies where people squat, an unstable mobile hip may be preferable to a stiff one, whatever the risk of later arthritis.

If reduction is difficult or impossible, consider other causes of dislocation:
(1) Partly treated septic or tuberculous arthritis.
(2) ARTHROGRYPOSIS, a rare congenital anomaly, usually affecting all joints, producing contractures without mental deficit, demonstrated by:
(a) the absence of skin creases,
(b) generalized rigidity of the muscles often of all 4 limbs, causing shortening. Defects in at least one other organ are frequent.
If you recognize this condition, *don't attempt reduction*, which may be impossible.

If groin pain and vomiting persist, think of the rare Narath type of femoral hernia which is not visible clinically, but results in early bowel strangulation.

A SIMPLE SPLINT FOR HIP DISLOCATION

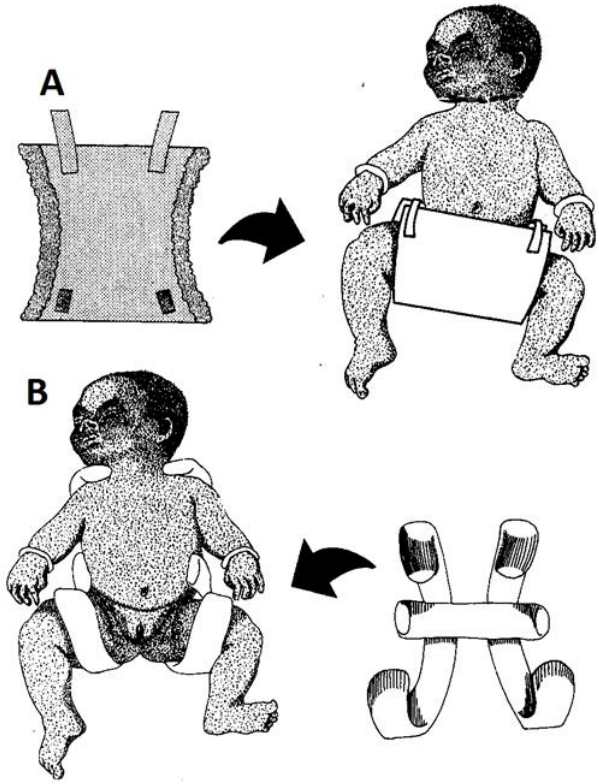


Fig. 32-33 A SPLINT FOR CDH.
A, a simple splint is a sheet of stiff polythene, padded round its edges, which passes between the child's legs over the nappy. B, ideally use the von Rosen splint made of washable malleable padded metal.

(b) Perthes disease (Hip osteochondritis)

Like congenital dislocation of the hip and slipped epiphyses, Perthes disease causes only minor symptoms in childhood, but may cause severe osteoarthritis in later life. It is a very controversial disease.

PERTHES DISEASE

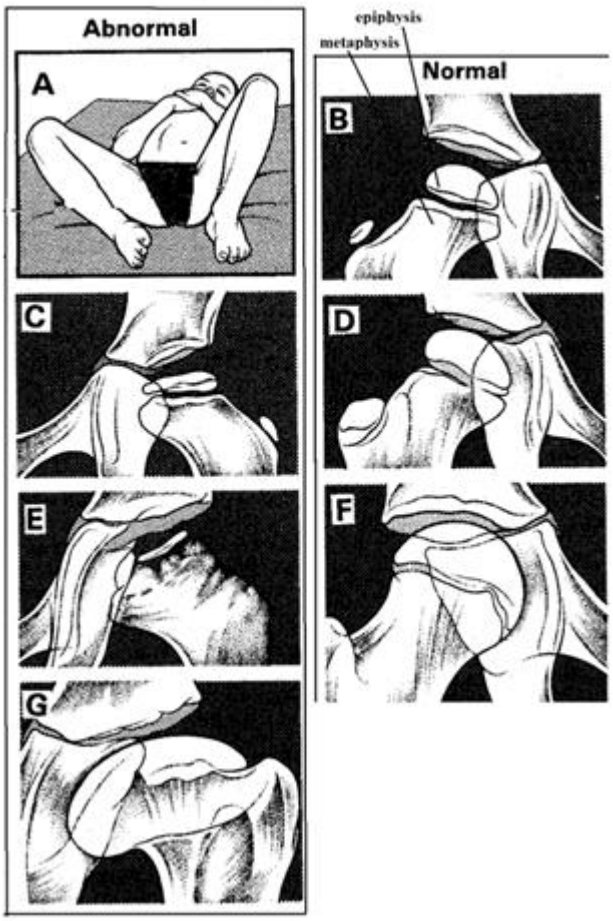


Fig. 32-34 PERTHES DISEASE.
This shows the progression of a patient in Catterall's Group IV. A, in Perthes disease there is always limitation of abduction. B, normal side. C, abnormal side showing the head of the femur is smaller and denser, and the joint space looks increased. D, normal growth. E, patchy fragmentation follows. F, continued normal growth, whilst G, the head becomes wide and flattened on the abnormal side. *After Apley AG, Solomon L. System of Orthopaedics and Fractures. Butterworth, 1982 p.260 Fig.19.26 with kind permission.*

A child with Perthes disease is aged 4-10yrs (occasionally 2-18yrs), and is usually male. If he presents early, he does so with intermittent episodes of pain in the front of the thigh, knee or groin, and a limp; in the early stages he is normal between these episodes. Sometimes there is no limp, but only some minimal abnormality of the gait, such as a tendency to walk with the leg turned inwards. Usually (but not always) all movements of the hip are mildly limited by discomfort rather than by pain, especially abduction and internal rotation.

There may also be some fixed flexion. If movements are limited, the child usually also has spasm, particularly in the *adductor* and *psoas* muscles. The thigh and buttock may be wasted.

There may be vague tenderness around the hip, but the baby is otherwise perfectly well. If presentation is late, after the disease has run its course, the only signs may be a slight loss of the normal range of abduction, extension, and medial rotation of the hip, or he may have no symptoms or signs. However, several present with permanent deformity.

Perthes disease is an avascular necrosis of all or part of the epiphysis of the head of the femur. Essentially, the disease passes through five stages over a period of 2-4yrs:
(1) To begin with, the radiographs are normal.
(2) All or part of the head of the femur looks abnormally dense, which indicates reduced vascularity. The cartilage surrounding it does not die; instead, it continues to enlarge, and makes the joint space appear larger.
(3) The epiphysis may fragment.
(4) New blood vessels gradually grow in, and the epiphysis looks less dense. The epiphysis and metaphysis may soften, so that the metaphysis bends and causes a mild *coxa vara* (the head and the neck of the femur are angled more medially from the shaft).
(5) Eventually, the head returns to its normal density, but it remains flatter, and the neck remains wider than normal.

Avascular necrosis of the femoral head also occurs in later life because of steroid therapy, alcohol abuse and HIV disease.

A child is more likely to get osteoarthritis later in life if the head of the femur flattens. The older he is, and the more misshapen the head, the worse the prognosis. But, even if it is seriously flattened, he will probably not get symptoms until he is middle-aged. Unlike in slipped femoral epiphyses, the involvement of both hips is unusual in Perthes disease (15% of cases, mostly in younger children).

The hope of treatment is to prevent deformity, as long as the epiphysis and underlying metaphysis are soft, which is during the avascular phase and during revascularization. There are various possibilities, either alone or combined, none particularly satisfactory:
(1) Prolonged traction (1-2yrs) produces little benefit, and is quite impractical.
(2) Attempting to avoid weight-bearing by restricting a child's activity is difficult.
(3) Weight-bearing callipers probably don't work and should be avoided.
(4) Salter's Toronto splint is expensive and impractical.
(5) *Surgery has never been proved to be better than non-operative management*.

TESTING FOR SPASM
IN EXTENSION. Lay the child supine, place your hands on the affected thigh, and roll it backwards and forwards (7-17). Compare both sides. If there is no spasm in extension, test it in flexion.
IN FLEXION. Flex the hip and knee to 90º. Rotate the leg inwards and outwards. Rotation is usually more limited than abduction or adduction.
ABDUCTION IN FLEXION is usually limited (32-34A).

RADIOGRAPHS
Take an AP and a lateral view. Abduct the hips, rotate the femurs inwards, and take an AP view to include both hips, so that you can compare them.

CLASSIFICATION AFTER CATTERALL

	GROUP I	GROUP II	GROUP III	GROUP IV
Height of head	No loss	Mild loss	Obvious loss	No height
Sequestration of head	None	½ to ⅔	<¼ normal bone only	Total
Epiphysis	Some lytic areas	Significant increased radiodensity	Marked increased density	Collapsed with mushroom-like protrusion
Metaphysis	Normal	Some radio-lucency	Generalized changes	Destruction

Risk factors:
(1) Lateral subluxation of the head leaving it partly uncovered.
(2) A translucent area in the lateral third of the epiphysis.
(3) Specks of calcium lateral to the epiphysis.
(4) Severe radiolucency of the metaphysis. A fragmented upper femoral epiphysis which appears to be extruding from the acetabulum is a poor prognostic sign.

TREATMENT
Don't intervene for Group I, or Group II and III if the child is <7yrs, unless risk factors are present when a femoral or pelvic osteotomy is needed to contain the femoral head in the acetabulum. Partial weight-bearing with elbow crutches and analgesics for 1yr is useful in the cooperative child: add active hip exercises, folic acid and aggressive antibiotic treatment for *staphylococcus* or *salmonella* infection.

The prognosis in Group IV is bad and *forcing the femoral head into the acetabulum makes things worse*.

(c) Other causes

Hip disease in a child can present as:
(1) A painful hip.
(2) A painful hip and a painful, but otherwise normal, knee, due to referred pain along the obturator nerve.
(3) A painful knee with no pain in the hip.
(4) A painless limp.
Be on the lookout for these 4 signs, which may be due to several diseases. *Don't miss tuberculosis* (32.3) *and septic arthritis* (7.16), because they need early treatment: septic arthritis urgently!

N.B. Don't forget that abdominal and spinal conditions can also cause pain in the hip.
Remember also the possibility of disease near the hip, *e.g.* iliac adenitis, pyomyositis (7.1), appendix abscess (6.16).

Suggesting transient synovitis: no radiographic changes, spontaneous resolution in a few weeks without further episodes. Some cases are viral, notably those due to the parvovirus, and several joints may be involved. There may be a history of mild trauma.

Suggesting septic arthritis/osteomyelitis (7.9,18): an acute onset, often a few hours. The hip is acutely painful, and is immobile in any direction, with general symptoms of acute infection. There are no bony changes for about 2wks. If a radiograph is good, you may see displacement of the fat shadow, or a widened joint space, indicating fluid in the hip joint. Partially treated cases are more difficult to distinguish clinically and by radiography.

Suggesting sickle-cell disease: onset 14-15yrs, especially in a boy, with crises of pain, especially on internal rotation and in the other hip (50% are bilateral) and other parts of the body also, due to infarction, with a +ve sickle-cell test.

Suggesting rheumatic fever: age 5-20yrs. Transient symptoms and the involvement of other joints.

Suggesting tuberculosis (32.3): any age, but common in childhood and adolescence. Bone erosion around the acetabulum (appearing to enlarge upwards), often with damage of the femoral head.

Suggesting rheumatoid arthritis: from childhood to 40yrs (at the onset). The involvement of several joints is usual, although mono-articular disease does occur.

Suggesting reactive arthritis (*e.g.* gonococcal or Reiter's syndrome): urethral discharge, conjunctivitis and/or anterior uveitis. A gonococcal arthritis is usually acute. Reiter's syndrome often follows a chlamydial infection, or *shigella*, *salmonella*, *campylobacter* or *yersinia* diarrhoea.

Suggesting a slipped upper femoral epiphysis: 12-18yrs. Usually a history of an acute onset, sometimes with a fall. Occurs typically in tall, obese, sexually immature children. Radiographs show a wide 'woolly' upper femoral growth plate and extrusion of the femoral head from the acetabulum on a lateral view.

CAUTION!
(1) If you diagnose transient synovitis, follow up the child carefully: some children develop Perthes disease later.
(2) Pain in the knee is often due to hip disease.

A PAINFUL HIP IN A YOUNG CHILD IS INFLAMMATORY, UNTIL PROVED OTHERWISE

32.15 Stenosing tenosynovitis

This is a chronic benign condition, in which the tendons no longer run smoothly in their sheaths. The symptoms depend upon which tendon is involved.

If the patient complains of pain (and sometimes an abnormal prominence) over the radial styloid, which may be worse on extending and/or abducting the thumb, its abductor and short extensor tendons are constricted in their sheaths, as they pass over the groove in the end of the radius (this is de Quervain's disease). They are tender and you can usually feel a thickening in the tendon sheath.

Flexion and adduction of the thumb causes pain over the radial styloid.

If he complains of a 'trigger finger or thumb' so that when he flexes one of the digits it locks, and he cannot extend it again, until he does so passively and forcefully, the flexor tendons are involved. The powerful flexors are able to pull the swollen part of the tendon proximal to the constriction, but the weaker extensors are unable to extend the finger again unaided.

TREATMENT

If you see the disease early, inject the thickened tendon sheath with 0·5ml lidocaine/hydrocortisone mixture using strict antiseptic precautions. Place the point of a very fine needle into the palpable swelling and inject between the tendon sheath and the bone. You will see a wheal appearing on the proximal side of the tendon sheath.

If injection is not successful (50% of cases), apply a tourniquet and get fine instruments. Make a small transverse skin incision. Use a fine tenotomy knife to make a longitudinal incision in the sheath to release the tendon. Leave the sheath open, suture the skin only, and start active movements immediately. The result will be good.

CAUTION!

(1) *Avoid the cutaneous branch of the radial nerve near the radial styloid and the digital nerves*, so look for them immediately you incise the skin.

(2) *Don't try to make the tendon narrower or thinner*.

(3) There may be several slips to the tendon, and thus several compartments in the sheath; make sure each is free. *Review the anatomy before you operate.*

32.16 Ganglions

A ganglion is a round cystic swelling that develops on the back of the wrist, or less commonly on the dorsum of the foot, usually in connection with a tendon sheath, or a joint capsule. Flex the wrist over the edge of a table; this will usually make the fluid in the cyst tense. You may be able to rupture the ganglion by pressing firmly with your thumb; the fluid is then absorbed.

If this fails, repeat it under general anaesthesia.

You may need to use a rubber mallet, or a similar object. The chances of recurrence are no greater than following surgery. Avoid operating because scar tissue will make it virtually impossible to rupture a ganglion easily.

Moreover, if you fail surgically to remove a ganglion completely, it is more likely to recur, and if you dissect too energetically, you may damage a tendon.

Never try to aspirate a ganglion!

If you have to explore for a ganglion, because you cannot rupture it, use a tourniquet, and remove some tendon sheath with the ganglion, trying if possible to keep the cyst of sticky synovial fluid inside the ganglion intact.

Sometimes, a 'ganglion' is actually a synovioma (34.15D), or even a tuberculous nodule! A synovioma gives a characteristic 'snowstorm' appearance on a radiograph.

32.17 Carpal tunnel syndrome

This is one of many nerve entrapment syndromes, which occur more often in women than in men and are often worse during pregnancy and before menstruation. They are the result of pressure on a nerve as it passes through a narrow tunnel. The median nerve passes through the carpal tunnel on the palmar side of the wrist. It causes:

(1) Pain, paraesthesiae and reduced sensation in the distribution of the median nerve (her thumb, the index and the middle finger, and the radial side of the ring finger).

(2) Weakness and wasting of the muscles of the thenar eminence; the hypothenar muscles are spared.

(3) Pain in the wrist, usually referred pain to the lower forearm, and sometimes even pain referred to the elbow and upper arm. The pain is worse at night, and she may get some relief by hanging it out of bed. Tapping over the flexor retinaculum (Tinel's sign) may bring on the symptoms.

A similar syndrome, known as ***meralgia paraesthetica***, affects the lateral cutaneous nerve of the thigh where it passes medial to the anterior superior iliac spine, and so may be entrapped under the inguinal ligament.

CARPAL TUNNEL SYNDROME

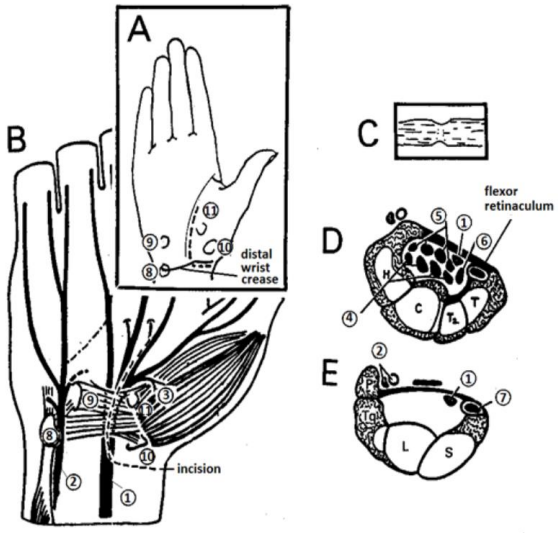


Fig. 32-35 CARPAL TUNNEL SYNDROME.
A,B, landmarks for the incision. C, median nerve with an annular constricting ring round it, caused by pressure from the edge of the flexor retinaculum. D, distal and E, proximal row of carpal bones and the carpal tunnel. H, hamate. C, capitate. T, trapezium. T_2, trapezoid. L, lunate. S, scaphoid. Tq, triquetrum. P, pisiform. (1) median nerve. (2) ulnar nerve and artery. (3) muscular branch of the median nerve. (4) *flexor digitorum profundus* tendons. (5) *flexor digitorum superficialis* tendons. (6) *flexor pollicis longus*. (7) *flexor carpi radialis*. (8) pisiform bone. (9) hook of the hamate. (10) scaphoid tuberosity. (11) trapezoid tuberosity.
After Basmaijan JV (ed) Grant's Method of Anatomy Williams and Wilkins, 9th ed 1975 Figs 32.17,20.

NON-OPERATIVE TREATMENT

(1) Reduce the oedema with hydrochlorothiazide 25-50mg bd during pregnancy. *Don't operate while she is pregnant*, unless the symptoms are severe.

(2) Encourage weight loss.

(3) Inject the carpal tunnel with hydrocortisone suspension 2·5ml and lidocaine 2·5ml.

INDICATIONS FOR SURGERY
(1) Wasting of the muscles of the thenar eminence.
(2) Failure of non-operative treatment.

METHOD (GRADE 2.3)
Apply a tourniquet to produce a bloodless field (3.4).
Make an L-shaped incision over the creases on the front of the wrist (32-35). Incise longitudinally for 4cm in the thenar crease, and then transversely for 2cm in the wrist crease. Divide the flexor retinaculum in the line of the arm. Look for the median nerve, *but don't injure it!*
Opposite the proximal edge of the retinaculum you should see an incomplete annular depression, most marked anteriorly, which is the site of pressure. *Don't close the deep tissues*. Close the skin only with 3/0 monofilament.

POST-OPERATIVELY: Apply a pressure dressing for 48h. Hang the arm up on a support, and watch the circulation in the hand hourly; if there is any problem with this, remove all dressings. Pain should normally be relieved immediately.
If not, *don't delay in reopening the wound!*

32.18 The hands in leprosy

A patient with leprosy can lose feeling in the hands suddenly during a lepra reaction, so that he complains of an immediate numbness, or so slowly that he hardly notices it. When this happens, neglected bruises, blisters, and cuts cause scars that progressively destroy the pulps of the fingers. Painless cigarette burns are a common presentation.

To prevent this happening, he must learn how not to injure himself. Persuade him that it is the injury to the hands which leads to wounds, and not the disease itself. If he fails to care for the fingers, and presents you with a severely disabled hand, there is little you can do, except to maintain such mobility as there is with physiotherapy. Patients are usually able to use their deformed hands quite well, and don't like having their fingers amputated.

Tendon transfers and arthrodeses are sometimes helpful, and you can make a Z-plasty to widen the web of the thumb, but these are not easy operations.

Protect the patient's hands during hard work, either by making sure he wears protective gloves, or by adapting the handles of the tools he uses. He is more likely to consent to wearing gloves than to using modified tools. If he smokes (persuade him not to), he must use a cigarette holder. Make sure that the insensitive hands are soaked and oiled in the same way as the feet (32.11).

If the flexor surface of the finger cracks, *don't let it heal with a short scar* which will be likely to reopen when it is stretched – splint it straight while it heals. Use plaster strengthened with a stiff longitudinal wire, or a short length of stiff plastic hose pipe, cut with a tongue which projects into the palm.

Observe the finger carefully for blueness. Initially, remove splints at night, until you are sure they are not occluding the circulation.

HANDS AT RISK

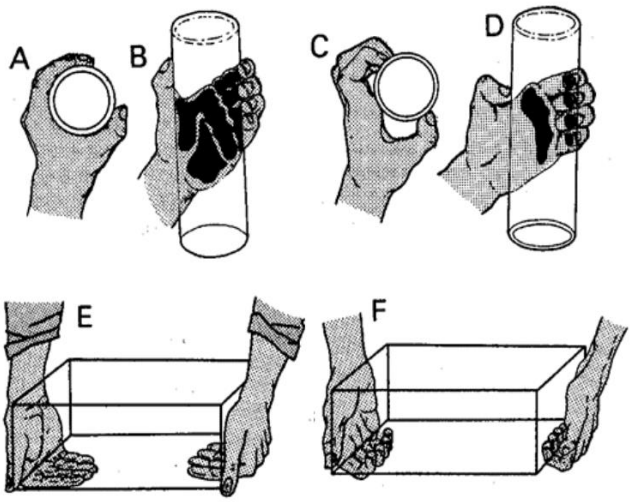


Fig. 32-36 HANDS AT RISK.
The area of skin in contact with the cylinder is black. A,B, when a normal hand bears the weight of a block or a cylinder, most of its surface bears its weight. C,D, when a clawed hand does the same, too much of its weight is borne by the fingertips. E,F, the same phenomenon when a patient lifts a box. This misuse of the fingertips is an important cause of finger absorption.
From Brand PW, Clinical Mechanics of the Hand, Mosby 1985, with kind permission.

If the dorsum of the hand is scarred, so that the mcp joints become hyperextended, severe disability will result. This can happen as the result of a lepra reaction, when a thick sheet of inflammatory tissue scars and perhaps ulcerates. Put the hand through a full range of movement daily during the reaction to keep it mobile. Later, a skilled surgical release may be possible.

If there is one or more severely deformed fingers, such as a terminal phalanx bent to 90º, consider amputation or, better, an arthrodesis with shortening of the bones to allow for the contracted tissue on the front of the joint.

If the little finger is badly deformed, remove it with half its metacarpal. Its absence will hardly be noticed.

If paralysis is acute, splint the hand in the position of function at night, and be sure it is moved by day. Ensure that all the joints of the hand are put through their full range daily, using the exercises (32-37D,E).

If the ulnar nerve is acutely involved, rest the arm in a sling with the elbow at 90º, and put the whole arm through its full range of motion at least once a day.

If the lumbricals are involved, there is danger of development of a claw hand, so teach the exercises described (32-37F,G).

If the median nerve is involved, the thumb web may need stretching. Do this by making a suitable mini-cast to wear at night.

If paralysis is chronic and slowly progressive, recovery is unlikely, so insist on exercises (32-37): a paralyzed hand is more useful if it is mobile rather than stiff, and is less likely to be damaged at work.

INFECTIONS are common. In leprosy patients they usually present late with abscesses (8.1), tenosynovitis (8.12), septic arthritis (8.15), osteomyelitis (8.16), and gangrenous fingers.

Watch for heat and swelling. Tenderness is often absent and fluctuation is too late to be useful. The first complaint may be painful glands in the axilla. The same principles apply as in normal hands (8.1), with one great difference: the pain which prevents a normal person from using the infected hand cannot protect an anaesthetic one. So make sure that a leprosy patient rests an infected hand, and apply a splint to make sure he does. Apply it in the position of safety with the mcp joints flexed, the ip joints almost fully extended, and the thumb abducted, as if holding a tennis ball.

CAUTION! Rest is essential: antibiotics on their own are inadequate.

If infection starts as a macerated skin crease in a paralyzed finger, splint it with a posterior splint in just sufficient extension to open out the finger and expose it to the air. If a posterior splint is difficult, use a palmar one. If there is any discharge, add an antibiotic.

If there is septic tenosynovitis, it is likely to be the result of spread from a pulp infection. Splint the hand in the position of function. If drainage is inadequate, make a further opening in the middle palmar crease (8-4: incision 2).

If you feel rough bone at the base of an ulcer or sinus in the hand and pus oozes from a joint, this is osteomyelitis or septic arthritis.

If you feel rough bone at the bottom of a sinus over the tip of the finger, this is osteomyelitis of the terminal phalanx. If only part of a phalanx is dead, allow dead bone to separate spontaneously. Otherwise, you are likely to open the joint, causing loss of more finger length.

If most of a phalanx is dead, disarticulate the joint and remove the base.

If there is septic arthritis, aim stiffness in a useful position. Splint the hand and fingers as nearly as possible in the position of function (7.17), and use cloxacillin or chloramphenicol. Immobilize the infected joint for at least 4-6wks after the infection is controlled, and the ulcer healed.

Put all the other joints through their full range of movement daily. If splinting one finger is difficult, you may be justified in splinting it with one of its neighbours, depending on their condition. Curette dead bone and granulations, and pack the cavity with hypochlorite ('Eusol'), honey, ghee or sugar to encourage sequestra to discharge and granulations to fill the cavity. An ankylosis usually takes 12wks and a fibrous arthrodesis 6-8wks.

HANDS & FEET IN LEPROSY

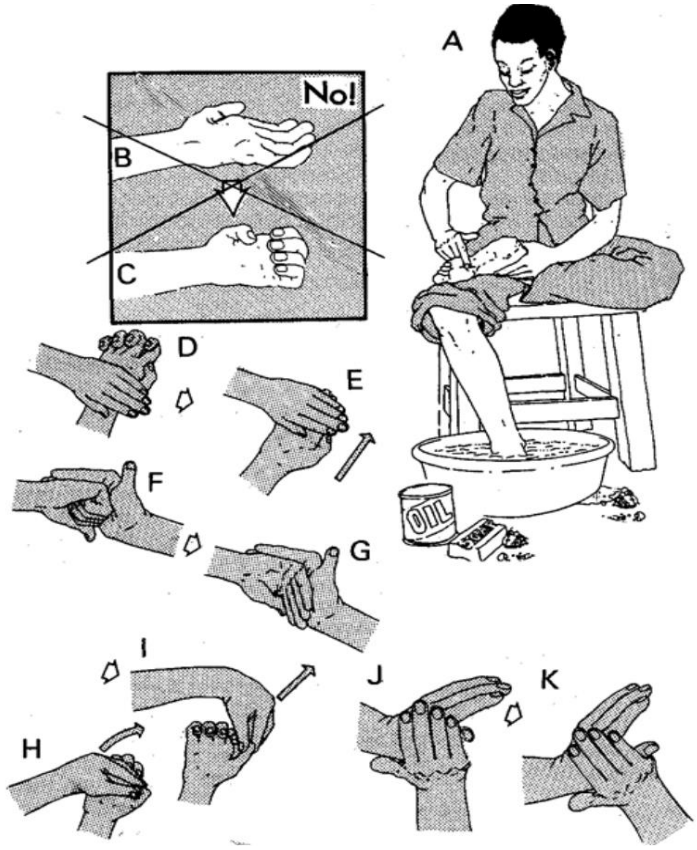


Fig. 32-37 HANDS AND FEET IN LEPROSY.
A, a patient inspecting his anaesthetic feet to find early wounds and 'hot spots'. He is soaking them, and is about to rub them with oil. The exercises shown here are for acute and chronic paralysis, and will prevent a hand like (B) from becoming a stiff claw hand (C) which physiotherapy cannot cure. Instruct the patient like this: D, "Rest the back of your hand on your thigh, or on a table padded by a cloth. E, Use your other hand to rub your fingers as straight as they will go, taking care not to crack any weak skin. F, Cup your knuckle joints in your other palm and keep them firmly bent. G, Then straighten the end two joints of your fingers, as firmly as you can. H, Use your other hand to straighten the end joint of your thumb, as straight as it will go. I, Pull gently and firmly, as if you were trying to lengthen your thumb, but don't pull it backwards. J, Rest the little finger side of your hand on your thigh. Use your other hand to support the back of your thumb firmly (to keep its mcp joint flexed). K, Then straighten the end joint of your thumb as firmly as you can."
After Watson JM, Preventing Disability in Leprosy, Leprosy Mission International, with kind permission.

If there is a grossly swollen hand, with pitting oedema of the dorsum, and obliteration of the concavity of the palm, there is probably a midpalmar space infection (8.9).

If the septic arthritis does not heal, excise the joint. Make a dorsal incision, remove the joint surfaces, and any dead tissues, and splint the joint in a position of function (7.17). Pack the cavity that remains, and allow it to heal by granulation. Keep the joint splinted in the position of function, and wait 12wks till the joints are no longer painful.

32.19 Ingrowing toenail

Ingrowing toenails are unusual in barefooted people. One of the hazards of a shoe is that it may press on the sides of the big toe over a long period, and make the side of the nail grow into the soft tissues and cause pain, inflammation, and the discharge of pus from the nail fold. Carefully cutting away the nail, or placing cotton wool under the nail, may relieve the symptoms, but if this fails, some or all of the nail needs to be removed. If the toenail is not deformed, you can excise a wedge of soft tissue; but if it is grossly deformed (*e.g.* from chronic fungal infection), a more comfortable toe will result if you remove the whole toenail, including its bed. Oral antifungal therapy for 6wks may prevent recurrence.

If the nail grows back in the same way, you can again remove a wedge, including a wedge of the nail bed. A tourniquet gives a bloodless field: you can achieve this with a rubber twisted around the base of the toe. Use a ring block with lidocaine. *Don't do this operation if there is peripheral vascular disease;* use prophylactic antibiotics with diabetics and advise elevation for 24h.

INGROWING TOENAIL

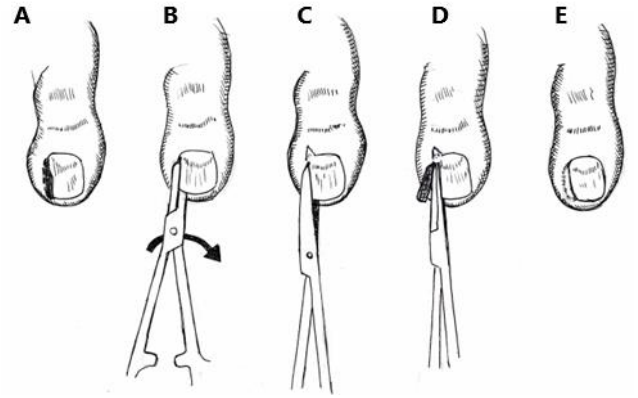


Fig. 32-38 SEGMENTAL TOENAIL MATRIX EXCISION.
A, lift the ingrowing edge off its bed. B, twist it so the matrix comes out from under the skin. C,D, cut this segment off the rest of the nail. E, final result. *There is no need for sutures.*

If the problem is bilateral, perform wedge excisions on both sides or remove the whole nail initially by inserting an elevator underneath it and twisting it off.

If there is infection, lift the skin off the nail gently to drain pus. If this is inadequate to release all the pus, remove a wedge of the toenail. If there is infection under the nail bed (paronychia), you may need to make an incision there.
Then wait 6wks till all signs of sepsis have settled. When sepsis has settled, remove the germinal matrix (the growth plate) of the nail.

SEGMENTAL TOENAIL MATRIX EXCISION
(GRADE 2.1)
Use a tourniquet and ring block. Introduce one blade of a straight artery forceps under the ingrowing nail. Close it over the nail and twist it inwards to pull the matrix out from under its bed. Cut this part off from the remaining nail right to its base. *There is no need for sutures.*

32.20 Malignant bone tumours

Primary tumours of bone are unusual, and have a characteristic age incidence. There are: osteosarcomas, mostly in the 10-25yr age group, chondrosarcomas (15-50yrs), Ewing's tumours (5-25yrs) and giant cell tumours (15-50yrs). Adamantinomas occasionally occur in the jaw, usually the mandible (31.6), and chordomas mainly in the sacrum. Fibrosarcomas arising from the periosteum behave like fibrosarcomas of the soft tissues. Secondary deposits in bones and myelomas are much more common.

Fine needle aspiration cytology is useful for metastatic carcinoma, lymphoma, myeloma and osteosarcoma, but of no real value in benign tumours. Remember a malignant bone tumour may be well vascularized, so use a tourniquet and have blood cross-matched. *Try to avoid the disaster of a pathological fracture or excessive bleeding after a biopsy, or obtaining an unrepresentative sample.* Remember to supply full details as well as radiographic films to the pathologist.

(a) Osteosarcoma

About half of all primary bone sarcomas are osteosarcomas; they occur either in teenagers or, rarely, as a complication of Paget's disease in men over 60yrs. They are aggressive tumours of osteoblasts, and spread either by local infiltration or in the bloodstream to the lungs, often quite early.

An osteosarcoma usually presents as a painful swelling or pathological fracture of the metaphysis of the lower femur (40%), upper tibia (20%), upper humerus (10%), or pelvis (10%).

RADIOGRAPHS
(1) Typically, there is an osteolytic lesion of the metaphysis, which expands the periosteum, and produces a triangle (Codman's triangle), of increased density where the tumour meets the normal shaft. You may see lines of 'sun ray' bone spicules.
(2) A few are small lesions with dense osteosclerosis round a lytic lesion with intramedullary 'fluff'.

DIFFERENTIAL DIAGNOSIS
(1) Very important: early acute osteomyelitis; this causes much pain and shows no bone changes (7.2).
(2) Later, osteomyelitis produces a periosteal reaction.
(3) Chronic osteomyelitis causes dense sclerosis, often with sinuses, and usually involves an extensive area of the shaft.
Other differential diagnoses include:
(4) ordinary fractures (especially if they present late),
(5) stress fractures (fatigue fractures),
(6) simple bone cysts (also hydatid) and exostoses,
(7) metastatic tumours, and other primary bone tumours.

CAUTION! Osteosarcomas may cause fever.
Confirm the diagnosis by cytology or histology.

PROGNOSIS is grim, and there are few long-term survivors. The tumour extends considerably beyond the area of the bone, which is involved clinically, or radiologically. 75% of presentations are with lung metastases, and these occur in 20% within 6 months if you perform an amputation alone. Chemotherapy is of limited value and very high-cost.

MANAGEMENT consists of amputation and chemotherapy: if there are no metastases, amputate if this is practicable, and try to organize a regime of cisplatin and doxorubicin which may allow survival in 60%.

(b) Chondrosarcoma

About 20% of primary bone tumours are chondrosarcomas; they occur in the pelvis (30%), the femur (the lower rather than the upper end, 20%), the ribs (10%), and the skull and facial bones (10%). Most arise *de novo*, but about 20% arise in patients with multiple chondromas, and <5% from patients with a pre-existing chondroma. They are less aggressive than osteosarcomas, and spread by local infiltration; bloodstream spread is late. The histological grading is useful in establishing the likely prognosis.

Presentation is with a bony swelling which is often slightly painful and tender. Pelvic masses are hidden by the overlying tissue, and present late.

RADIOGRAPHS show an area of translucency with trabeculae, multilocular areas of bone destruction, and scattered fluffy areas of calcification. There is usually a surrounding area of soft tissue swelling. Cortical destruction is late, and periosteal reaction is limited.

DIFFERENTIAL DIAGNOSIS includes:
(1) subacute and chronic osteomyelitis,
(2) chondromas (benign tumours of cartilage),
(3) bone cysts (including hydatid, 15.12),
(4) fibrous dysplasia,
(5) other bone tumours.
Confirm the diagnosis by cytology or histology.

PROGNOSIS. Without treatment, 5% of patients survive 5yrs, and none 15yrs. Adequate surgery enables 50% of patients to survive 5yrs and 35% 15yrs.

MANAGEMENT. Amputate through the bone or joint proximal to the tumour. The recurrence rate after adequate excision is low. Early rib lesions have the best prognosis. If you suspect a rib lesion, resect it with at least 5cm of rib on either side, and preferably remove some of the neighbouring ribs. This requires GA and intubation, in case you open the pleura (9.1).

(c) Giant cell tumours

These unusual tumours form about 5% of all primary bone tumours; most arise *de novo*, and a few in Paget's disease of bone. The common sites are the epiphyses around the knee (femur, tibia and fibula, 50%) the lower radius (15%), the pelvis and sacrum (12%), and the maxilla (29.16).

They consist of giant cells (like osteoclasts) and fibroblasts, and are graded histologically as I (low grade), II (intermediate) and III (relatively malignant). First they expand the cortex, and then they spread through it. Lymphatic spread is rare, and distant metastases unusual, but local recurrence after inadequate excision is common.

RADIOGRAPHS. Typically, there is an eccentric osteolytic lesion in the epiphysis which extends into the metaphysis in larger tumours, and has a 'soap bubble' appearance. There is usually little sclerosis of the cortex. A defect in it is a sign that the tumour has penetrated it. In small bones there are non-specific lytic lesions.

DIFFERENTIAL DIAGNOSIS is as for chondrosarcoma.

PROGNOSIS is good because metastases may never occur. After total excision, 70% of patients survive 35yrs. After curettage, the 5, 10, and 35yr survival rates are 45%, 40%, and 35% respectively.

MANAGEMENT. Total excision of the lesion and replacement with bone grafts is necessary.

(d) Ewing's tumour

Ewing's tumour is rare. It consists of densely packed small round cells. It commonly arises in the diaphysis of a long bone: the femur (20%), the tibia (20%), the humerus (10%), and the pelvis (20%). The patient presents with:
(1) a moderately painful, tender, warm, bony swelling, mild fever, and a leucocytosis,
(2) a pathological fracture. There is a 30% chance that he already has widespread metastases in the other bones.

RADIOGRAPHS show a patchy osteoporosis, which is either 'moth eaten' or has defined lacunae. There is usually a periosteal reaction (typically an 'onion skin' appearance) in the intermediate stage. This is not present at first, and disappears as the tumour expands.

DIFFERENTIAL DIAGNOSIS
(1) Subacute and chronic osteomyelitis,
(2) Metastatic neuroblastoma,
(3) Non-Hodgkin's lymphoma in bone,
(4) Fibrous dysplasia.
You must confirm the diagnosis by biopsy.

PROGNOSIS. Untreated, almost no patient survives 5yrs.

MANAGEMENT. Best results are achieved by surgery, chemo- and radiotherapy combined.

(e) Multiple myeloma (Myelomatosis)

This malignant tumour of plasma cells is more common than the primary tumours of bone, and causes widespread osteolytic lesions in any bone, particularly the vertebrae, pelvis, ribs, and skull. When extraosseous lesions occur, they are usually formed by tumour growing from a bone.

The patient, male or female, usually between 40 and 70yrs, presents with bone pain, especially in the back (75%), anaemia (50%), ill health, and loss of weight. He may also have renal impairment, hypercalcaemia, and decreased resistance to infection. In practice, the diagnosis is difficult, because of the non-specific nature of the presenting symptoms.

RADIOGRAPHS may show well-defined osteolytic lesions, usually without cortical thickening or sclerosis, but sclerotic lesions can occur, especially after treatment. Sometimes, there are no discrete bony lesions.

SPECIAL TESTS
(1) Bence-Jones protein is present in the urine of 50% of cases (this precipitates during heating, and dissolves again near boiling point).
(2) Increased immunoglobulins in the blood (95%).
(3) The alkaline phosphatase is nearly always normal, the prothrombin index is increased, and the ESR greatly so.
(4) Bone marrow aspiration and biopsy confirm the diagnosis, and show many abnormally large plasma cells, with poorly defined chromatin. Take a core specimen in addition to aspirating it, because tumour cells are usually in clumps.

DIFFERENTIAL DIAGNOSIS includes:
(1) senile osteoporosis (especially when this produces kyphosis),
(2) carcinomatosis of bone,
(3) myelofibrosis.

PROGNOSIS. Untreated, most patients die in 6 months to 3yrs. Melphalan or cyclophosphamide with prednisone increase the average survival from 17-52 months.

MANAGEMENT
Treat anaemia by transfusion. Treat infection of the chest and urinary tract. Maintain good urine output by encouraging a high fluid intake. Decide if chemotherapy is possible, or worthwhile, in relation to other problems. You may also need to treat pathological fractures, paraplegia from spinal deposits, amyloidosis and hypercalcaemia.

If there appears to be only one tumour (solitary myeloma), you will probably find other deposits if you look hard enough. If there really is only one deposit, and it is affecting vital structures, remove it, if you can, and add chemotherapy. Otherwise, manage it like multiple myeloma.

32.21 Other orthopaedic problems

(a) Problems in young children

If a child is born with an extra digit (common and often bilateral), it usually consists of skin and subcutaneous tissue only. If so, tie cotton tightly round its base; it will soon necrose and fall off. If it is larger and contains bone, leave it for six months, when anaesthesia will be safer, and perform a formal amputation (35.4). For a true double thumb (with functioning joints in each half), perform a hemisection, leaving the most appropriate part. Remember to examine all 4 limbs for extra digits, and look out for kidney anomalies, often associated.

AMNIOTIC BANDS

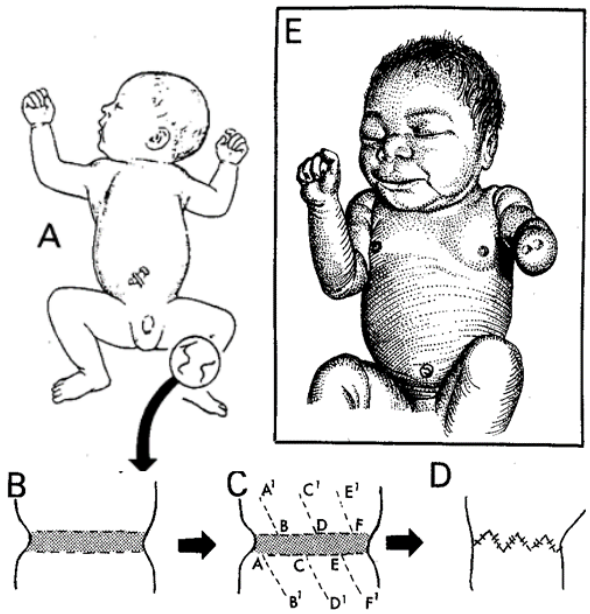


Fig. 32-39 AMNIOTIC BANDS sometimes amputate a limb in utero, as in the neonate E. If they merely cause a constriction (A), excise the constricted area (B), plan multiple small flaps (C) and perform a Z-plasty (D).
Kindly contributed by Jim Thornton.

If there are congenital constrictions of one or more limbs (rare), they are probably due to compression by AMNIOTIC BANDS. These are like adhesions in the peritoneal cavity. A scar is formed which leads to amputation *in utero* (32-39E), or to circumferential constriction of a limb. The limb may become ischaemic, because the constricting tissue does not grow.

Apply a tourniquet <150mm Hg and for <30mins. Excise the lesion down to normal tissue (usually, only the skin and subcutaneous tissue are involved) (32-39B). Close the defect with multiple Z-plasties (34.2). Bring A to A^1 and B to B^1, etc. (32-39B,C,D).
If you join the skin edges side to side, the constriction is more likely to recur.

If fingers are fused together (SYNDACTYLY, fairly common), usually several fingers are involved. The hand may be quite functional especially as an infant.
Don't try to separate them with straight cuts through the webs, because a severe flexion contracture will follow. At 2-3yrs of age, multiple Z-plasties, an inverted 'V' procedure for the web, and skin grafts for the defects are indicated. This is difficult surgery.

The importance of doing this depends on:
(1) how many fingers are involved,
(2) which fingers are involved,
(3) the skill of the surgeon.
A web between the index and middle fingers is more serious than one between the ring and little fingers.

If the toes are fused together, leave them alone.

If the legs are folded in ≤50º of hyperextension (*genu recurvatum*), flex them to 45º and hold them there with plaster backslabs for 3wks. Normal growth without any disability will probably ensue. Occasionally, this is due to a true congenital contracture of the quadriceps which needs surgery.

(b) Problems in older children

If a young adult complains of severe hip pain without signs of fever or inflammation, think of avascular necrosis of the femoral head, which occurs with HIV, steroid injections, alcohol and certain medications.

Severe cases may benefit from a femoral osteotomy to avoid pressure of the upper end of the femur against the acetabulum. Otherwise, look on a radiograph for a flattened femoral head or bony protuberance of the acetabulum which prevents full hip abduction.

If there is a bony outgrowth on the metaphysis, which also has a marrow cavity and a normal bony structure, this is an EXOSTOSIS. There may be one, or many (diaphyseal aclasis). If possible, leave it until growing has stopped, unless it is in an awkward place and is causing disability. Then chip it off with an osteotome.

If you have to remove a prominence before growth has stopped, *take care not to damage the epiphyseal line*. Otherwise, a severe growth deformity may result.

If a bone cyst develops, usually in a child in the shaft of the humerus or femur, the possibilities include a benign bone cyst, fibrous dysplasia, benign osteoblastoma, non-osteogenic fibroma, enchondroma, Brodie's abscess (7-2A), and tuberculosis. If it is benign, it is probably a BENIGN BONE CYST, or an area of fibrous dysplasia. *Aim to avoid a pathological fracture*. The cyst may need to be opened, scraped out, and filled with a cancellous bone graft, if it does not resorb spontaneously.

If the bone fractures across a small cyst, it will probably heal spontaneously.

(c) Epicondylitis (Tennis elbow)

This is a common condition in people who use the extensor muscles of their forearms vigorously, and not only in tennis players. It is caused by minute tears in the origin of the forearm extensor muscles. The patient complains of pain just distal to the lateral epicondyle of the humerus, without any history of trauma. The pain is worse when you press over the radio-humeral joint during pronation and supination. It lasts for months, or years, and may eventually disappear spontaneously. If it is debilitating, treat by injection of hydrocortisone suspension 2·5ml with an equal volume of 2% lidocaine into the tender area. One injection has an 80% chance of success, and a second one 2-3wks later another 10%. Take very careful aseptic precautions, and *don't use >3 injections*. If the disability is severe, a muscle slide on the extensor origin is necessary.

33 Paediatric surgery

33.1 Surgery in children, infants and neonates

There is an increasing proportion of children in LMICs. The number of specialized paediatric surgeons available in Europe is c.1:50,000 population; in South Africa 1:2,669,000, and elsewhere in Africa much less. These figures reflect the fact that in Africa well over 50% of the population are children.

More relevantly, there are c.500 live births per year per European paediatric surgeon; in South Africa over 35,000. So the specialist in Europe will wait 6yrs to see a case of oesophageal atresia (c.1:3,000 live births), whereas the specialist in South Africa sees one about every month! Taking this argument further, it is obvious that every child needing surgery cannot hope, in the poorer parts of the world, to be treated by a specialist. This is why we describe the simpler procedures here for the non-specialist.

CHILDREN ARE NOT SMALL ADULTS

You may be accustomed to operating on adults but find the child patient, especially the neonate, an unfamiliar and intimidating prospect. You may find also you have to pay special attention to the concerns of the parents and family.

"TO TREAT AN ADULT AS A CHILD
IS NO MISDEMEANOUR;
BUT TO TREAT A CHILD AS AN ADULT
MOST OFTEN ENDS IN DISASTER"

Children differ from adults in every anatomical, physiological, pathological or psychological sense. They cannot handle fluids, blood sugar, electrolyte changes and heat like adults. Their fluid requirements are different and their anaesthetic requirements likewise. Neonates tolerate fluid and electrolyte loss particularly badly: they don't have the ability to retain sodium and water post-operatively or concentrate urine like adults, and they also easily become cold, dehydrated and hypoglycaemic. They bleed easily and have little physiological reserve, so they can deteriorate quickly, and don't tolerate delays in treatment (*e.g.* referral to a distant hospital).

Specific paediatric surgical problems are described in this chapter; other important aspects of paediatric surgery are described elsewhere (consult the list at the end of this chapter).

Trauma, which covers fractures, burns, snake bites, and other violence including sexual abuse, is covered in Volume 2.

Perioperative management

(1) POSITIONING AND WARMING
Whenever possible, keep the child next to his mother or nearest relative. If he is old enough, try to explain what is happening and don't lie by saying things won't hurt when they will! Wherever possible, let a neonate breastfeed; otherwise use glucose on a stick, finger or dummy to pacify him.

If you need to perform an invasive procedure, wrap the child in a warm blanket to immobilize him safely and to prevent him from fighting and kicking you! Prepare what you need beforehand, and get the mother and a nurse to assist you.

Make sure the theatre is warm. Place a child on a well-padded cross made of two splints, and bandage the arms and legs to it. Cover the rest of the body except for the part to be operated with cotton wool or commercially produced silver foil. Use warmed solutions for preparation, infusion and washouts.

(2) INTRAVENOUS LINES. An infant requiring GA should have a good IV access, namely, in the antecubital fossa, scalp, or neck. *Avoid using a tourniquet to bring up a vein*: the pressure of a finger in a small child suffices. You rarely need to make a cut-down, but may need a central venous line preferably using the subclavian route. *Never attempt this twice on the same side without checking that no iatrogenic pneumothorax has developed*. If you are unable to find a suitable vein to cannulate, try in the OT under good light and with the child sedated. Use ultrasound to help you, if possible.

(3) UMBILICAL VEIN CANNULATION is a good and reliable option; *avoid cannulating the femoral vein in children wearing nappies* because of the high risk of infection. If available, use ultrasound to detect veins suitable for cannulation and to facilitate insertion.

(4) INTRA-OSSEOUS CANNULATION into the tibia is a fast and reliable route in children of all ages. *Take care to avoid the epiphyseal plate.* Remember many substances are toxic to bone marrow as well as venous endothelium and can easily cause thrombosis or even extravasation in neonates. Therefore fix the cannula properly and re-check its position and functioning repeatedly.

(5) FLUIDS. Replace *all the initial fluid deficit* with Ringer's lactate or 0·9% saline during the 3-6h of pre-operative preparation time. Prescribe the post-operative fluids yourself. *Don't leave this to the nurses,* and *don't exceed 5ml/kg/h* unless the fluid deficit is uncorrected. Where possible, provide the fluid needed as half-strength Darrow's solution with 5% dextrose, or Ringer's lactate with 5% dextrose, or half-strength (0·45%) saline with 5% dextrose.

Fluid balance in a neonate is even more critical: take great care he gets enough *but not too much*. A term baby usually needs c.100ml/kg/day (less in the first 48-72h), and a premature baby 120-150ml/kg/day. For a child between 10-20kg, use 80ml/kg; and >20kg, 60ml/kg.

It is best to administer IV fluids as boluses rather than relying on an unreliable or unmonitored infusion.

For major surgery, make sure you monitor post-operative urine output. A child should pass 1-2ml/kg/h, and a neonate 2-4ml/kg/h. *You don't need an indwelling catheter unless you are absolutely sure the urine output will be observed and measured*. A urethral catheter is often not appropriate; so, for a boy, use a condom catheter (Paul's tubing: 27-3) of suitable size; alternatively, use a fine feeding tube. If you do need a urethral catheter, pass it yourself and take the precautions described (27.2), making sure a girl's hips are fully flexed and externally rotated for a good view. Make sure you have a good light, and can see the urethral orifice. In a boy, you might find the foreskin quite sticky with smegma: carefully clean it with sterile water while pulling back the foreskin gently. In girls, spread the labia to expose the vulva: the urethral orifice may be very difficult to see. It might help to push gently in the suprapubic area to cause some urine to come out: watch carefully from where it emerges! If you have accidentally put a catheter in the vagina, leave it there temporarily before trying again *with a new catheter* (to show you which is the wrong passage). Always be very careful, because you can tear the urethra if you use force!

Replace blood with blood ml for ml if you lose >10ml (or less in premature neonates); a child has a blood volume of approximately 75ml/kg, a neonate 85ml/kg and a premature baby up to 100ml/kg. Monitor and replace blood loss with the greatest care (3-1). Weigh all blood-soaked swabs accurately, on a scale borrowed from the pharmacy, if necessary. Try to avoid blood loss by all means! Make sure all neonates get 1mg vitamin K IM or IV pre-operatively.

(6) POTASSIUM. Normal daily maintenance is 1-3mmol/kg. No extra potassium is needed in the 1st 24h of life. If a child is not taking oral fluids by 24h, he needs a potassium supplement. Add 10mmol to 500ml of IV fluid (=20mM K^+). *Don't infuse >10mmol/h or 3mmol/kg/day*. Or, use 5% dextrose in half-strength saline, which contains 18mM K^+. *Potassium replacement can be very dangerous in children* if it is handled incorrectly. *Avoid adding potassium if renal function is disturbed.* If a child becomes drowsy post-operatively (and the glucose is correct), and the bowel becomes silent, suspect ileus, and add more potassium.

(7) SODIUM. Normal maintenance is 2-4mmol/kg/day. No extra sodium is needed in the 1st 24h of life. Replace nasogastric and intestinal losses carefully. A preterm infant may require up to 6mmol/kg/day in the 1st month of life.

(8) CALCIUM. Normal maintenance in the neonate is 2·25-4·5mmol/kg/day, which means 10-20ml/kg of 10% calcium gluconate per day.

(9) NASOGASTRIC TUBE (4.9)
Insert a well-lubricated tube of appropriate size, which has been stiffened by placing it for a short time in the freezer, through the nose with the neck fully flexed. In neonates, you can pass the tube through the mouth.

(10) NUTRITION
Interrupt feeding as little as you can. *Don't starve a child for >4h before an operation*, and restart feeding as soon afterwards as you can. Ask whether he has passed flatus. Listen every 4h for the return of bowel sounds, and note whether he has passed faeces or flatus; these signs show that feeding can start. Bowel sounds alone are not so reliable in children, so you can assess gastric emptying more accurately by aspirating the stomach hourly, before each intake of feed. Adjust the amount of feed tolerated according to the amount aspirated. Proceed with a staged feeding regime: start with ¼ of a normal (pre-operative) feed portion, diluted 1:2 with water; double this volume after 2h and then again after a further 2h, and then give the full undiluted feed after a further 2h. If the child brings up the feed, go back one stage, and try again. Most children are back on feeds 48h post surgery. If a child was starved <4h, and feeds are restarted soon, he is unlikely to be short of energy.

Where nutrition is going to be delayed for some time, you can provide 50% glucose through a central venous line, using it to replace the energy deficit resulting from starvation. Reckon that, if he cannot feed orally for more than 3 days, he needs 1-2g/kg/day. Test the urine and watch for glycosuria and an osmotic diuresis. Alternatively, dilute 25ml of 50% dextrose in 500ml of half-strength Darrow's solution and increase the concentration gradually.

In working out the energy content of various fluids, remember that 1L 10% dextrose contains 1700kJ (=400kcal). A child's daily post-operative energy needs are:

Neonate	**3-10kg**	**10-25kg**	**25-35kg**	**35-60kg**
420-840kJ/kg	250-330kJ/kg	190-270kJ/kg	145-190kJ/kg	125-145kJ/kg
(100-200kcal/kg)	(60-80kcal/kg)	(45-65kcal/kg)	(35-45kcal/kg)	(30-35kcal/kg)

Note that stress, cold, infection, trauma and surgery increase ordinary nutrition requirements; these should be 2-3g/kg protein and 10-15g/kg carbohydrate per day for the neonate.

CAUTION! If a child becomes drowsy, or unconscious, or behaves strangely, suspect hypoglycaemia, or, less commonly, water intoxication, or lack of electrolytes.

If you are operating proximal to the upper jejunum, a jejunostomy at the time of the operation is a good way to re-establish feeding (11.7).

(11) ANAESTHESIA. If you want to use LA on a neonate:
(i) Dilute 1ml of 2% lidocaine to 10ml, and use half of this as the maximum dose.
(ii) Infiltrate the line of the incision with 0·25% bupivacaine, or 0·2% lidocaine. The maximum dose is about 5ml. *Avoid adrenaline in the neonate.*
Beware of using diazepam as pre-medication: its effects are unpredictable and may be paradoxical.
Ketamine is useful for short minor procedures for children >2yrs.

If a neonate requires an urgent operation, operate at 24h after birth, or as soon afterwards as possible. Lung function is poor if you operate before 24h, when the lungs are not yet fully expanded.
Aspirate the stomach, especially if there is pyloric obstruction (33.4) or bowel obstruction (33.2,3).
GA in neonates and small babies requires a skilful anaesthetist.

33.2 Neonatal alimentary tract obstruction

Most babies regurgitate or vomit a few times during the first few days of life, but bile-stained vomiting soon after birth almost always indicates bowel obstruction. When a newborn baby vomits repeatedly, he may have a medical condition such as:
(1) Infection, typically arising from the umbilicus,
(2) Meningitis,
(3) Intracranial haemorrhage.

You must be able to distinguish these from true intestinal obstruction as the medical conditions are often readily treatable, if you diagnose them early.

A child's alimentary tract can obstruct at any level: oesophagus, stomach, duodenum, small bowel or, rarely, colon; but it most often obstructs at the rectum or anus.

Anorectal malformations form a separate group, and present as the failure to pass meconium, combined with abdominal distension, rather than vomiting (33.6).

Congenital atresia or stenosis of a child's small bowel presents as bilious vomiting shortly after birth, and often (but not always) the failure to pass meconium, which may be incomplete and irregular.

A minimum length of small bowel to survive is 25cm with an intact ileocaecal valve and colon, and 40cm without. However, without sophisticated support, some 100cm is probably the survivable limit.

If the obstruction is proximal to the middle of the small bowel, the abdomen does not distend significantly, but if the obstruction is below this point, it does. The distension may be localized or generalized. The child often presents only after 3-5 days, with severe dehydration.

An obstructed bowel is an emergency. Electrolyte and calorie loss affect a baby more dramatically than an adult, so he needs urgent treatment, within a few hours.

If you are a careful, dextrous operator, you may be able to save a few of these children despite not having sophisticated back-up: you will not have time to transfer these patients elsewhere.

2 CAUSES OF NEONATAL OBSTRUCTION

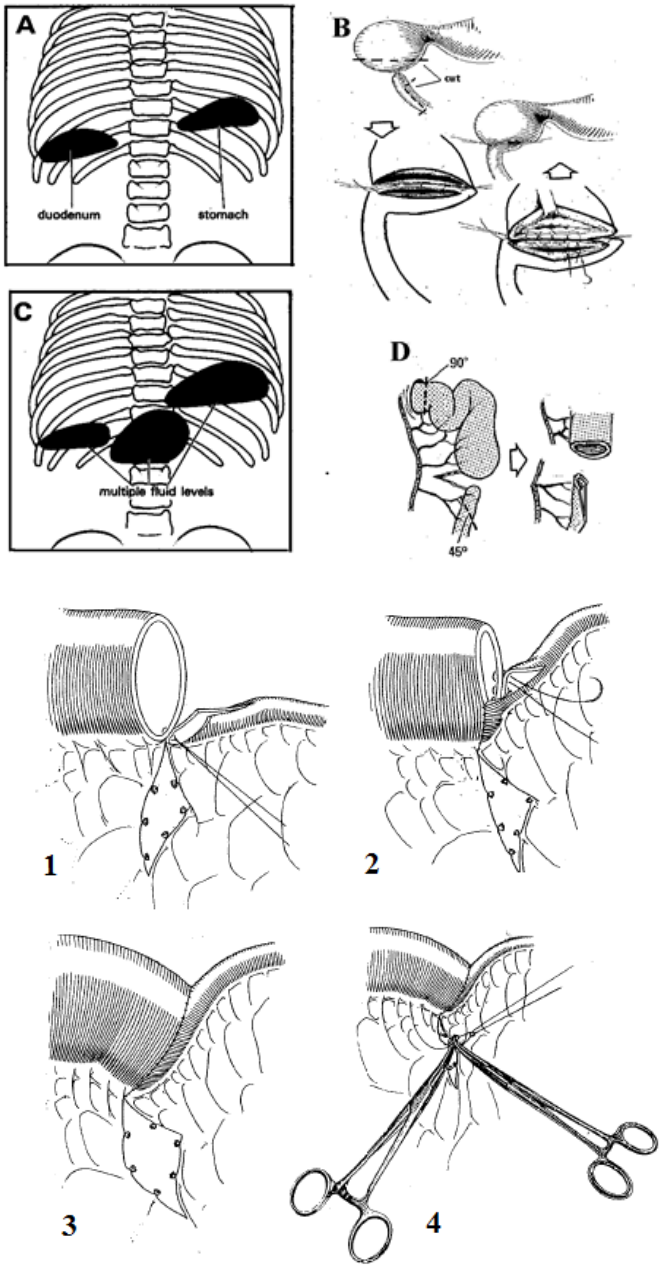


Fig. 33-1 TWO CAUSES OF NEONATAL BOWEL OBSTRUCTION. A, radiograph showing the 'double bubble' of duodenal atresia. B, duodenal atresia relieved by a duodeno-duodenostomy. C, radiograph showing the multiple fluid levels of jejuno-ileal atresia. D, jejuno-ileal atresia showing how the proximal dilated loop is decompressed and resected at 90º, and the narrower distal segment divided at 45º with an added 'cutback'. E1-4, a single layer end-to-back anastomosis.
Partly after Mustard WJ, Ravitch MM (eds). Paediatric Surgery 3rded 1978 Yearbook Medical, with kind permission. E, after Lister J, Irving IM. Neonatal Surgery 3rd ed, Butterworths, 1990 p.462 Fig. 32.8.

Hypertrophic pyloric stenosis (33.4) presents later, as a previously healthy baby 3-6wks old, commonly male, who starts to vomit milk feeds, and *does not have diarrhoea*. This form of obstruction is readily treatable.

(a) Examination
Assess the distension of the abdomen, look for visible peristalsis. Feel for the distinctive mass of a hypertrophic pylorus (33.4). Examine for visible and palpable coils of terminal ileum that feel as if they might be filled with thickened meconium.

CAUTION! Neither the passage of meconium during the first 3 days, nor the absence of distension, excludes obstruction.

(b) Radiographs
After 12h the baby will have swallowed enough air to show air-fluid levels. If possible, take erect straight films before you start aspirating the stomach. The sign of intramural intestinal gas is diagnostic of necrotizing enterocolitis. Look for free air under the diaphragms to indicate a perforation.

Don't use contrast media introduced from above. In oesophageal atresia you may fill the lungs with it! *It is also dangerous in obstruction lower than the jejunum, and may make it worse!*

CAUTION!
(1) The neonatal jejunum, ileum, and colon all have the same smooth outline, and normally contain a few fluid levels. *They can often not be differentiated from each other radiologically*.
(2) *Don't use barium contrast media*.

(c) Differential diagnosis of neonatal bowel obstruction
Suggesting septicaemia: a site of origin for the infection, such as an infected umbilicus; child more ill than you would expect from obstruction alone; a +ve blood culture.

Suggesting meningitis: a stiff neck, headache, fever, irritability, fits and a +ve lumbar puncture.

Suggesting raised intracranial pressure: signs of cerebral irritation, a swollen fontanelle, photophobia; enlargement of the head and papilloedema are late signs.

Suggesting subarachnoid haemorrhage: impaired conscious level; also look for signs of hydrocephalus.

Suggesting some other cause of abdominal distension: a part of the abdomen which is dull to percussion. Causes include distension of the bladder in urethral obstruction, tumours, ascites, congenital cystic kidneys, and hydronephrosis.

Suggesting necrotizing enterocolitis (see below): a toxic lethargic preterm (<2kg) or HIV+ve baby, refusing feeds, with abdominal distension, vomiting and rectal bleeding (>25%), with radiological signs of intramural intestinal gas, free intra-peritoneal gas or gas bubbles in the portal vein.

(d) General management
As soon as you suspect the diagnosis, pass a nasogastric tube, strap it to the face, see that it is aspirated at least every 30mins, and let it decompress into a bag: *aspiration is a common cause of death in neonates*.
Start IV fluids to rehydrate the baby and correct electrolyte losses. Keep him warm. Perform gentle washouts with a rectal tube and prepare for surgery.

(e) Oesophageal atresia presents with regurgitation. It is usually associated with a tracheo-oesophageal fistula. The proximal oesophageal pouch fills with saliva, so there is excessive dribbling. Milk or water is likely to overflow into the trachea. The baby then froths, coughs, and becomes cyanotic with aspiration pneumonitis. In order not to miss a case, you should pass a feeding tube on all neonates who regurgitate, especially underweight babies. Once the baby has an aspiration pneumonia, he is unlikely to survive surgery.

Confirm the diagnosis by passing, as far as it will go, a small (Ch8 in a full term and Ch5 preterm baby) nasogastric tube with a radio-opaque line in it, and then taking an AP chest radiograph. Make the tube firm by putting it beforehand in a freezer: it is then less likely to curl up; but *be very gentle so as not to perforate the oesophagus!*

The radiograph will show the oesophagus ending in a blind pouch; a lateral view is rarely necessary. *You don't need contrast medium*. Leave the tube reaching the blind end of the proximal oesophagus in place and aspirate frequently through it to prevent aspiration of saliva. Major thoracic intervention by an expert is necessary: generally, *if the weight is <2kg, or there is respiratory distress, it is not beneficial to intervene*. Rarely, there is no fistula, or a very narrow one, and the lungs remain clear: in this case, you might be able to bide time by forming a gastrostomy (13.9).

(f) Duodenal atresia or stenosis presents as vomiting on the 1st day of life. The vomit is usually bile-stained, because the obstruction is usually below the ampulla of Vater. The upper abdomen is distended. If the obstruction is above the ampulla of Vater, there will be no bile in the vomit. Erect AP radiographs and ultrasound show a characteristic 'double bubble', with no air (or very little) in the bowel beyond.

The bubble on the right is in the distended duodenal cap, and that on the left is in the stomach. There may also be other abnormal findings including Down's syndrome. A duodeno-duodenostomy or a duodeno-jejunostomy (33.3) will be necessary.

(g) Jejuno-ileal atresia or stenosis may occur at any point in the small bowel. Typically, it presents as bilious vomiting within 24h of birth, slightly later than with duodenal atresia, perhaps 1h after the first breastfeed; but it may be delayed for 2-3 days.

If there is jejunal stenosis (narrowing) rather than atresia (blockage), vomiting may be delayed for as long as 2wks. Obstruction in the upper jejunum is more common.

If the obstruction is low, it presents more slowly, with distension more evident than vomiting. About 50% of these children pass some meconium!

Hydramnios is common in the mother prenatally. Erect AP radiographs show considerable gaseous distension, ending at the site of obstruction, with *several* fluid levels. Unfortunately, by the time that several fluid levels are present, obstruction is advanced. A bowel resection (33.3) is necessary.

(h) Small bowel volvulus may present in older children (12.8) with sudden abdominal pain, distension and shock, or it may present in the 1st week of life, as an acute abdomen with bile-stained vomiting and abdominal distension. Volvulus usually involves the distal small bowel and proximal colon, and is due to a congenital malrotation of the intestine. If Ladd's bands (see below) are responsible, surgery is usually simple, because there is no need to resect bowel, if you managed to detect the obstruction early. *However, strangulation in volvulus develops rapidly*, and then resection is mandatory. This often means an extensive bowel removal, leading to short bowel syndrome.

(i) Necrotizing enterocolitis occurs in preterm (<2kg) or HIV+ve babies within the 2nd and 4th wks of life, and sometimes with duplication of the bowel (33.3). It is the most frequent cause of an acute abdomen in preterm babies, and is related to hypovolaemia, hypothermia, cardiac malformations, and umbilical catheterization. The fragile bowel wall becomes necrotic through a combination of effects of hypoperfusion and bacterial translocation, resulting in the classical presence of gas in the bowel wall. As the disease progresses, the bowel perforates and septicaemia ensues, characterized by erythema of the flanks, a generally tender abdomen. Radiological and ultrasound signs of gas in the bowel wall or in the portal vein, or free in the peritoneal cavity are typical of such deterioration. Look at bowel wall thickness and perfusion if you have Doppler ultrasound.

Start IV gentamicin, ampicillin and metronidazole, and monitor the baby carefully. Decompress free gas in the abdomen by a right hypochondrial puncture. If the condition improves, try to start breastmilk feeds early.

You should try to continue conservative treatment unless:
(1) you see radiological signs of intramural intestinal gas or a persistent single dilated small bowel loop,
(2) you feel a tender abdominal mass,
(3) there is erythema of the abdominal wall,
(4) you aspirate brown infected fluid from the peritoneal cavity,
(5) you see sudden deterioration and progressive acidosis.

At laparotomy, you need to resect the affected bowel and fashion one or even multiple enterostomies (11.5). The mortality is high (up to 50%), and in severe cases, you might get better results by simple lavage and drainage of the abdomen. *Unless you can manage mechanical ventilation and careful intensive care of these babies*, their prognosis is very poor.

(j) Meconium ileus is rare: it occurs in babies with cystic fibrosis (mucoviscidosis) because absence of proteolytic enzymes from the pancreas makes the intestinal content putty-like, and failure of mucus production from the intestinal mucosa leaves it unlubricated.

The baby's sweat has high levels of sodium (>80mM). There is bile-stained vomiting and gross abdominal distension, with visible peristalsis and often palpable compressible bead-like stool in the intestines. Erect AP radiographs rarely show fluid levels, and distended loops of bowel may vary greatly from one part of the abdomen to another. You may be able to detect the hyperechoic intraluminal bowel content on ultrasound. Occasionally the baby is born with a red swollen abdomen, indicating a perforation *in utero* which has walled itself off.

You can often break up the thickened meconium with a warmed gastrografin enema (which may show a thin long micro-colon), but you need an IV infusion running to correct fluid shifts, and *must beware of bowel perforation*. *Never use barium* because this can block the bowel solid! Continue with repeated rectal washouts with warm water. Rarely you might need to perform a washout *via* an ileostomy (11-12G).

33.3 Operating on a neonatal acute abdomen

Anastomosing neonatal bowel is no easy task, but if you are surgically dextrous, are well experienced with adult bowel, and have the right sutures and devoted nurses, you may succeed. You have one advantage: the contents of a neonate's bowel are sterile, so that contamination of the abdominal cavity is less of a hazard than it is in an adult. Whatever the difficulties, you may be sure that if the operation is not done, death is certain.

LAPAROTOMY FOR NEONATAL BOWEL OBSTRUCTION (GRADE 3.5)
Make a transverse muscle-cutting incision (11-1), as this breaks down less often than a midline opening. Use the finest haemostats, and handle the bowel with the greatest care (11.3). Hold it with stay sutures, and *don't use bowel clamps* unless you have vascular 'bulldog or Blalock' clamps (3-2). Cover exposed bowel with moist warm swabs. Examine the whole bowel, because there may be more than one area of obstruction. Inject air into the distal bowel through the wall with a very fine needle and milk it along to see if there are any further sites of stenosis. Once you have opened both limbs of the bowel between stay sutures, insert a small tube and flush both clear of inspissated meconium completely and repeatedly, taking care not to spill the contents. By doing this, you will be able to check for any further areas of stenosis or atresia. Furthermore, in the dilated proximal segment you will note where bowel movement is present and where you can expect recovery. In the distal unused part, irrigation will provide some dilation and lubrication for subsequent passage of stools.

The proximal bowel is hugely distended and the distal bowel collapsed, so a straightforward anastomosis is difficult. Tailor the ends so that their sizes are a little more equal by dividing the distal part at 45˚ (33-1D) and make a cut on the anti-mesenteric border to give it a V-shape (33-1D). Make a single layer of instrument-tied 4/0 or 5/0 long-acting absorbable sutures (33-1E). Gently invert the posterior wall of the bowel, as you insert the first sutures.

If you can do this satisfactorily, inverting the anterior wall should not be difficult. As you go round the corner from the posterior wall of the anastomosis to the anterior, continue to invert the bowel wall. Suture the serosa if appropriate. Always check that the anastomosis is free of tension, watertight and well vascularized.

A child grows, so suture the bowel and the wound with absorbable sutures. Suture everything meticulously.

For DUODENAL ATRESIA, the best operation is a duodeno-duodenostomy. Bring the proximal (dilated) and distal (collapsed) duodenal loops next to each other by carefully separating adhesions to the liver, gallbladder, bile duct and pancreas. Where there is a significant gap between the atretic segments, you can usually mobilize the distal part, together with the duodeno-jejunal junction to the right behind the superior mesenteric vessels. If there is an annular pancreas (very rare), *don't divide it*: mobilize the duodenum enough to make an anastomosis in front of the pancreatic ring.

Aim to make a large (2-3 cm) diamond-shaped side-to-side anastomosis between them (33-1B), by making a transverse incision in the dilated and a longitudinal incision in the collapsed segment. If you encounter an intraluminal web, cut this out carefully leaving a thin rim circumferentially.

Pass a thin, soft, long nasojejunal tube through the anastomosis for feeding post-operatively. Stiff plastic tubes cause problems, so use a soft silicone tube (VP shunt tubes work well). You will also need a nasogastric tube for suction.

Occasionally, you will not be able to perform a duodeno-duodenostomy if the atresia is too distal; then an end-to-side retrocolic duodeno-jejunostomy is the alternative. *Don't fashion a gastrojejunostomy* as this will result in anastomotic peptic ulceration later.

If you cannot do either, fashion a feeding jejunostomy (11.7) to bide time till you can arrange the definitive surgery.

For JEJUNAL ATRESIA, you will have to anastomose the dilated jejunum above the stenosed segment to the collapsed jejunum below it. The blood supply of both ends of the affected bowel is often deficient, so resect it proximally at a point where it is about 1-1·5cm in diameter, *i.e.* normal. Fashion a 'fish-mouth' opening so as to make an 'end-to-back' anastomosis as described above (33-1D,E).

Don't make an end-to-side anastomosis because this results in the blind-loop syndrome (bacterial overgrowth leading to malabsorption).

Make quite sure there are no further stenotic segments by injecting saline into the distal bowel lumen and massaging it into the caecum. If there are (in 20%), try to include them all by doing one resection, if this does not remove excessive bowel. Otherwise make multiple resections or multiple stomas. Decompress the proximal intestine with suction through a Ch12 Foley catheter. *Don't try to milk intestinal contents into the stomach, as they may spill out through the oesophagus into the trachea.*

For VOLVULUS OF THE MIDGUT, when you open the abdomen, you will see distended coils of small bowel, which may be cyanotic and congested, and obscure the right colon. Deliver the small bowel to the surface, and protect it with warm moist packs. Examine the base of the mesentery to see which way it has twisted (usually clockwise). Untwist it (there may be 2 twists), and check that its normal colour returns.

There may be an incomplete malrotation where the caecum is found in the right hypochondrium fixed by Ladd's bands passing across the 2nd and 3rd parts of the duodenum to the lateral abdominal wall. This means the caecum is very close to the duodeno-jejunal flexure, and the base of the mesentery is narrow and so prone to twist. Divide the constricting bands (33-2): there are frequently more than one. Free up the ligament of Treitz, and so straighten the duodenal loop. Then carefully separate all adhesions between loops of bowel, and make sure the duodenum is not kinked, and is patent. Do this by manipulating the stomach contents through into the small bowel. Make sure that the caecum is now resting in the *left* iliac fossa. Remove the appendix attached, because of its subsequent abnormal position.

DIVIDING LADD'S BANDS

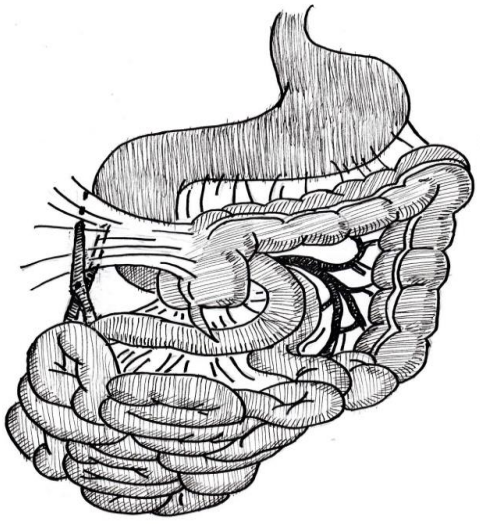


Fig. 33-2 DIVISION OF LADD'S BANDS.
Incomplete malrotation where the caecum lies in the right hypochondrium, tethered by 'bands' to the right lateral abdominal wall; these may obstruct the duodenum.
After Greenfield LJ et al. Surgery. Lipincott 2nd ed 1997 p.2057 Fig 103-30.

Rarely, the malrotation is reversed so that the transverse colon lies behind the mesenteric vessels; usually it is possible to rotate the whole bowel through 360º in an anticlockwise manner to correct this.

If the bowel is not viable, resect the gangrenous part, decompress the proximal bowel and anastomose viable bowel or fashion a stoma. If you are not sure about the viability, place warm sponges over the bowel for 15mins and then reassess it. If you have to resect much bowel, the outlook is poor, because of the consequent short bowel syndrome.
There is nothing to be gained by fixing the caecum or colon in their new positions; this may actually do harm. However, do remove the appendix!

Occasionally, bowel twists around a remnant of the omphalomesenteric (vitelline) duct; divide this and assess the bowel viability as before.

Difficulties with neonatal bowel obstruction

If you find a tube (often solid) of duplicated small bowel, resect both the 'normal' and duplicated segments; *don't try to separate them* as the blood supply of the normal part will be compromised.
Don't drain the duplication into the normal bowel as malignant change may result later on. If the duplication is one large cyst, open it and try to strip its mucosa.

If you find extensive or patchy gangrenous bowel without volvulus, this is neonatal necrotizing enterocolitis (33.2). Resect or exteriorize the gangrenous part, and make a spectacles colostomy (11-14). The prognosis is poor, particularly if there is HIV disease. Resuscitate aggressively, replace electrolyte deficits, correct acidosis and use IV gentamicin, ampicillin and metronidazole.

33.4 Omphalocoele (exomphalos) and gastroschisis

Not uncommonly, a child is born with a defect in the abdominal wall which involves the umbilicus, and leaves the viscera exposed or covered only by a translucent layer. In **hernia into the cord**, there is a fascial opening where bowel protrudes into the umbilicus; in **omphalocoele minor**, there are only a few loops of bowel inside a flimsy sac of peritoneum and amnion; but in **omphalocoele major** (33-3), it may contain most of the abdominal organs, including even the liver. Often there are no other defects, but you should always look for them, especially cardiac anomalies.

The sac of an omphalocoele is thin, and easily ruptures or becomes infected, causing peritonitis. It rarely ruptures during delivery, but then careful management is urgent.

If the umbilical cord appears hollow, a patent vitelline duct is attached (33.4,5). If it is lumpy, there may be a urachal remnant attached. There may be macroglossia and/or gigantism in <10%, and other serious abnormalities associated with the Beckwith-Wiedemann syndrome.

In **gastroschisis**, there is no membrane covering the viscera, which protrude to the *right* of the umbilicus. The stomach and intestines, but not the liver, herniate and are usually grossly thickened and shortened with a leathery appearance. There is rapid heat loss through the exposed intestines. There is often short bowel and sometimes ischaemic loops.

MANAGEMENT
You may have been able to make the diagnosis prenatally by ultrasound. If so, have everything prepared before delivery. As soon as the child is born, examine the baby carefully in a warm environment with sterile gloves.

If the membrane is intact and the viscera are covered, delay surgical intervention. Non-operative treatment is simple but needs particularly good nursing care, and has the disadvantage of not being able to inspect the internal organs.

Clean the sac with an antiseptic, and apply 70% alcohol or 1% gentian violet hourly for the 1st 48h, and then less often as a crust forms which takes about 3wks. The crust will separate from the periphery, as epithelialization takes place. This will then result in a large skin-covered ventral hernia (33-3H) which will need repair later (18.10).

N.B. Don't put dry dressings on an intact omphalocoele: you may tear the thin membrane when you change the dressings. It is best left exposed (under a mosquito net). If, however, you intend to treat the baby as an outpatient, you will need to apply paraffin gauze covered by large padded dressings to avoid trauma to the thin sac.

If the membrane is ruptured or the defect is a gastroschisis, wrap the baby up well, preferably in a large sterile plastic bag encasing the legs with a string tied around the chest (33-4), and *keep him warm*. Start an infusion of warm IV saline, administer ampicillin 10mg/kg and metronidazole 7·5mg/kg IV. Insert a nasogastric tube and suction every 10mins (or with low-grade aspiration), and also pass a rectal tube and perform warm irrigations of the bowel. Prepare theatre.

Don't try to reduce the abdominal contents unless you find an omphalocoele minor: just cover them with a silo, made from a pre-washed *sterilized female condom (without spermicide)*. This should be transparent so you can see what is happening inside, and of the smallest possible size to make a good fit around the exposed viscera.

Place these within the silo gently, taking time for oedema to reduce, making sure that you aspirate the stomach at the same time. Tuck the open end of the silo with its ring into the peritoneal cavity under the abdominal wall skin, and try to fix it under the abdominal wall. Extend the defect by incisions superiorly and inferiorly.

Retract the abdominal wall edges and suspend the silo gently from above the bed and wait for the bowel to reduce spontaneously (usually 5 days), and then wait 24h before removing the silo. The defect will close spontaneously if you pull the umbilical cord to the right over it. Monitor the neonate for respiratory problems from pulling up the diaphragm or increased intra-abdominal pressure, and start feeding as soon as the reduction is complete.

These babies are a serious challenge, and if you have the option of transfer to a specialist centre where the babies can be fed intravenously, do so! *But always keep the baby warm!*

OMPHALOCOELE MAJOR

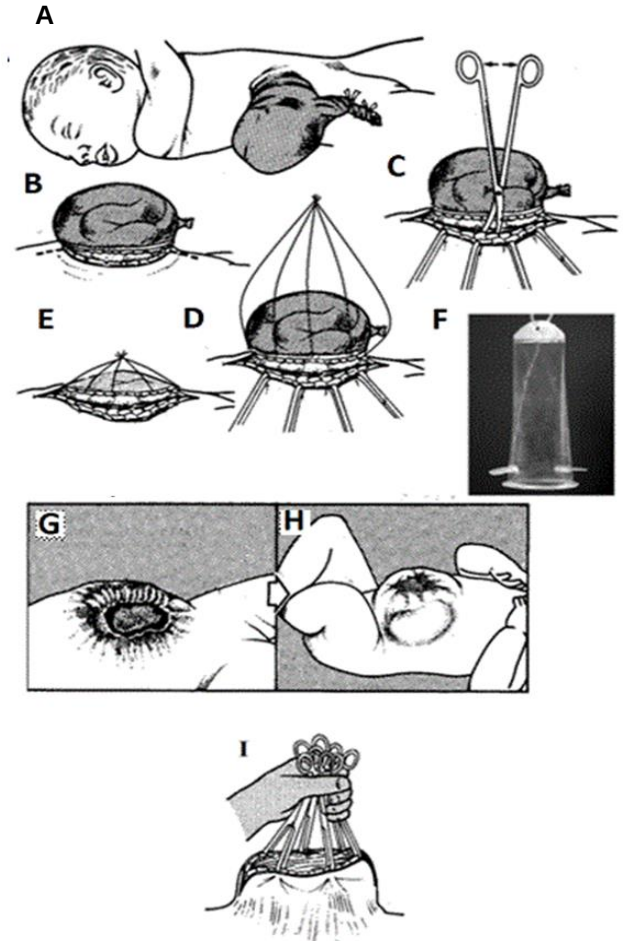


Fig. 33-3 OMPHALOCOELE.
A, this hernia is too large to reduce in a single stage (omphalocoele major); its sac is intact. B, extend the opening superiorly and inferiorly. C, mobilize the edges of the sac. D, suture a silo pouch *(left transparent in this illustration)* **to the edges of the defect. E, reduce the sac in volume gradually over days, by gently twisting the pouch. F, for gastroschisis, a silo made from a female condom with skin tabs attached at the sides to fix under the abdominal wall, works well. G,H, early and late stages in the non-operative treatment with gentian violet. I, when the edges come together readily, close the abdominal wall.**
Partly after Mustard WJ, Ravitch MM (eds). Paediatric Surgery. Yearbook Medical 3rd ed 1978, permission requested.

If there is an omphalocoele minor and you succeed in reducing the contents of the sac into the abdominal cavity with ease, you can proceed to close the defect; but do so only if you have good back-up, because the non-operative method works just as well.

However, operation may be difficult if the liver is adherent to the membrane, if there is a patent vitelline duct, and if reduction of the prolapsed viscera causes respiratory difficulty. You may, moreover, have little chance to do anything about any abnormalities found anyway!

GASTROSCHISIS TREATMENT USING A STERILE SILO

Fig. 33-4 EXPOSED BOWEL IN A NEONATE.
Use this method for ruptured omphalocoele or gastroschisis: enclose the exposed bowel in a sterile silo suspended from above and make sure the baby stays warm (note head and legs covered). If the bowel does not fit in a suspended silo, encase the whole abdomen and legs in a sterile plastic bag.

SURGERY FOR OMPHALOCOELE (GRADE 3.3)

Prepare the skin, including the omphalocoele sac. Tie and divide the cord, as it emerges from the sac, with '0' absorbable suture (monofilament cuts through too easily). Incise the skin ½cm, or less, from the edge of the defect.

At the edge of the sac, find and tie the umbilical arteries infero-laterally in the 5 & 7o'clock positions; tie the umbilical vein superiorly (in the 12o'clock position). Expose the edge of the fascia and peritoneum, and remove a ring of tissue. Excise any tissue of doubtful viability from the sac wall.

Reduce the hernia, and close the defect in three layers. If necessary, hold up the edges of the defect with haemostats. Close the abdomen with long-lasting absorbable sutures and the skin with simple sutures of 4/0 monofilament. Post-operatively, feed with expressed breast milk by nasogastric tube, until the baby is sucking well, usually in 2-3 days.

If you fail to reduce the contents of the sac, or if the sac is ruptured, leave as much of it intact as possible. Find and tie the vessels as above. Excise any tissue of doubtful viability from the wall of the sac, including the stumps of the vessels.
Stretch the abdominal wall with your fingers to allow as much of the viscera to reduce as possible; free any adhesions of the sac from the liver.
As for gastroschisis above, fix up a silo; gently twist this to reduce the visceral content after 5 days, every 2-3 days. When you can reduce the omphalocoele easily, you can remove the bag and close the defect as above.

33.5 Disorders of the omphalomesenteric (vitelline) duct

In foetal life, the intestinal tract and the yolk sac are joined by the omphalomesenteric (vitelline) duct. Remnants of this may persist and present as:
(1) A persistent discharge from the umbilicus which is occasionally faecal.
(2) Gastrointestinal bleeding from ectopic gastric mucosa in a Meckel's diverticulum (33-5I, J).
(3) Bowel obstruction caused by bowel twisting around a persistent vitelline duct (33.3, 33-5F).
(4) Intussusception through a patent vitelline duct (rare). You may occasionally have to resect and anastomose bowel in connection with any of these, or, rarely, with the other abnormalities (33-5).

Difficulties with umbilical discharge

If a child discharges urine from the umbilicus and the urethra, there is a persistent connection to the bladder: a URACHUS (rare). Sometimes urine discharges in a small spurt during micturition. You may demonstrate this using a gentle probe or guide wire, or with contrast. Presentation may be at 1yr or later. *Dissecting out and excising the track does not require a full laparotomy.*

If there is a dirty umbilicus which discharges, smells, and occasionally bleeds, this is an UMBILICAL GRANULOMA (very common), caused by an infected remnant of the cord. Clean it with spirit daily, apply zinc powder, and keep it dry.
Treat an infected umbilicus (omphalitis) in the acute stage with gentamicin, and add metronidazole if it is smelly. *Watch out for signs of necrotizing fasciitis* (6.23), *and beware of bowel evisceration!*

ABNORMALITIES OF THE OMPHALO-MESENTERIC DUCT

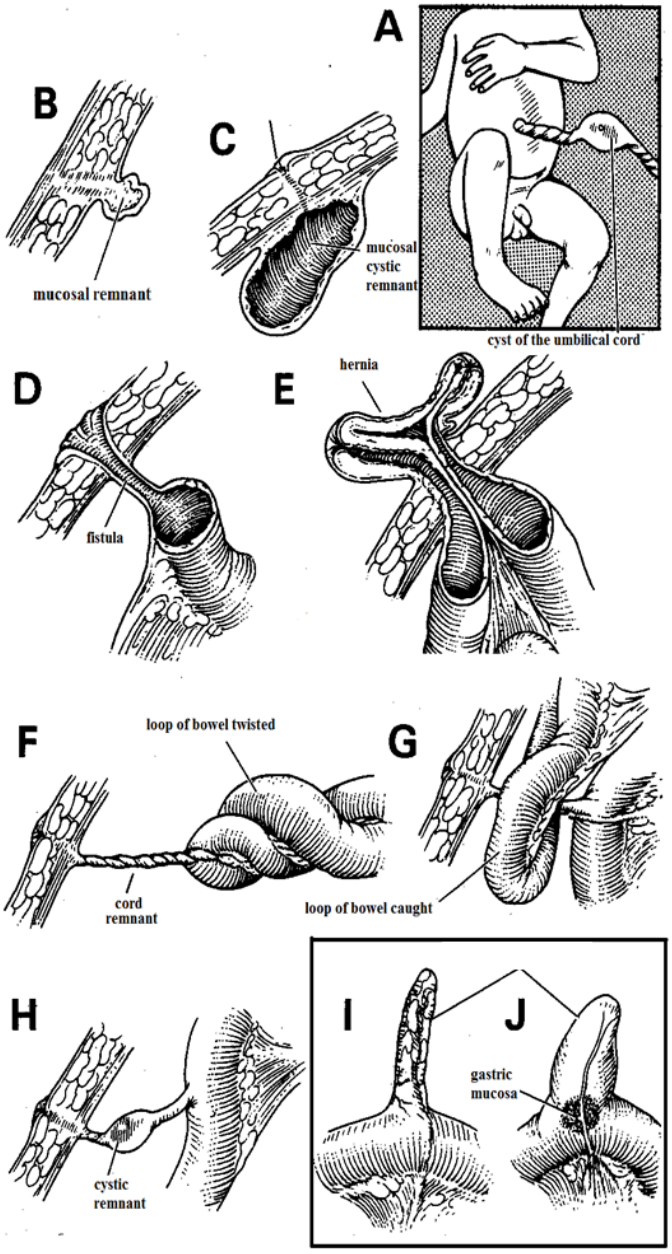


Fig. 33-5 ABNORMALITIES OF THE OMPHALOMESENTERIC (vitelline) DUCT are usually rare.
A, harmless cyst in the cord. B, umbilicus covered with red intestinal mucosa, which may dip down into the abdominal wall. *Don't confuse this with an umbilical granuloma*, which is much more common. C, mucosa-lined cyst communicating with the skin. D, communication between the ileum and the umbilicus. E, bowel herniating through this communication. F, persisting cord around which bowel may twist (33.3), or G, bowel may be caught. H, a solitary cyst within the cord. I, remnants of the duct persisting as Meckel's diverticulum, which may become inflamed. J, some gastric mucosa in the duct ulcerating and bleeding.
Adapted from Mustard WJ & Ravitch MM (eds). Paediatric Surgery, 3rd ed. 1978 Fig.88-17, Yearbook Medical, with kind permission.

33.6 Anorectal malformations

Anorectal malformations are relatively common. The child presents soon after birth with abdominal distension and the failure to pass meconium. Some lesions are incomplete, and present later with difficulty passing faeces, or distension. Around 15-40% of these children have one or more other anomalies, particularly vertebral, cardiac, oesophageal, renal and skeletal. Take time to record your findings carefully.

ANORECTAL MALFORMATIONS

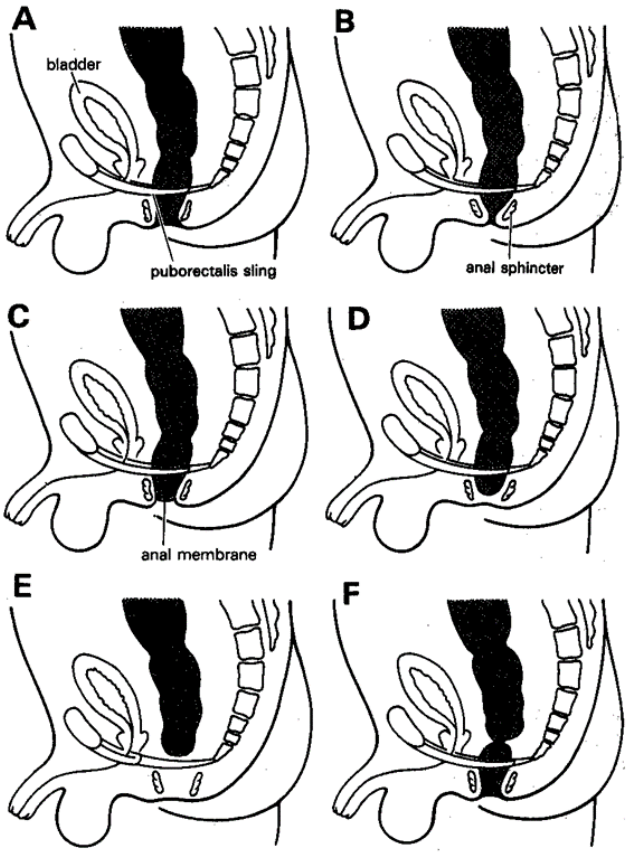


Fig. 33-6 ANORECTAL MALFORMATIONS.
Low lesions (B-D) have rectum extending below the *puborectalis* sling (infra-*levator*), high ones (E,F) remain above it (supra-*levator*). The low lesions are much easier to treat. Function a colostomy for the high lesions.
Adapted from Mustard WJ & Ravitch MM (eds). Paediatric Surgery.Yearbook Medical 3rd ed 1978 Fig. 98-2 with kind permission.

There are many kinds of lesions, but what matters most is whether a child's rectum ends close or far from the skin. If the rectum extends close (<1cm) to the skin (infra-*levator* lesions), you can create an anus, or dilate a stenosed anus relatively easily. But if the rectum ends further from the skin (intra- and supra-*levator* lesions), make a temporary colostomy, and arrange later for the sigmoid colon to be pulled through to the perineum, or an anorectoplasty to be performed. This is difficult surgery, and continence is often not perfect.

The anus or rectum may fail to develop entirely (agenesis), it may partly fail to develop (atresia), or the rectum or anus may be narrowed (stenosis). Agenesis (but not the other lesions) may be combined with fistulae between the rectum and the urinary or genital tracts of either sex. These variables combine to produce a complex series of lesions. Some fistulae are useful, because you may be able to dilate them to make an anus. About 75% of cases are low lesions with fistulae.

(a) Low anorectal lesions:
(1) Anal stenosis or imperforate anal membrane in boys or girls (33-6B,C).
(2) Anocutaneous fistula in boys and girls.
(3) Anovestibular fistula in girls (33-8).

(b) Intermediate anorectal lesions:
(1) Rectobulbar urethral fistula in boys; rectovestibular and rectovaginal fistula in girls.
(2) Anal agenesis without fistula in boys and girls (33-6D).

(c) High anorectal lesions:
(1) Anorectal agenesis with rectoprostatic, urethral or rectovesical fistula in boys; or rectovaginal fistula in girls.
(2) Anorectal agenesis without fistula in boys and girls (33-6E).
(3) Rectal atresia in boys and girls (33-6F). There is a normal-looking anus in the normal place, with low bowel obstruction (no meconium, abdominal distension and only vomiting late in the presentation).
N.B. A contrast enema is safer than probing in this situation.
(4) Cloaca (a combined opening of urethra, vagina and rectum) in girls. This looks like a 'one-hole' perineum.

You should be able to diagnose which kind of lesion a child has from the clinical signs and a simple radiograph (33-7) or ultrasound. The risk in trying to repair an anorectal lesion is that, *if the lesion is higher than you expect, you may significantly damage the rectal anatomy*. *Unless you find the rectal stump just underneath skin level, stop, make a colostomy, and arrange for repair later. Don't divide any muscle*. If you are in any doubt, a colostomy would be wiser, even though you may occasionally fashion one unnecessarily. *The penalty for failure is incontinence*. Because bowel washout for the definitive procedure is much more effective with a sigmoid colostomy, do this rather than a transverse colostomy.

Examination
Ask your midwives to examine the anus in all children. Ask them to refer:
(1) any child with an abnormal anus,
(2) any child who passes no faeces for 12h, and whose abdomen distends. Examine him, and if necessary, pass a rectal thermometer or a stiff catheter.

If there is no anus, make an opening before the bowel distends. Look for other congenital abnormalities.

If there is a mass of irregular epithelium where the anus should be, the diagnosis is almost certainly anal stenosis. Probe it and look for even a trace of meconium to confirm the presence of an opening, however tiny.

If there is a thin veil of epithelium overlying the anal orifice, surrounded by normal skin folds and *rugae*, there is an imperforate anal membrane.

If the anus looks normal, until you put a probe into it, when you find that the rectum is almost or completely blocked, the diagnosis is rectal atresia. You may find a very small hole which you can dilate.

If the anal skin is smooth, there must be some kind of lesion other than stenosis.

LOOK FOR FISTULAE:
Check for meconium in the urine, or in the vagina or vulvar vestibule. Look for a dark blob of meconium under the skin, especially in the midline *raphe*. A perineal ultrasound scan, demonstrating a fluid-filled track, may well help you in diagnosing a fistula.

INVERTOGRAMS OF IMPERFORATE ANUS

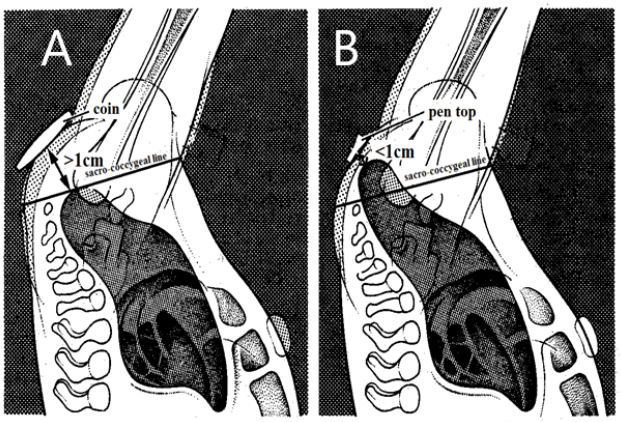


Fig. 33-7 RADIOGRAPHS OF AN 'IMPERFORATE' ANUS showing high (A) and low (B) rectal lesions. Lift up the child's legs and buttocks and stick a piece of metal over the anus. A, high lesion with the gas bubble in the rectum, below the sacro-coccygeal line, >1cm from a coin placed over the anus. B, low lesion with a pen top on the anus <1cm from the gas bubble.

Radiographs (INVERTOGRAM, 33-7)
Wait until 12-16h after birth, when the gas the child has swallowed has reached the blind lower end of the bowel. Place the baby prone on a pillow so that the anus is uppermost. Flex the knees. Strap a small piece of metal such as a paper clip flat on the skin where the anus should be.

Take a lateral film. The bubble of gas in the bowel will be uppermost. The distance between it and the metal will show you how much tissue there is between the bottom of the bowel and the skin.

Interpret the films as follows: if the gas bubble is <1cm from the anus, this is a low lesion; if it is >1cm from the skin, it probably is a high lesion. More accurately, draw a line between the posterior part of the pubis and the coccyx.

If you see any blind dilated bowel distal to this line, this is a low lesion. If it remains proximal to this line, the lesion is high. Anything in between is intermediate.

CAUTION!
Radiographs are useful *but not completely reliable*. There may not have been enough gas in the bowel, or there may be a fistula higher up.

Ultrasound shows the rectum as a hypo-echoic structure. There may be a rectovesical fistula and so gas producing a fluid level in the bladder (uncommon). Exclude this by taking a supine lateral film.
Intravenous urography is not indicated at the early stage.
A fistulogram may provide important information, though.

Management
If a child has a low lesion, operate early. The obstructed bowel is going to distend, so, as soon as he has swallowed enough air to help make the radiographic diagnosis, pass a nasogastric tube. Maintain hydration and add 1mg IM vitamin K_1.

CAUTION! If you are in doubt, fashion a colostomy.

(1) ANAL STENOSIS AND IMPERFORATE ANAL MEMBRANE (boys and girls)

ANAL DILATION (GRADE 1.2):
Incise the epithelial covering of the anus if there is one. If the anal opening is very small, use a filiform urethral catheter with a metal follower or the smallest Hegar dilator. Teach a parent to dilate the child's anus daily, then every other day, then weekly with the little finger, and provide a supply of disposable gloves to do this.

(2) ANOCUTANEOUS FISTULA (boys and girls) AND ANOVESTIBULAR FISTULA (girls)

CUTBACK OPERATION (33-8B, C) (GRADE 1.4):
Introduce a grooved dissector through the fistula and point it posteriorly strictly in the midline. Cut down onto the dissector to the point where the anus should be.

N.B. If you cut deeply, you may divide sphincter muscle!

Suture the anal mucosa to the skin, using interrupted long-lasting absorbable sutures. Dilate the anus as in (1) after 5 days. You can deal with the 'anterior ectopic anus' (really a type of anocutaneous fistula) in the same way with a cutback operation.

(3) RECTOVAGINAL AND RECTOVESTIBULAR FISTULA (girls)

In practice it is difficult to differentiate between the high and the intermediate anorectal agenesis. Where there is a rectovaginal fistula, the lesion tends to be lower. However, it is of no great consequence in the neonatal period. Dilate the fistula tract with Hegar's dilators. *Make sure the track does not stenose*: repeat a dilation every 3 months.

An anoplasty may be required when the child is >1yr. If this is impractical, it is not a disaster, but it will mean that she will always defecate from the fourchette. This is much less of a disability than it might seem; it is, for example, compatible with a normal sex life, and the husband may be quite unaware of it! However, some faecal incontinence may always be a problem.

ANOCUTANEOUS FISTULA

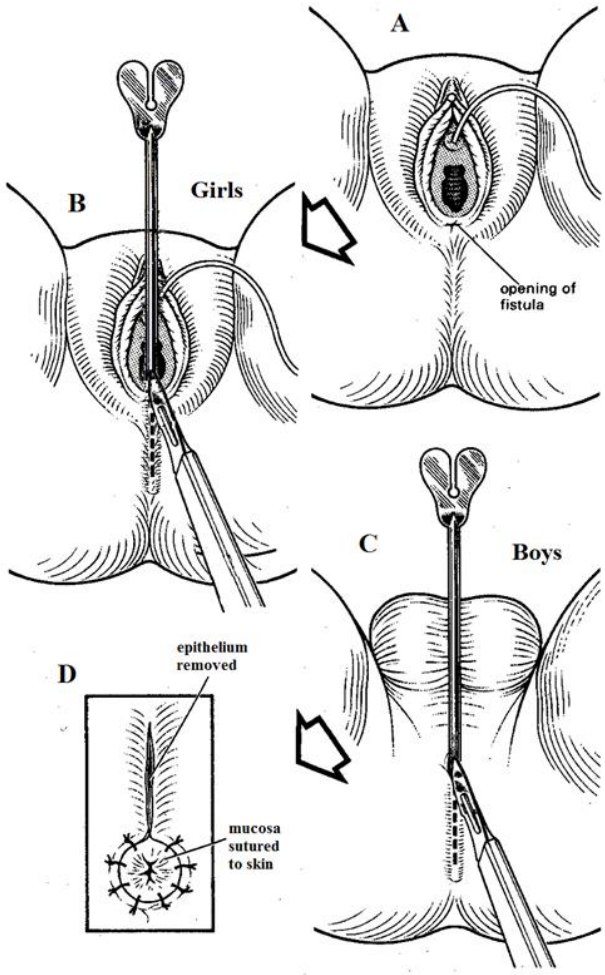


Fig. 33-8 ANOCUTANEOUS FISTULA.
A, fistula opening close to the introitus. B,C, cutback operation; note that the director points posteriorly, and remains superficial. Cut along this grooved director to where the anus should be. D, remove epithelium of the track, pull out the anal mucosa and suture it to the skin.
Adapted from Mustard WJ & Ravitch MM (eds). Paediatric Surgery. 3rd ed 1978 Fig.98-4. Yearbook Medical, with kind permission.

(4) ALL OTHER ANORECTAL LESIONS

Create a SIGMOID COLOSTOMY (11.6). Make a small Lanz-type transverse incision (11-1) in the left lower abdomen and carefully retract the muscle layers until you reach the peritoneum. Open this very carefully; underneath you should find colon: gently pull some out, perforate it and aspirate its contents. This will reduce abdominal distension and hopefully allow you to extract enough bowel to make a skin bridge to let you really divide the colon. If you cannot do this, fashion a loop colostomy.

Wash the distal colon free of meconium with warm saline: *if you don't do this early, the meconium will solidify like concrete!* Make sure you do frequent washouts if there is a recto-urinary fistula in order to minimize urinary tract infection.

Don't perform a formal laparotomy: it is difficult and unnecessary and the baby may well not survive it.

You don't need stoma bags for a neonate! Wrap the nappy over the stoma, and advise mother to clean gently around the stoma as she would for the anus.

Don't be tempted to operate on the anus of any intermediate or high anorectal agenesis; leave fashioning a neat anoplasty to an expert. This will not be possible if you have damaged the sphincter musculature.

Difficulties with anorectal malformations
If the anus becomes stenosed, it has become fibrosed. Unfortunately, regular dilation perpetuates the cause of the fibrosis. A formal anoplasty will eventually be needed.

If there is a mucosal prolapse or skin excoriation after an anal operation, apply talc powder and carefully prevent soiling of the perineum. The prolapse will reduce spontaneously.

If excess fluid is lost from a colostomy, wait for 14 days, while adding extra fluid to maintain a satisfactory fluid balance. The stools will usually become formed.

For other colostomy complications, see 11.6.

33.7 Hirschsprung's disease

In this not-uncommon disease, the neuroganglion cells in the wall of the rectum are absent, so that faeces are not propelled onwards as they should be. The length of the aganglionic segment varies although it is usually confined to the rectosigmoid in 75%, but may be tiny (ultra-short segment) or extend to the caecum or beyond (c.5%). This results in dilation and hypertrophy of the proximal (neurologically intact) segment with transition to a normal-sized or narrowed distal (aganglionic) segment. The child becomes constipated, and the abdomen distends; and in acute cases the bowel may obstruct. There may be a meconium plug in the rectum.

(a) Subacute presentation: a child presents soon after birth with subacute intestinal obstruction; its onset is more gradual than with complete mechanical obstruction, but may still be fairly acute. There is usually a history of delayed passage of meconium >24h after birth.

Rectal examination often results in explosive projectile passage of meconium or faeces, which may result in visible deflation of the abdomen. Abdominal distension is always present, and may be quite alarming if there is enterocolitis or bowel perforation (20%), resulting in a TOXIC MEGACOLON. There are then signs of peritoneal inflammation. *In this situation, repeated washouts will make the situation worse!*

(b) Late presentation: an older child has a chronic history of constipation *without laxative abuse or psychosocial problems*. The abdominal distension may be gross and affect breathing. Intermittent bowel actions tend to be explosively incontinent and infrequent.

(c) Investigations
Abdominal radiographs show multiple loops of distended bowel occupying the whole abdomen, with only occasional air-fluid levels and minimal or no air in the rectum; if there are gas bubbles in the bowel wall, this is a sign of enterocolitis. If there is a pneumoperitoneum (which may be huge), this is a sign of bowel perforation.

A barium (or, safer, gastrografin, which does not become solid) enema may conclusively make the diagnosis: look for a dilated proximal segment of colon and narrow distal rectum. This delineates the 'transition zone'. Repeat the film after 24h to see if there is retained barium proximally. A post-evacuation film can be particularly helpful.

Check the thyroid function: hypothyroidism may totally mimic Hirschsprung's disease.

(d) Full-thickness rectal biopsy (GRADE 1.4)
You need this investigation to make a definitive diagnosis. If the presentation is acute, do it at the same time as a sigmoid colostomy (11.6).

Put the child in lithotomy position under anaesthesia (*e.g.* ketamine), and dilate the anus with Hegar's dilators. Clean the anorectum with non-alcoholic antiseptic solution, and leave a swab soaked with this inside the rectum, proximal to the operation site. *Remember to record this swab inside!* It is best if you tie a suture to it to hang outside the anus on a mosquito forceps.

Evert the posterior wall of the anus on both sides with Allis forceps and place all-layer stay sutures postero-laterally on both sides at the level of the internal sphincter (33-9A). Retract the anterior wall of the anus upwards, exposing the rectal wall.

Make an incision and excise a 1·5cm triangular 5mm wide full-thickness segment with fine scissors (33-9B), and close this with 4/0 long-acting absorbable *through all layers* of the rectal wall. Remove the gauze you put in the rectum.

FULL-THICKNESS RECTAL BIOPSY

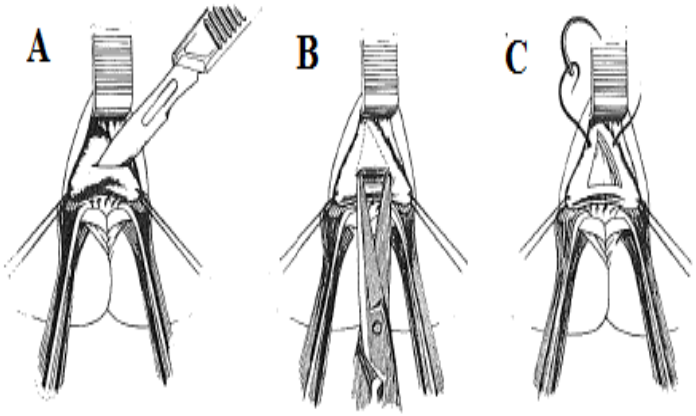


Fig. 33-9 FULL-THICKNESS RECTAL BIOPSY.
A, exposure of rectal mucosa and incision. B, excision of a triangular piece of rectal wall. C, suture of rectal wall defect.
After Lister J, Irving IM. Neonatal Surgery, Butterworth 3rd ed 1990 p.534 Fig 37.10.

N.B. There are more sophisticated methods of getting histological diagnosis; consult colleagues if these are available because a full-thickness rectal biopsy may make the definitive pull-through operation more difficult.

Try to refer such a child for definitive 'pull-through' surgery to an expert, because a repeat intervention for problems hardly ever gives a very good result. In the meantime, show the parents how to perform bd washouts with a soft catheter, using warm clean water. If the delay will be significant, perform a defunctioning transverse colostomy (11.5,6).

33.8 Neonatal jaundice

Most neonatal jaundice is 'medical', and only occasionally is it 'surgical'. Physiological jaundice (with levels of unconjugated bilirubin >85μM) fades after 1-2wks; however, levels >220μM (or >255μM in preterm babies) are *not* physiological. *Jaundice after 2wks of life is not physiological.* However, elevated *conjugated* bilirubin levels are surgically significant. The biliary tract can be blocked by epithelial debris or biliary sand (less common), in which case it may be temporary and clear spontaneously, or by atresia of the biliary tree (more common), for which the only hope is surgery. This blockage appears to be caused by a sclerosing inflammatory process starting *in utero*, and is not due to a simple single stenosis. Hepatomegaly is common, and stools may be pigmented initially before becoming white.

If much of the bile duct is not patent (as in 25% of cases), nothing can be done other than very major surgery (the Kasai procedure) by an expert.

If however the bile duct is sufficiently patent for the gallbladder to be distended, and in connection with the proximal duct system (75%), it can be anastomosed to the proximal jejunum.

BILIARY ATRESIA

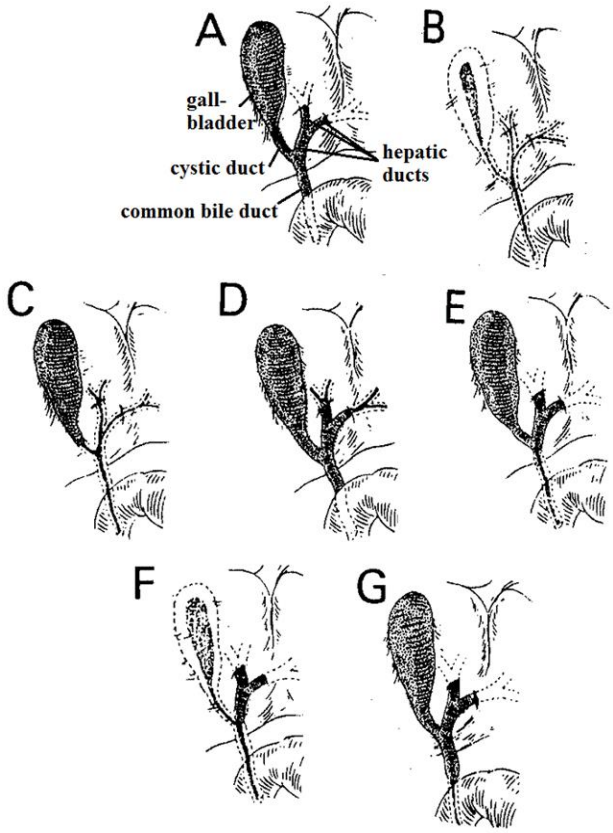


Fig. 33-10 BILE-DUCT ATRESIA.
A, normal. B, total atresia of all ducts. C, atresia of all ducts except the gallbladder. D, intrahepatic atresia. E, atresia of the common bile ducts. F, atresia of the common bile duct and gallbladder. G, segmental atresia of the distal common bile duct.
After Schwartz SI. Surgical Disease of the Liver. McGraw-Hill 1964, permission requested.

If there is an extra-hepatic stump, this too can be anastomosed. Without surgery, death is inevitable. Even in experienced hands the outlook is poor, and 30% of children develop liver failure.

Ultrasound (38.2c) can help with the diagnosis: you should be able to differentiate biliary atresia from dilation of the common bile duct (known as a **choledochal cyst**), which itself may obstruct the proximal common bile duct.

The time of onset is of great diagnostic value.
If a child starts to become jaundiced in the 1st 6-24h, jaundice is likely to be haemolytic from:
(1) Septicaemia from an umbilical infection. Look for signs of infection of the cord and the surrounding tissues (septicaemia from cord infection can also occur later).
(2) Haemolytic disease of the newborn. Usually, the baby is Rh D+ve, the mother is D-ve and has anti-D antibodies as the result of having had a previous D+ve gestation, or of having been given D+ve blood. Other blood group incompatibilities may have the same effect (*e.g.* ABO).
(3) Congenital syphilis (severe infection).

If mild jaundice starts at 24-72h, this is likely to be 'physiological jaundice' owing to a deficiency of glycuronidase (common, especially in 'small for dates' babies). This usually clears spontaneously, but can be helped by sunlight and phenobarbitone 1-2mg/kg bd.

If jaundice starts at 24-72h, and becomes severe by the 3rd-5th day, there may be G6PD (glucose-6-phosphate dehydrogenase) deficiency. This is common, especially in Southeast Asia and the Mediterranean littoral; a less severe form occurs in parts of sub-Saharan Africa.

If there is little or no jaundice till 3-6wks, by which time the liver is enlarged, and the stools are clay-coloured, there probably is some 'surgical' reason for the jaundice. Sometimes there is slight jaundice at birth, or a few days later; the urine is dark brown early.

If there are recurrent bouts of cholangitis, there may be a congenital dilation of the common bile duct (choledochal cyst) which may be saccular or diverticular. The best treatment for this is a Roux-en-Y choledochojejunostomy.

33.9 Hypospadias

If a boy's urethra opens on the ventral surface (underside) of the penis, this is a HYPOSPADIAS. There may be a glandular, penile, scrotal or perineal opening (or anywhere in between), often with a chordee (tight band) causing ventral curvature of the penis, especially seen in erection.

If there is perineal hypospadias with undescended testes, examine carefully regarding gender.

HYPOSPADIAS REPAIR: STAGE 1

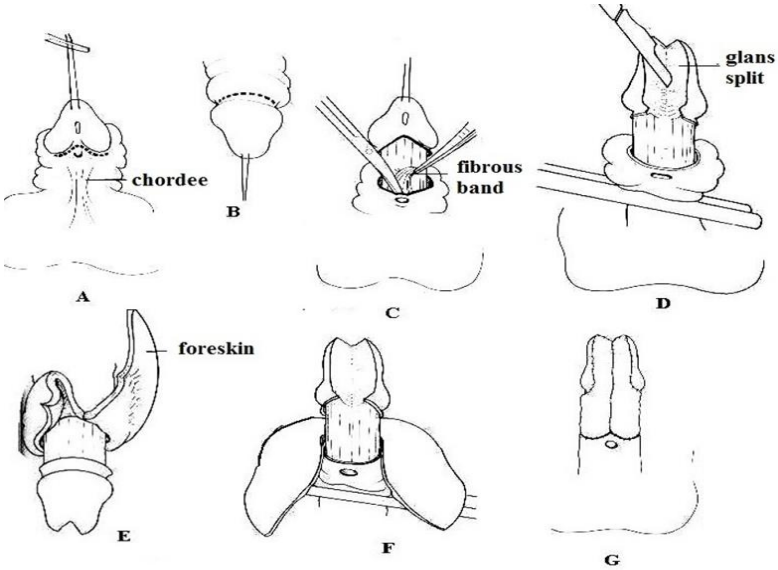


Fig. 33-11 HYPOSPADIAS CORRECTION: 1ST STAGE.
Chordee correction. A, apply a tourniquet. B, correct the chordee: circumferential incision. C, excise the fibrous band. D, slit the glans penis. E,F, divide the dorsal foreskin into 2 long flaps. G, bring flaps round to cover raw ventral penile surface.
After Blandy J. Operative Urology, Blackwell 2nd ed 1986 p.206 Figs 14.2-6.

Never circumcise the hooded prepuce in hypospadias patients because it is needed for urethroplasty repair. Do this corrective surgery around 1-2yrs. If there is a chordee, the two-stage procedure is more reliable for the non-expert.

PENO-SCROTAL HYPOSPADIAS CORRECTION: 1ST STAGE (GRADE 3.4)

Make sure the child is thoroughly bathed with antiseptic and bowel action is well controlled.

Place a stay suture at the tip of the penis and a tourniquet at its base. Widen the meatus if it is stenosed either by dilation or a meatotomy (27-29).

Make an incision right round the coronal sulcus (33-11A,B), and dissect the urethra off the *corpora cavernosa*. Firm bands may tether the urethra and these must be excised to correct chordee (33-11C).

See if you have achieved correction by injecting saline in the corpus to create an erection: if not, complete the dissection proximally to the bulb. Then open up the glans penis by incising it up to the tip on the ventral surface in the midline until it looks quite flat (33-11D), and then divide the hooded foreskin on the dorsal surface in the midline, separating its inner and outer layers (33-11E,F).

Bring the two flaps round to cover the raw ventral surface (33-11G) of the penis with 5/0 absorbable sutures and leave in a Ch6 Foley urethral catheter or feeding tube for 4-5 days.

2ND STAGE (GRADE 3.5)

About 6 months later, making sure the child is thoroughly bathed, fashion a suprapubic cystotomy (33-12A). Then mobilize the skin on the ventral surface of the penis and wrap it snugly round a Ch8 feeding tube with 6/0 absorbable sutures, trying to keep the suture line off midline. Try swinging around an intermediate layer of *dartos* tissue from the dorsum over the suture line. Close the outside skin around this suture line, preferably avoiding one suture line lying over the other. Keep the catheter *in situ* 7-10 days with urine dripping freely: *if you attach a urine collection bag, it is likely to pull on your repair and disrupt it!*

Nurse the child in a 'double nappy' with a hole for the genitalia in the inner nappy, in order to separate stools from the operative site.

HYPOSPADIAS REPAIR: STAGE 2

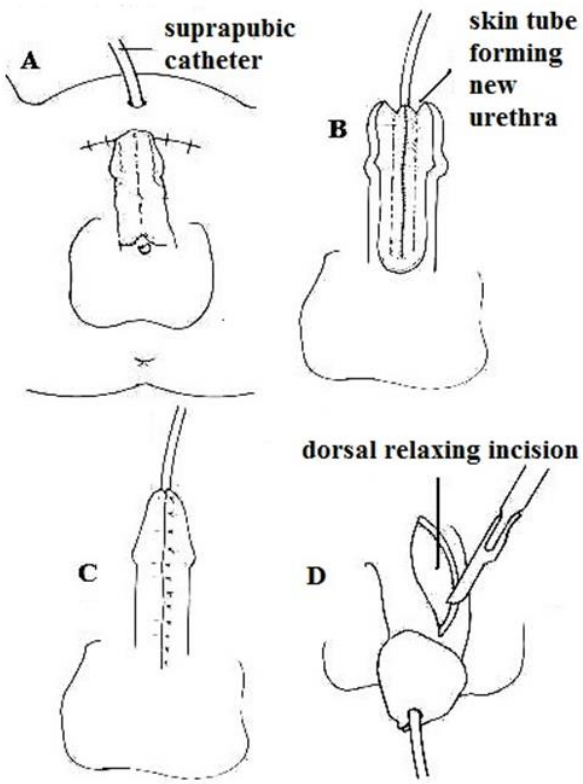


Fig. 33-12 HYPOSPADIAS CORRECTION: 2ND STAGE.
A, insert a suprapubic catheter. B, outline a full-thickness skin tube using a suitable catheter as template for its width. Suture a skin tube over the catheter, *preferably not in the midline*. C, close glans and penile skin over the skin tube. D, make a dorsal relaxing incision.
After Blandy J. Operative Urology, Blackwell 2nd ed 1986 p.208 Figs 14.8-11.

DIFFICULTIES WITH HYPOSPADIAS CORRECTION

If a small urinary fistula develops, make a Y-shaped incision over the hole (33-13A), and mobilize the skin thoroughly to expose the fistula hole (33-13B). Close the hole with an inverting long-acting absorbable 4/0 suture, and then advance the skin over the hole to cover it (33-13C).

If there is complex scarring, chordee and fistulae, the whole scarred urethral segment must be excised and a new urethra created using a pedicled *dartos* scrotal skin flap. This is something for an expert.

URETHRAL FISTULA CLOSURE

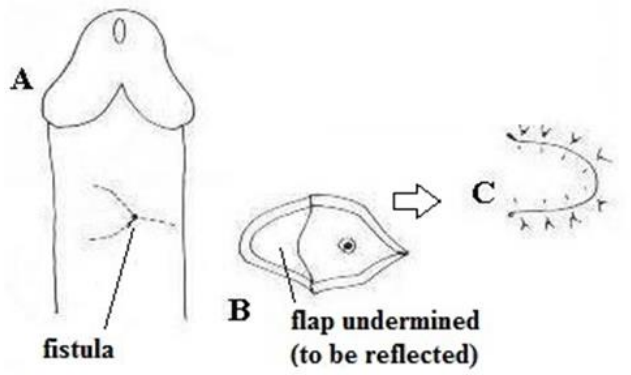


Fig. 33-13 CLOSURE OF URETHRAL FISTULA.
A, Y-shaped incision. B, mobilize the skin to expose the fistula. C, U-closure of skin.
After Blandy J. Operative Urology, Blackwell 2nd ed 1986 p.208 Fig 14.12.

33.10 Spina bifida & encephalocoele

Congenital abnormalities of the spinal cord and vertebral column are not uncommon. You may see:

(a) Spina bifida occulta, in which the arches of the vertebrae remain open but the skin is closed, usually in the lumbar region. There is often a brownish spot over the defect (this is less easily seen in dark skin, but it is there if you look for it), and/or some extra hair and fatty tissue. Spina bifida occulta is usually symptomless, but the child may develop a tethered cord as he grows, particularly during growth spurt periods: so watch him carefully. If the legs become weak or urinary or faecal incontinence develop, he will need prompt untethering of his spinal cord: leave this to the experts!

(b) A meningocoele is an extension of the spinal canal, filled with CSF but without any spinal cord or spinal nerves in it; commonly in the lumbar region, and usually associated with spina bifida. The cord is normal and there is no neurological abnormality. It is a relatively simple procedure to obliterate the sac by closing the dura, and then to close the skin. This is a closed lesion, so there is no hurry.

(c) A myelomeningocoele is more common than a simple meningocoele, and takes two forms:
(1) There is a closed swelling containing spinal cord and/or spinal nerves.
(2) More often, the spinal canal is open and leaks CSF, with the flattened cord forming a plaque on its surface.

Both varieties may occur in the cervical (rare), thoracolumbar, lumbar, or lumbosacral regions, and other abnormalities are frequent, particularly hydrocephalus. Many children have irreversible paralysis of their legs, and loss of sphincter control, but some can still achieve quality of life through surgery and good follow-up. The decision of whether to operate or not on these children is sometimes difficult and must be made with each family. *Don't operate without discussing the long-term orthopaedic, urinary and faecal continence and psychological problems extensively and repeatedly.*

N.B. A myelomeningocoele may be obscured by a lipoma: *beware!*

(d) An encephalocoele is a condition where the brain herniates through a cranial defect, usually occipital or naso-frontal. The neural function may be entirely normal, though large defects may be associated with microcephaly and other brain anomalies. Occipital defects without involvement of the medulla oblongata are easier to repair while naso-frontal ones, especially if affecting the olfactory nerve, are much more difficult. Try to get an ultrasound to delineate the true situation, and decide if you had better leave this intervention to an expert.

MENINGOCOELES

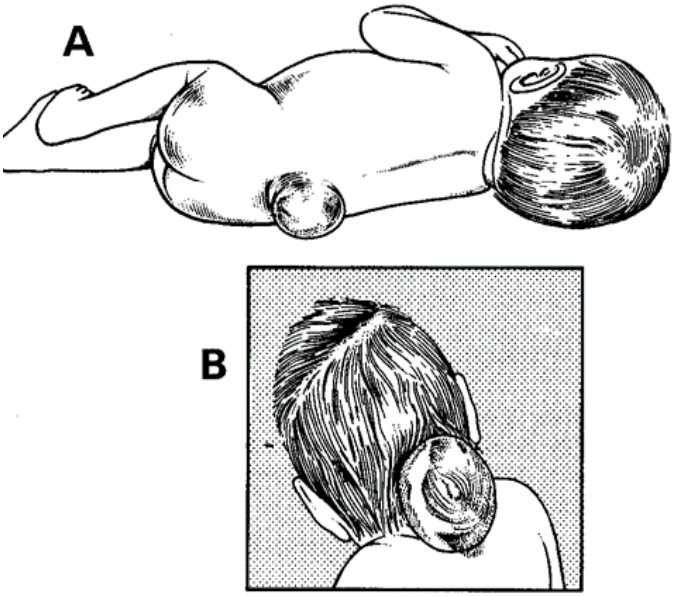


Fig. 33-14 MENINGOCOELES.
A, skin-covered sacrolumbar meningocoele. You may be justified in operating on this child. B, cervical meningocoele. *Leave this for the experts.*

Diagnosis
Make sure you examine the baby carefully in a warm room, repeatedly if necessary. Look for other abnormalities, especially hydrocephalus (present in 80%: measure the head circumference), and cardiac defects. Ultrasound is very helpful if you can interpret the images.

If the swelling on the child's back is covered by skin, the legs are not weak, the anus is not lax, and he micturates normally, he probably has a simple meningocoele or lipomeningocoele. Otherwise, you cannot tell a meningocoele from a myelomeningocoele from its site, or its covering.

If you transilluminate it, you may occasionally see nerves outlined inside it. Ultrasound will give you an idea how much fluid is inside.

If there are any neurological signs, there probably is a myelomeningocoele. If it is open, there will almost certainly be some neurological defect such as:

(1) Partial or complete paralysis of buttock, thigh, leg or feet muscles, often with deformities such as *talipes equinovarus* (32.10) or *genu recurvatum*.
(2) An absent or meagre response to tickling or pin-prick (*again tested in a warm, relaxed environment*).
(3) A lax anal sphincter, an absent anal reflex, or a rectal prolapse.
(4) Urinary retention which can be expressed by forceful suprapubic pressure, associated with overflow dribbling, or rarely constant dribbling with an empty bladder.

N.B. Differentiation between voluntary and reflex movement is difficult; if the baby is cold, voluntary movements may disappear!

If there is a low solid sacral lesion which on pressure does not distend the anterior fontanelle, this is likely to be a sacrococcygeal teratoma (33.13). Unlike spina bifida, it tends to displace the anus forwards, and can extend into the abdomen when it is bimanually palpable.

If there is a solid spinal tumour infiltrating bone, this is probably a chordoma, a rare slow-growing tumour of notochord. Resection is the only hope for cure.

Management of meningocoeles

If the meningocoele is covered with normal skin (usual), surgery is not urgent. Close the defect when the baby is bigger (>6-9 months). This should be done by an expert if the lesion is in the cervical or thoracic region. If the lesion is not fluid-filled, it is likely a lipomeningocoele: a very complex lesion that must be repaired within the first 2yrs of life, but only by an expert.

If there is an open meningocoele or a myelomeningocoele with severe neurological signs (commonly in the thoracolumbar or lumbar regions), there is nothing to be done except compassionate palliation.

Some movement in the legs and anal tone imply incomplete nerve damage: you must explain carefully then to the parents the prospects of the child needing difficult and extensive rehabilitation before embarking on operative intervention. Once you start, you signal a commitment to continue!

Note that closing the defect will *not* improve any neurological deficit. Therefore discuss the situation fully with the family before deciding on surgery.

MYELOMENINGOCOELE CLOSURE

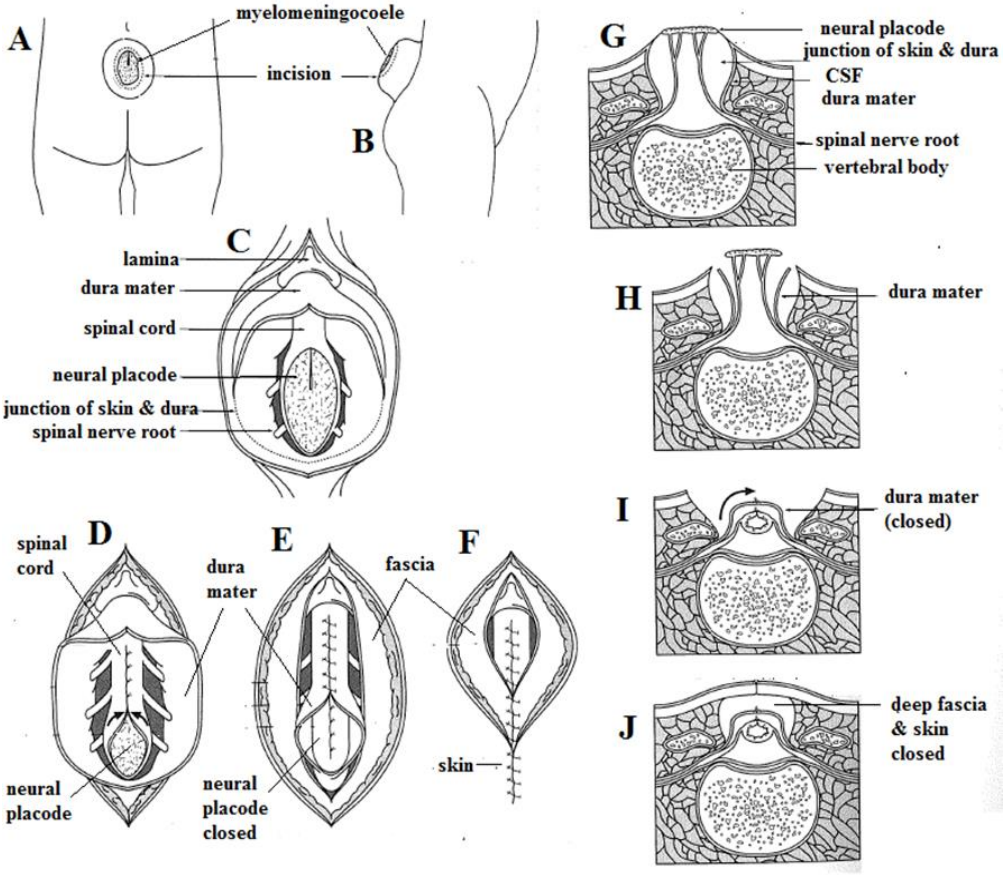


Fig. 33-15 MYELOMENINGOCOELE REPAIR.
A,B,G, line of excision at edge of neural plaque (placode). C, free the elements of the defect to show the anatomy. D,H, fold over the placode to create a neural tube. E,I, close the dura. F,J, close the deep fascia and skin. G-J coronal views: G, before dissection.
After Rosenfeld JV, Watters DAK. Neurosurgery in the Tropics. Macmillan, 2000 p.386-8 Fig.11.8 with kind permission.

If there is a lumbo-sacral myelomeningocoele, with reasonable power in the legs (normal sphincter control is unusual), operation may be justified. You will probably have to deal with variations of club foot deformity (32.10) later, as well as the development of hydrocephalus in many.

CLOSING A MYELOMENINGOCOELE OR MENINGOCOELE (GRADE 3.5)

INDICATIONS

A fairly small lumbo-sacral meningocoele, with minimal neurological signs, no hydrocephalus, *and no other congenital abnormalities*.

CONTRAINDICATIONS

(1) Complete or virtually complete denervation below the level of the lesion.
(2) Progressive hydrocephalus.
(3) A very large lesion, in which you will have difficulty closing the skin and subcutaneous tissues without tension.
(4) Lesions in sites other than the lumbar or sacral region.
(5) Untreated HIV disease.
(6) A severely infected lesion: *drain the pus first!*

PREPARATION. Take the strictest aseptic precautions and *be careful not to damage the cord*. Lay the child prone with the head of the table depressed at least 30º, to minimize the loss of CSF when you incise the sac. Prepare and drape a wide operative field. Clean the sac meticulously with aqueous disinfectant.

OPERATION

If the swelling is covered by skin, make a longitudinal elliptical incision, through normal skin at the base of the swelling (33-15A,B). Cut through the subcutaneous tissue to the deep fascia, and define the neck of the sac by blunt dissection. The defect in the fascia will usually be quite small, and easy to expose. Free all surfaces of the sac, and open it over its dome. Send the CSF for culture if possible. If there are no nerve filaments, amputate the sac at its base, and close it with continuous 4/0 or 5/0 non-absorbable suture.

If you find nerve filaments or the meningocoele is open, preserve the nerve filaments with the greatest care, and try to free them from the sac (33-15C). *Don't excise more healthy skin than you need*, but be sure to remove all the skin covering the placode in order to prevent the development of an epithelial remnant cyst later. If possible, after you have freed the neural plaque (placode), fold it over and close it along its axis (33-15D). Then close the dura over it with 4/0 or 5/0 prolene after freeing it on both sides of the plaque (33-15E).

Now free up the thick fibrous layer overlying the deformed spinal laminae on each side of the defect, and approximate these with the musculofascial layer (33-15F) so that you can obtain tension-free and solid skin cover.

Seal the wound meticulously, and nurse in the prone position. Apply a corset of orthopaedic strapping so that the abdomen is pulled upwards (33-16). This keeps tension off the suture line and allows faeces and urine to drain away from the wound. Leave a nasogastric tube *in situ* for 24h, and then start feeding. Keep the prone position for 10-14 days till you remove sutures. If there is a neurogenic bladder, make sure you train the mother to empty the bladder regularly by suprapubic pressure. You will then later have to teach her intermittent catheterization. *Keep faeces away from the wound!*

If the child also has hydrocephalus, a shunt (33.11) or ventriculostomy will be necessary, but wait at least 1wk after the back closure, till the risk of infection is diminished. Proceed when the back is healed, the ultrasound (38.2m) shows enlarging hydrocephalus, the child is well, and the CSF (from a ventricular tap) is clear.

The long-term management of neurologically impaired children with spina bifida is extensive, requiring bladder evaluation with possible clean intermittent catheterization, renal follow-up with ultrasound, bowel management, and occupational therapy. Find a centre where such care exists: the International Federation for Spina Bifida and Hydrocephalus can assist in the process.

SUSPENDING A BABY AFTER MYELOMENINGOCOELE REPAIR

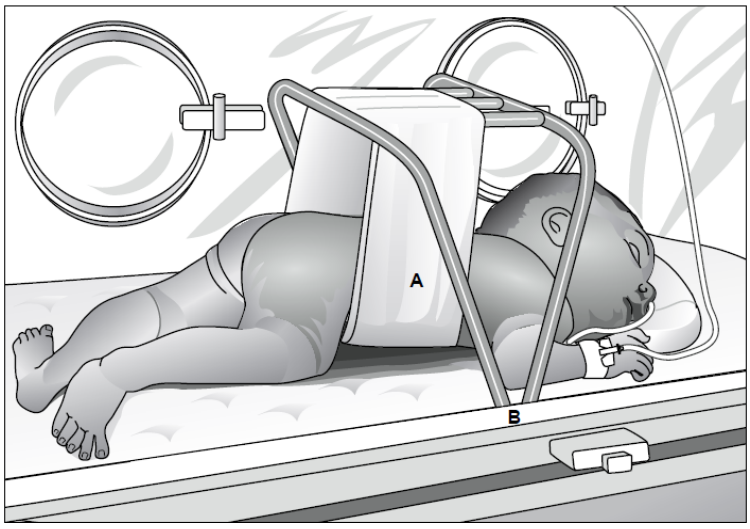


Fig. 33-16 POST-OP SUSPENSION OF A BABY AFTER MYELOMENINGOCOELE REPAIR.
A, orthopaedic strapping. B, special supporting frame. This keeps tension off the suture line; *don't dress the wound but make sure faeces and urine drain away from it.*

33.11 Hydrocephalus

When there is inadequate absorption of CSF in the head, its volume builds up in the ventricular system and a child's head consequently expands in size. When the CSF is unable to flow from the ventricular system to the subarachnoid space, it is described as a non-communicating hydrocephalus.

Congenital hydrocephalus usually arises from obstruction of the aqueduct of Sylvius, and in children with spina bifida from cerebellar and medullar herniation through the foramen magnum (the Chiari II malformation). Acquired hydrocephalus commonly arises from meningitis, but may result from intraventricular haemorrhage of prematurity, posterior fossa tumours or ventricular cysts.

(a) Symptoms in toddlers and children

Vomiting, drowsiness, irritability, fever, headache and loss of cognitive function or coordination are the commonest symptoms.

(b) Signs

The anterior fontanelle is bulging and tense, and scalp veins may be prominent; skull sutures separate and may become palpable, and the head may give a 'cracked pot' sound on percussion. In advanced cases, the child becomes lethargic and anorexic. When the 3rd ventricle expands, pressure on the oculomotor nerves causes down-turned ('setting sun') eyes. Cranial nerve palsies (especially VIth causing a squint, or Xth causing stridor) are not uncommon.

(c) Ultrasound (38.2m) will demonstrate dilated ventricles, as well as showing up intraventricular haemorrhage, cysts in the 3rd ventricle or a fourth ventricle tumour. You may be able to detect hydrocephalus antenatally.
You can measure the thickness of the cerebral cortex: if this is <20mm, shunting will almost certainly be required, although the relationship of intelligence and cortical thickness is by no means proportional.

(d) Differential diagnosis
Distinguish hydrocephalus from **craniosynostosis**, where there is premature fusion of cranial suture lines and an odd-shaped enlarged head, but no bulging fontanelle.

ANATOMY OF THE VENTRICULAR SYSTEM

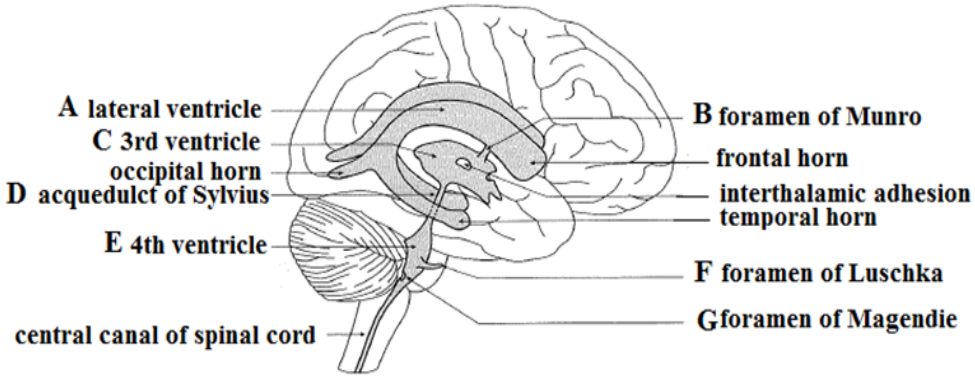


Fig. 33-17 ANATOMY OF THE VENTRICULAR SYSTEM. About 20ml/h of CSF is actively secreted in the choroid plexuses in the lateral ventricle (A), the 3rd ventricle in the diencephalon between the 2 thalami (C). It leaves these by the aqueduct of Sylvius (D) to the 4th ventricle in the hindbrain (E), where more CSF is secreted. From here it passes through the 2 lateral foramina of Luschka (F) and the midline foramen of Magendie (G) to bathe the brain and spinal cord in the subarachnoid space. The total volume of CSF in an adult is c.130ml. *After Rosenfeld JV, Watters DAK. Neurosurgery in the Tropics, Macmillan 2000 p.90 Fig 4.1 with kind permission.*

You should measure the head against a standard head circumference chart:

OCCIPITAL CIRCUMFERENCE

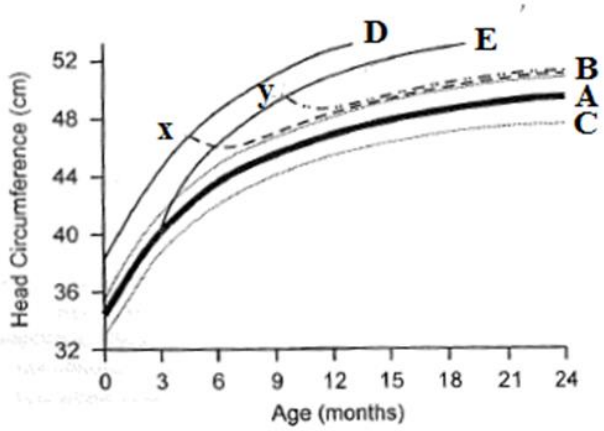


Fig. 33-18 HEAD CIRCUMFERENCE CHART, measured horizontally around the occiput just above the level of the eyebrows. A, 50th centile. B, 98th centile. C, 2nd centile. D, congenital hydrocephalus treated at point 'x'. E, hydrocephalus, acquired at age 3 months, treated at point 'y'.
After Rosenfeld JV & Watters DAK. Neurosurgery in the Tropics, Macmillan 2000 p.92 Fig 4.2 with kind permission.

(e) Ventricular tap is useful: shave and clean the head and disinfect the site carefully; aspirate through the anterior fontanelle with a 20G needle and syringe. Start at the level of the coronal suture just off the midline, aim slightly away from the midline, advance while aspirating until CSF is found. If the pressure is high, aspirate until the fontanelle is flat. *Don't try to do this slowly: it is unnecessary and you will anyway fail!* Send the fluid for CSF analysis. You can measure the CSF pressure by attaching the needle to an infusion line and measuring the level above a zero point at the level of the head.

(f) Management
The commonest treatment for hydrocephalus is to insert a shunt to drain the CSF either into the peritoneal cavity or rarely into the atrium. The ventriculo-peritoneal (VP) shunt is simpler to insert, but will probably not be effective if there has been peritonitis previously, because of adhesions.

Various types of shunt exist, with different valve mechanisms, but it is not necessary to use commercially produced shunts. An affordable shunt is the Chhabra shunt from India (provided free to qualified centres by the International Federation for Spina Bifida and Hydrocephalus).

Don't delay shunt insertion in hydrocephalus, except until intraventricular haemorrhage or meningitis has recovered and the CSF is clear (<10 cells/ml) with a low protein content.

Don't attempt to treat a child with a head circumference >60cm if there is gross neurological deficit.

VENTRICULO-PERITONEAL SHUNT
FOR HYDROCEPHALUS (GRADE 3.4)

PREPARATION
Shave the scalp and clean it thoroughly with alcohol and betadine before operation. Administer prophylactic cefalosporin, or gentamicin and ampicillin.

Position the head turned laterally on a head-ring, with the table elevated 10º and a support under the shoulders to keep the neck extended so that there is a straight line from head to abdomen along the anterior chest wall in front of the clavicle (33-19B).

INCISION
Make a semicircular flap 3cm above the centre of the pinna and 4cm behind its top edge, in the occipito-parietal area (33-19A). *Don't make this incision too low* where you may hit the transverse venous sinus.

Make a burr hole (or if the bone is very thin, nibble it away with forceps or scalpel) but don't open the dura; before you do so, make an opening in the peritoneum through a small transverse right hypochondrial incision and make sure you are actually inside the peritoneal cavity by attaching haemostats to the edges of the peritoneal layer.

VENTRICULO-PERITONEAL SHUNTING

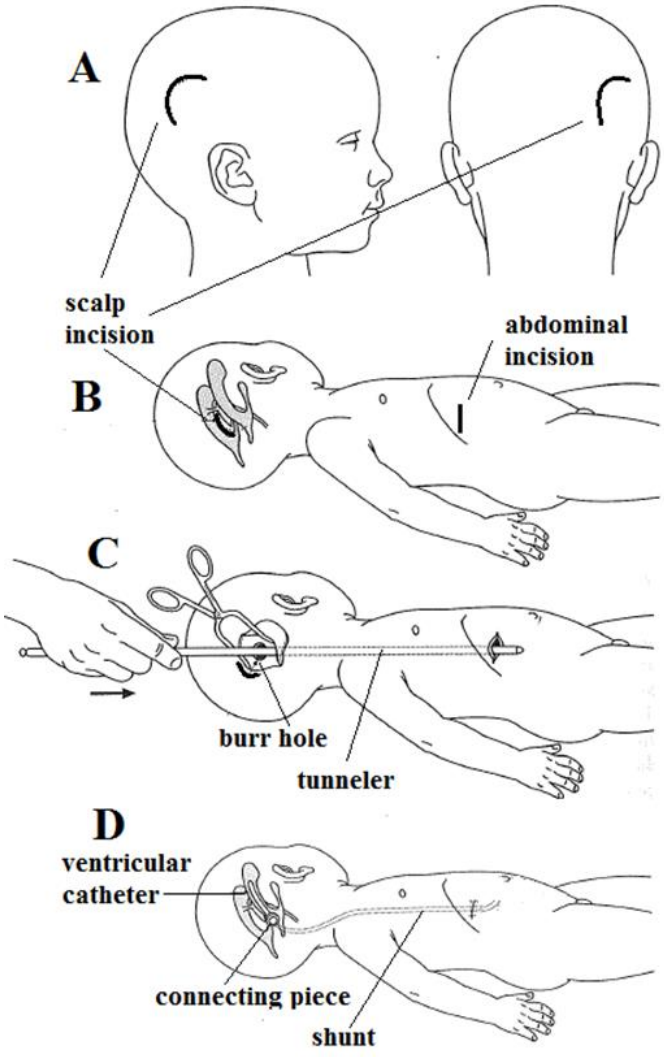


Fig. 33-19 PLACEMENT OF A VENTRICULO-PERITONEAL SHUNT.
A, scalp incision. B, operative position, showing orientation of ventricles and site of right upper quadrant abdominal incision. C, passing the tunneler. D, final placement of shunt.
After Rosenfeld JV & Watters DAK. Neurosurgery in the Tropics, Macmillan 2000 p.380 Fig 11.6 with kind permission.

Pass a tunneler subcutaneously between the 2 wounds (33-19C), either from above to below or *vice versa*. If you don't have a tunneler long enough, you may need to make an extra incision in the neck.

Attach the distal shunt tubing to the tunneler and pass it under the skin from neck to abdomen, but leave it outside the peritoneal cavity. Check its patency with saline. When it is correctly in place, remove the tunneler and fix the shunt tubing to the valve or connecting L-piece.

Then make a small cruciate opening in the dura just big enough to pass the shunt through. (An incision too big will allow CSF to leak.) With the proximal shunt mounted on a stilette, guide it forwards towards the inner canthus (corner) of the opposite eye (felt through the drapes). As soon as the shunt has entered the ventricle, you will see CSF emerge; remove the stilette: you should see a substantial flow of clear CSF. Send this for culture, if possible.

Then, check with ultrasound, if you can, that the shunt is well placed in the anterior horn of the lateral ventricle, withdraw it 2cm and attach it to the valve, or a connecting L-piece, trying not to handle the shunt except with instruments.

Fix the valve, or L-piece, with non-absorbable sutures to the skull periosteum. Make sure that CSF is dripping regularly from the distal end of the shunt, and then pass this into the peritoneal cavity either over the liver or deep into the pelvis.

Fix it to the peritoneum with an absorbable suture and close the abdomen taking care not to include the shunt tubing in your stitch or kink its position. Close the scalp flap over the tubing so that sutures lie well away from it.

DIFFICULTIES WITH VENTRICULO-PERITONEAL SHUNTING

Advise the parents to return the child in case of any serious symptoms: *late presentation of complications is the commonest cause of death and long-term disability in these children!* Complications are common, and usually occur in >30% of cases! You must warn parents that you may have to replace the shunt several times, and particularly as he grows.

If the shunt blocks, it may do so at the ventricular end (where the choroid plexus adheres to the tubing) or the peritoneal end (where the omentum or adhesions may occlude its end). Symptoms and signs depend on the rate and degree of the blockage, but essentially are worsening of the original hydrocephalus problems, especially vomiting, headache and loss of neurological function.

To treat the blockage, you need to explore the shunt, disconnect it and test the flow through it at the peritoneal and ventricular ends. You may be able to unblock the ventricular catheter by introducing a stilette down it, and passing a diathermy current along it. If it remains blocked, and remains stuck, it is best to place another shunt next to it rather than forcibly removing it.

If the shunt disconnects or migrates (which may be visible on simple radiographs), especially during a growth spurt, the peritoneal end may end up disconnected inside the abdomen, or under the skin. Reconnection with new tubing will solve the problem.

If the shunt becomes infected, either *de novo* or more commonly within a few months as a result of sepsis spreading from elsewhere (*e.g.* teeth, throat etc.), the child deteriorates with headache, fever, vomiting and maybe fits.

Try antibiotics alone, but if this fails, disconnect the shunt tubing and drain the CSF externally into a closed drainage system (an external shunt). When the CSF is clear and no longer infected, re-insert the distal end of the shunt. However, it is frequently necessary to remove the shunt entirely and replace it with a new one.

If the skin over the shunt necroses, it has probably been closed under tension. The skin needs debridement, and usually you will have to reposition the shunt on the opposite side of the head.

If the child develops peritonitis, the distal peritoneal end of the shunt needs to be removed from the abdomen, and allowed to drain into an external closed drainage system till the inflammation has settled down.

If there was only localized peritonitis (*e.g.* from appendicitis), the abdomen can probably still be used for drainage, but if the peritoneal inflammation was generalized, it may no longer absorb properly. In this case, a ventriculo-atrial shunt may be necessary.

If the abdomen distends with fluid, the CSF may not be absorbed properly as above. Alternatively the fluid may collect into a loculated 'pseudocyst' which is visible on ultrasound (38.2l). In this case, perform a laparotomy to break down the cyst walls and reposition the shunt if it remains patent.

N.B. More sophisticated surgery for hydrocephalus consists of endoscopic 3rd ventriculostomy, which has much fewer complications and is effective in the majority of cases. This procedure is not that difficult to grasp and has been effectively performed up-country in Mbale, Uganda. You need a flexible paediatric endoscope like a cystoscope, and to be shown how to do the procedure by an expert.

33.12 Congenital vascular lesions

Congenital vascular lesions are not uncommon, and may worry a parent, so you should know how to advise. Some will regress, some will grow as the child grows, and some increase in size alarmingly. Try to identify them correctly to advise the right treatment. Differentiate between angiomas (which are tumours) and vascular malformations (which are not). You may be able to diagnose cystic lesions prenatally with ultrasound.

A **capillary haemangioma** is, characteristically, a bright red, raised lesion varying from mms to cms in diameter. It is well circumscribed and only partially compressible.

A **cavernous haemangioma** is nodular and may be very large in diameter and depth. It is often bluish or purple and easily compressible. Mixed variants exist; they are histologically benign neoplasms of proliferative endothelium.

A **capillary cavernous haemangioma** consists of abnormal capillaries, arteries, and veins, and is partly compressible. It is usually present at birth, and commonly occurs on a child's face, axillae, or neck, where it may extend into the mediastinum. It may occasionally resolve spontaneously over several years (unusual), or it may enlarge rapidly.

Occasionally it connects with arteries and pulsates, expanding progressively: the feeding vessels then need ligation. Excision is indicated if there is functional disability (*e.g.* amblyopia from an eyelid haemangioma, 28.9), gross disfigurement or significant haemorrhage, or skin necrosis overlying the lesion.

CONGENITAL VASCULAR MALFORMATIONS

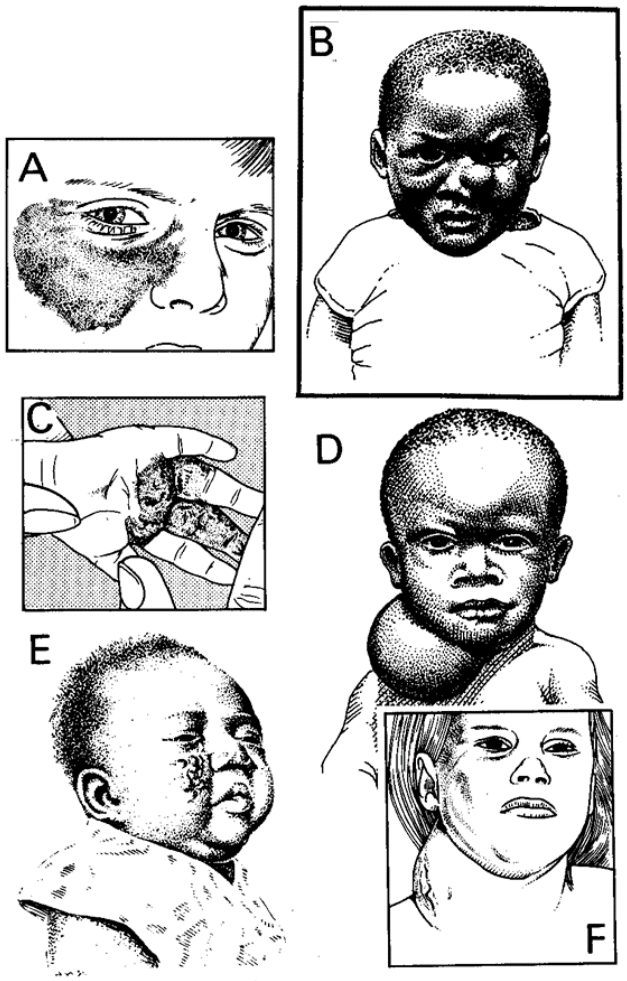


Fig. 33-20 CONGENITAL VASCULAR ANOMALIES.
A, a port-wine stain. B, a cavernous haemangioma. C, a capillary cavernous haemangioma of the hand. E, a capillary haemangioma of the face. D,F, cystic hygromas.
A,D,E, after Bowesman C. Surgery and Clinical Pathology in the Tropics, Livingstone 1960, permission requested.

The so-called '**strawberry naevus**' (33-20E) is a haemangioma skin lesion, and is commonest on the face. It is bright red, and a little raised, sometimes quite markedly. It may occur on the mucous membranes and may bleed severely. The lesions appear 1-4wks after birth, and enlarge for a few months.

Rapid growth is unusual after 6 months, but slow growth is common up to 1yr. *If you leave them alone, 95% will resolve spontaneously*. The first sign that this is happening is the appearance of a lighter, flatter area. Resolution may be precipitated by trauma, but this usually causes only minor scarring.

Advise the mother that the lesion will probably disappear slowly. Warn her not to allow traditional healers to scarify the lesion, which may cause bleeding, infection, and worse scarring. If resolution is slow, and parents pressurize you, refer the child to a plastic surgeon specialist because the final results of excision may otherwise be very disfiguring.

Lesions on the face, in the area of distribution of the ophthalmic and maxillary branches of the Vth nerve, may be associated with vascular abnormalities of the cerebral cortex (Sturge-Weber syndrome), and present with seizures. Glaucoma of the ipsilateral eye sometimes occurs, and is treatable (28.6).

The so-called '**port-wine stain**' or '**flame naevus**' (33-20A) is a malformation of cavernous channels, and usually occurs on the face or neck, but is not uncommon on the trunk. It is usually present at birth and does not progress, but it may be quite extensive. The texture of the skin is normal, and is not usually thickened; occasionally there is some hypertrophy and irregularity. If a lesion is particularly prominent, as in light skin, cosmetic creams may help. Otherwise, there is no treatment. Reassure the parent that the lesion will not enlarge.

The so-called **lymphangioma** is actually a malformation of cystic cavities filled with clear or straw-coloured fluid (actually lymph) which grow slowly, often infiltrating or surrounding adjacent structures. Occasionally it regresses, but it may grow alarmingly especially when infected.

It occurs usually in the neck and axilla, but may also be in the mediastinum, retroperitoneum, or the groin. It usually appears early, and may be present at birth. The mass is not attached to skin and is fluctuant. It may be very large, being known as a **cystic hygroma** (33-20D,F), where it may cause respiratory distress due to pressure effects on the airway.

Review the child carefully, and at each visit use a measuring tape to record the exact size of the lesion in 2 dimensions at right angles. At each visit record changes in appearance and complications, and if possible take photographs, measuring the size of the lesion.

Complications are ulceration, infection and bleeding, and rarely a consumptive coagulopathy and thrombocytopenia (Kasabach-Merritt syndrome).

You can try to cause thrombosis and regression by injection of 50% dextrose into the lesion. Simple aspiration will reduce the size of a lymphangioma temporarily, but with the risk of introducing infection.

Surgery is likely to be difficult though, and bleeding severe. In the neck, post-operative haemorrhage may cause acute neck swelling and respiratory compromise.

Differential diagnosis
Suggesting cystic degeneration in a lymph node: HIV disease, multiple asymmetric lesions in typical sites of lymphadenopathy.

Suggesting a branchial cyst: unilocular and low in the neck, along the anterior border of the sternomastoid; aspiration yielding a thicker opalescent fluid, instead of the thin, clear, watery fluid from a hygroma. If you are a careful operator, and have diathermy available, try excising this.

Suggesting a haematoma: associated with torticollis from neck injury at birth.

33.13 Other paediatric problems

If a male neonate, infant or occasionally older child has retention of urine with overflow, the neck of the bladder is probably obstructed by URETHRAL VALVES. These usually present in the 1st 6 months of life. They may present as retention, urinary tract infection, dribbling incontinence or renal failure. Sometimes the urinary symptoms are overlooked and the child presents with vomiting, failure to thrive, uraemia and acidosis. The urine may leak from the kidneys into the peritoneum (urinary ascites). You may feel that the bladder is distended, and you may be able to feel the kidneys. Ultrasound will demonstrate a distended bladder (38.2i) and perhaps also hydro-ureters and hydronephroses (38.2f). The distended bladder will disappear on catheterization. Pass a urethral catheter (a Ch6-8 feeding tube is suitable) under ketamine. Treat any infection. If endoscopic resection of the valves is not available, you can try to pass a Fogarty arterial balloon catheter in an attempt to disrupt the valves, though try to arrange endoscopy which is better. To temporize the problem, you may need to drain the bladder with a Foley catheter or suprapubic cystostomy long-term.

If a neonate has a fleshy swelling on the lower anterior abdomen weeping urine, this is a BLADDER EXSTROPHY (ectopia vesicae). In the male, there is complete epispadias, *i.e.* the urethra opening on the dorsal (exterior) side of the penis. The scrotum is wide often with maldescended testes and inguinal herniae. The anus is anteriorly placed and there is often sphincter laxity with rectal prolapse. Occasionally there is a cloacal exstrophy which includes a rectal agenesis.

Bladder reconstruction for exstrophy is a complex procedure, with results especially poor in older children. Surgery may include pelvic osteotomy and require several stages; this needs an expert! If the final bladder capacity is likely to be small, urinary diversion may be the best long-term option.

If a neonate has ambiguous genitalia, examine the baby carefully with good light in a warm room. If there is a small phallus, and a vagina or bifid scrotum, there is doubt as to the gender of the baby. Two conditions are most common:
(1) *severe hypospadias with undescended testes*, where the phallus is normal with a urethral opening in the perineum; there is no vagina or uterus.
(2) *congenital adrenal hyperplasia (CAH)*, where there is a small phallus with normal vagina and uterus: this is caused by 21-hydroxylase deficiency leading to virilization. It has an autosomal recessive inheritance and so may occur more than once in the same family, and more commonly where there is consanguinity.
This also leads to low cortisol levels: glucose, IV fluids and hydrocortisone are needed urgently!
N.B. Females with CAH have a normal reproductive potential.

A specific diagnosis in intersex states may be difficult without sophisticated karyotyping. You must be frank with parents, however, especially with regard to fertility and the sex of rearing. *You should never refer to a child as an 'in-between'*; dispel fears that malformations might lead to homosexuality.

You should aim to limit psychological disturbance in the child: before suggesting a child is a male, think of the viability of rearing him as such (especially if there is a micro-phallus, bifid scrotum or vagina) especially in terms of micturating standing up, possible sexual intercourse and social embarrassment.

Surgically, it is easier to construct female appearances than male, though this may of course not be the most important criterion. All this needs an expert. Whilst some traditions recognize a 'third gender', the choice of how to bring up a child with ambiguous gender remains complex.

If a neonate is born with a large solid mass below the sacrum, it is probably a SACROCOCCYGEAL TERATOMA. This is a solid mass protruding usually from the coccyx, and displacing the anus anteriorly. It may extend into the pelvis, and its malignant potential increases rapidly with age (>30% at 1 month) and with internal extension. There may be rudimentary limbs growing from the teratoma, and it may contain well-differentiated foreign tissues, especially teeth but also including brain! Elevated levels of α-fetoprotein strongly suggest malignant change.

If possible, preserve a blood sample for an initial α-fetoprotein baseline level. Ultrasound will demonstrate pelvic or intra-abdominal extension, and distinguish from a sacral myelomeningocoele (33.10) which is higher up. Pressure on a teratoma will cause no bulging of the anterior fontanelle.

If the tumour is intact and the baby stable, there is no need for immediate operation. You will reduce the risk of infection by operating within 24h of birth because the bowel is not yet colonized, but surgery will be more technically difficult.

Don't wait long though, because a tumour, benign at birth, is often malignant by the age of 2 months.

EXCISION OF SACROCOCCYGEAL TERATOMA (GRADE 3.5)

Place the baby prone with legs abducted and the lower abdomen raised on a small sandbag (33-21).

Pack the rectum with paraffin gauze to ease its identification in the operation. Make sure you cross-match blood.

REMOVAL OF SACROCOCCYGEAL TERATOMA

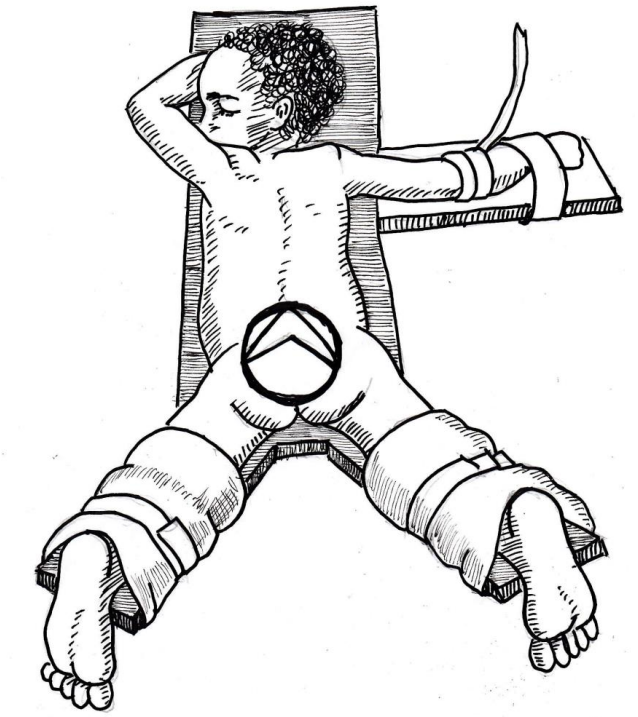


Fig. 33-21 OPERATION FOR SACROCOCCYGEAL TERATOMA. Showing the upper chevron incision, a 2cm-long apical extension, and a lower chevron incision.
After Lister J, Irving IM. Neonatal Surgery, Butterworth 3rd ed 1990 p.148 Fig 14.11.

Make a chevron (inverted V) incision at the upper limit of the teratoma, and extend it cranially at the apex for 2cm. Divide between the 4th and 5th segments of the sacrum, removing tumour with the coccyx and 5th sacral segment. Ligate and divide the middle sacral vessels, taking care to avoid heavy bleeding, and then dissect the tumour from the lateral skin flaps. Good clearance is necessary, and this is difficult when the tumour is wrapped closely round the ventral aspect of the rectum, and when the tumour cannot be readily distinguished from ischiorectal fat.

Mobilize the tumour completely, and then incise the skin at its lower border with another chevron, some distance above the anus, and so remove the teratoma. Repair the *levator ani* (26-1) behind the rectum and hitch it up to the presacral fascia. Close the skin in an inverted Y shape with 4/0 nylon, with a suction drain *in situ*, and leave it usually in place for 48h.

Nurse the baby prone post-operatively (as for myelomeningocoele, 33-16) till you remove sutures after 10-14 days. Do 6-monthly follow-up rectal examinations to detect presacral recurrences, as well as serial α-fetoprotein levels, if possible.

If a neonate (or older child) has respiratory difficulties and a radiograph shows bowel or stomach in the chest, this is due to a DIAPHRAGMATIC HERNIA. This may be congenital or, rarely, traumatic.

Presentation is usually later in childhood with respiratory distress on exercise. Diagnosis is evident on chest radiography and ultrasound. *Avoid ventilating the child by a mask*, because air inevitably introduced into the stomach will make thoracic compression of the lungs worse.

Repair the defect in the diaphragm through the abdomen with non-absorbable mattress sutures, *taking care not to damage branches of the phrenic nerve*. Pulmonary hypoplasia or pneumothorax are hazards and often demand ventilatory support. Older children with this condition do better, as their lungs are better developed.

If two neonates are conjoined ('Siamese twins'), this is a very rare instance. The join may be trivial or involve vital structures like heart, lungs, brain etc. The publicity gained by such cases may be advantageous to bring support to your hospital! Prompt investigation and treatment is mandatory before you can decide what sort of surgery is feasible.

If a baby is born with extra limbs, these may be surprisingly easy to remove. However, get some radiographs done and try to get advice. If there is a good chance of improving function, and at the same time making the baby look normal, encourage the parent.

CAUTION! This does not apply to fingers and toes: don't be tempted to remove extra digits until a child is older (32.21).

N.B. Other childhood problems are discussed elsewhere:

amblyopia and squint (28.9),
ascaris infestation (12.5),
bladder stones (27.17),
Burkitt's lymphoma (17.6),
cancer pain control (37.2),
cancrum oris (31.5),
circumcision (27.29),
cleft lip (31.7),
club foot (32.10),
congenital glaucoma (28.6),
contractures (32.1),
corneal scarring (28.4),
corrosive oesophagitis (30.3),
deciduous teeth (31.1),
extra digits (32.21),
eye tests (28.1),
foreign bodies in the ear (29.6),
foreign bodies in the nose (29.11),
foreign bodies in the throat (30.1),
haematocolpos (23.17),
hearing tests (29.2),
hypertrophic pyloric stenosis (13.6),
inguinal hernia and hydrocoele (18.5),
intussusception (12.7),
maldescended testis (27.27),
myopia (28.8),
nephroblastoma (27.35),
noma (31.5),
osteomyelitis (7.3),
otitis media (29.4),
painful hip and limp (32.14),
pelvi-ureteric junction obstruction (27.14),
phimosis and paraphimosis (27.30),
physiological goitre (25.4),
poliomyelitis (32.7),
proptosis (28.11),
pyomyositis (7.1),
rectal prolapse (26.8),
rectovaginal fistula (23.17),
retinoblastoma (28.16),
spinal TB (32.4),
testicular torsion (27.25),
tongue tie (31.9),
tracheostomy (29.15),
trachoma (28.13),
umbilical hernia (18.10),
urethral stones (27.18).

34 Surgery of the skin & soft tissues

34.1 Hypertrophic scars and keloids

A surgical scar, especially if it is on the face, should be nearly invisible if made along Borges's lines (34-1). These are relaxed skin tension lines and differ from the Langer cleavage lines, especially on the forehead, mid-cheek, scalp and lateral to the eyes, and over joints. Sometimes a scar becomes very visible indeed as the result of hypertrophy and keloid formation. Both these processes can follow surgery, tattooing, infection or almost any breach of the skin surface. Both cause large scars, and are identical histologically, but they behave differently. Keloids are difficult to treat. If you excise one through normal skin and graft the gap, it is likely to recur round the edges of the graft, or in any gaps or splits within the graft. Both a hypertrophic and a keloid response are more likely if a wound is infected, contaminated by foreign material (even monofilament sutures), or under tension.

TYPES OF SCARS AND LANGER'S LINES

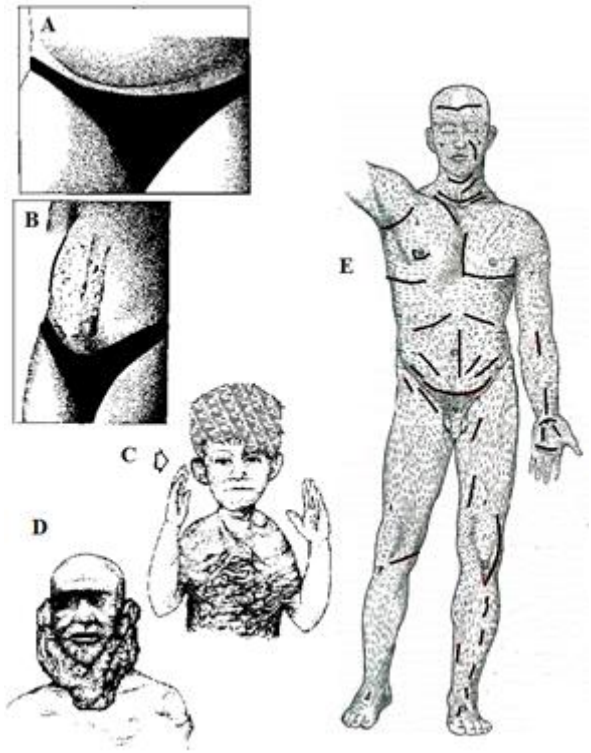


Fig. 34-1 TYPES OF SCARS.
Both patients had laparotomies c.8 months previously: A, the Pfannenstiel incision in the skin crease has healed almost invisibly. B, the vertical scar has formed a keloid. C, hypertrophic scarring on the chest after burns. D, keloid neck scar. E, incisions along Borges's lines are not under tension and form less keloid: they differ from Langer's lines on the face and joints.
After Bowesman C, Surgery and Clinical Pathology in the Tropics, Livingstone, 1960 with kind permission.

(a) Differential diagnosis may be difficult early on.

Suggesting a hypertrophic scar (34-1C): abnormal uniform growth starting within weeks of injury, growth restricted to the confines of the original scar, darker than surrounding skin, spontaneous regression in 6 months to 3yrs to become broad, soft, thin, and level with the surrounding skin, anywhere in the body, very common in burns scars, itching commonly which may be severe.

Give reassurance that it will eventually regress naturally. *Never operate during the active phase*. If you decide to operate, do so during the mature phase, ≥3yrs after the original wound. Then, excise the scar, and apply the preventive measures above. Considerable improvement is possible.

Suggesting a keloid (34-1D): onset delayed for months/yrs, invasion of the surrounding skin, growth stops in due course but there is no regression, localized commonly on the earlobes, chin, neck, presternal area, and the midline, not uncommon in young black patients, uncommon in burn scars, very uncommon below the groin and where vascularization is poor.

If diagnosis is difficult, remember that a keloid becomes increasingly raised, and extends beyond the confines of the original scar.

(b) Prevention of keloids and hypertrophic scars

Minimize tension in the scar by planning incisions in skin creases where possible. If you have to cut across a crease, use a Z-plasty (34-4).

Avoid scars in areas that are normally under tension:
(1) in the neck especially,
(2) in the coronal plane in the upper arm, especially its lateral side,
(3) in the upper back.

CAUTION! Midline sternal and abdominal scars and longitudinal incisions in the arm are particularly likely to develop keloids: they cross skin creases.

Maintain careful asepsis, minimize trauma when you operate, and control bleeding carefully at the end of the operation. *Don't pull sutures too tight,* and *avoid mattress sutures*. *Avoid incisions in the midline and in visible sites.*

POST-OPERATIVELY
If a patient is particularly likely to develop a hypertrophic scar or a keloid, as shown by his previous history, apply pressure to the scar for 9-12 months after an operation. Ideally, a tight elastic garment (like sportswear) should be made to fit. This may not be practical, but you may be able to cut a piece of foam rubber to fit a smaller scar, and hold it in place with an elastic bandage. Tell the patient not to remove it except to wash. Unfortunately, both an elastic garment and an elastic bandage are difficult to tolerate for long, especially in a hot climate.

(c) Treatment is more successful if you start it early.

(1) For developing keloid, within 1-2 months of the injury, apply pressure and inject a 2ml suspension of hydrocortisone at each site spread out subcutaneously. Or, better, use triamcinolone 1ml at 3wk intervals x4.

(2) For an established keloid, *steroids have no effect. Resist the request to operate*. The worse the keloid, the more likely it is to recur if you excise it. Operate only on those keloids that are infected or cause functional deficit (*e.g.* over a joint). If you operate, excise the abnormal tissue *within* the keloid, leaving a margin of keloid tissue all round (34-2). Avoid sutures: use steristrips. If necessary, graft the bare area. You may be able to shave skin off the keloid and use this as a graft. All this is difficult, as is closing the wound tidily. Complete the incision and then inject steroid suspension into the scar. Post-operatively, administer 4 more steroid or triamcinolone injections at 3wkly intervals. Apply a pressure bandage or an elastic garment for 9 months: this is essential! You can use specially made compression ear-rings, but make sure they are worn rigorously, because otherwise your patient will end up with a bigger and uglier keloid than before!

A 'SHAVING' METHOD FOR KELOIDS

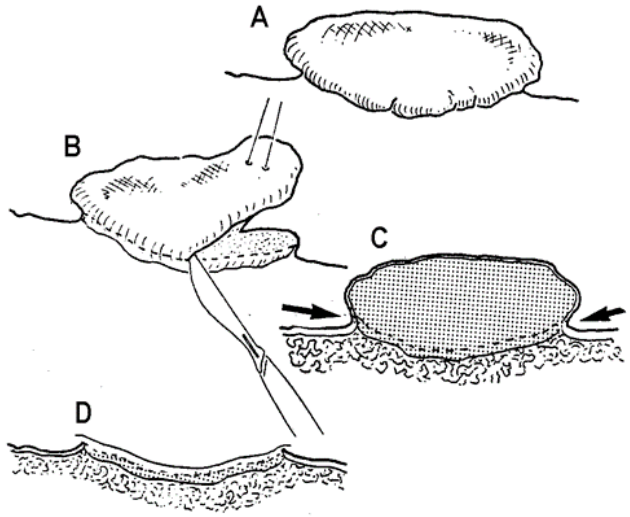


Fig. 34-2 A 'SHAVING' METHOD FOR KELOIDS.
A, keloid mass. B, the way to shave it off. C, the plane through which to remove it. D, the graft in place ready for dressing.
After Bowesman C. Surgery and Clinical Pathology in the Tropics, Livingstone, 1960 Fig. 194 with kind permission.

CAUTION!
(1) Use a sharp knife.
(2) *Don't pull on the keloid as you excise it*, or you may enter the subcutaneous tissue. Instead, if necessary, depress the surrounding tissues.
(3) *Don't use diathermy.*
(4) *Don't place sutures.*

34.2 Skin contractures

Whilst all contractures are functionally disabling, the approach to their treatment depends on whether it is the skin and underlying tissue or the muscles and joints that are primarily involved. Obviously skin contractures can lead to joint stiffness eventually, and joint stiffness to muscle atrophy and tightening of the skin; before these end-stage developments occur, you can still do much to alleviate the problems. However, try to determine whether the main problem is in the skin or the muscle and joint (32.1). Infected wounds and burns, especially across skin creases, will cause skin contractures, whilst ischaemia, poliomyelitis, leprosy, neuropathies, cerebral palsy, severe soft tissue and bony injuries, soft tissue and bone infections and arthritis of all kinds will lead to muscle and joint contractures.

Such may require repeated complex interventions which may daunt you. Consider gradual stretching of such contractures by using a distracting external fixator at a rate of 2mm/day (32.1). This is usually only possible after releasing a skin contracture, but may well avoid complex tenotomies.

Remember patients need close follow-up and prolonged splinting to prevent recurrent contractures.

Another odd cause of hand contracture, associated with excess alcohol intake, is Dupuytren's which causes a flexion deformity especially of the pip joint of the middle and ring fingers, particularly in men of 40-50yrs. Treat it like other contractures.

(a) Broad burns contractures

If you wait for the expertise of a plastic surgeon in a referral hospital, a burn contracture is likely to become an almost incurable deformity. If you are persistent and careful, you will not find them as difficult to treat in a district hospital as you might expect. You have skin loss to cope with, so they are more difficult than polio contractures (32.7). The post-operative care is half the battle. Insist on taking graft dressings off *yourself*: do this gently, with much soaks of water!

Contractures of the larger joints are not too difficult, but those of the hand are tasks for an expert; yet you may have to try. *They are certainly not the contractures to start with*. Contractures on the palm are slightly less difficult than those on the back of the hand, where the mcp joints readily become hyper-extended, as part of a claw hand. The joints are close to the surface and are involved early.

Fortunately, a child's joints don't become stiff nearly so easily as those of an adult. After you have grafted the flexor surfaces of a child's fingers, you can safely immobilize them in extension. Contractures may be linear or, more commonly, broad.

Excise linear contractures with a Z-plasty. Release broad contractures widely without excising all the scar tissue; then graft the bare area with a medium or thick split skin graft. Splint the limb in a position opposing the contracture, and start exercises as soon as the graft has taken (c.10-14 days).

Use sheets of thick split skin, meshed if necessary. The result is less good, though, than if you use unmeshed full-thickness graft. Make children your 1st priority: you will be much less successful with adults.

Don't try to relieve burns contractures by using serial casts (32.1).

AXILLARY AND NECK CONTRACTURES

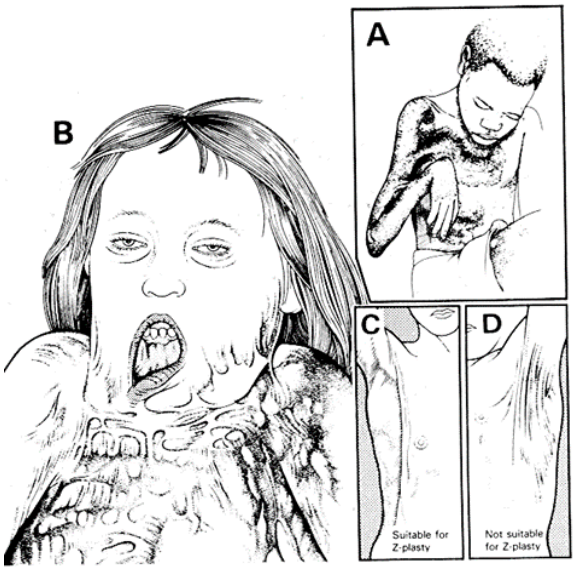


Fig. 34-3 AXILLARY AND NECK CONTRACTURES. A, contractures of the axilla, elbow and wrist. If the joints had been splinted in the positions of function, their contractures would have been prevented. B, contractures of the neck and axilla. C, a narrow contracture which is suitable for simple Z-plasty. D, a broad contracture which needs excision and skin grafting.
Kindly contributed by Jack Cason, Ian MacGregor and Peter Bewes.

(1) MANAGEMENT OF A BROAD CONTRACTURE (GRADE 2.3)

Wait until a burn has healed completely. This operation is not a task for the minor theatre. Administer iron and folate so that the Hb is >100g/L. Have blood cross-matched if you cannot use a tourniquet (3.4).

Infiltrate into and under the contracture a mixture of saline 80ml, 2% lignocaine 20ml, 1:1000 adrenaline 0·5ml, and preferably hyaluronidase 1500U. This solution will:
(1) demonstrate the tissue planes more clearly,
(2) allow you to separate the scar more easily,
(3) control bleeding,
(4) reduce the amount of GA needed.

Cut perpendicularly through the scar down to the subcutaneous tissue, in the middle of the contracture. Keep it under tension as you do so. If necessary, cut right down to the tendons.

Try to separate the scar from the deeper tissues by blunt dissection. Push your scissors into the tissues, then open them (4-9B). This will help you to avoid any superficial veins. It is wise not to try to excise the scar initially, either in the main part of the contracture, or at its upper or lower ends.

CAUTION!
(1) Release the contracture first, and *then* decide how much more scar tissue you need to excise. You will always need more extensive incisions than you expect!
(2) *Don't cut the deep fascia*, unless the scar tissue extends right through it.
(3) Vessels in scar tissue do not contract, so bleed a lot. So, beware of congested veins, especially in the axilla and neck.
(4) *Beware you don't expose nerves, tendons or joints that you cannot later cover!*

Carry the incision beyond the limits of the scar tissue, and beyond the axes of the joint on each side. If you don't do this, the contracture will recur. Or, make a double-Y (34-5D); this will reduce the length of the incision you need to make.

Start taking skin from the donor site. When the contracture is straightened out, you will need more skin than you expect. Cover the bare area with a sheet split skin graft, and suture it in place preferably with a tie-over dressing. Immobilize the area carefully, with splints or plaster of Paris in the position of full release of the contracture.
CAUTION!
Graft the exposed raw areas immediately, especially over joints. This will reduce the risk of the contracture recurring, and the risk of infection reaching the joint.

Remove the dressing *yourself* after 5 days. Keep the area in a night splint *for ≥ 3 months.* Maintain a regular review; you may need to make serial releases with several operations.

(2) RELEASE OF NECK CONTRACTURES

If the chin is contracted down on the sternum (34-3), *the anaesthetic problems are considerable: you cannot extend the neck to see the vocal cords*, unless you have a flexible bronchoscope over which you can thread an endotracheal tube. You may be able to use a laryngeal mask, but it is probably safer to infiltrate the scar with LA, administer ketamine, cut through the scar quickly, and then perform the intubation.

METHOD. Incise the scar transversely, if necessary almost from ear to ear. Carefully release the scar tissue by blunt dissection to reveal a huge gap in the front and sides of the neck. Try to apply a single sheet of split skin graft secured with a tie-over dressing. Immobilize the neck with the head well extended. To prevent recurrence, keep the neck in extension. Apply a soft collar as soon as the skin is soundly healed, and leave it there for at least 6 months.

A night splint is mandatory for several more months. If necessary, repeat the procedure, several times if required, to obtain a little more movement each time, particularly if the lips are involved.

(3) RELEASE OF AXILLARY CONTRACTURES

Try to restore full abduction and elevation in a single operation. If there is a broad contracture, incise the scar as above, and abduct the arm. Apply a large medium-thickness split skin graft to the bare areas, and secure it with a tie-over dressing. Cover this with plenty of dry wool, and bandage this (preferably with crepe bandages) to include the whole arm as well as the axilla and chest.

In a small child, a large ball of cotton wool bandaged into the axilla may hold the arm in the right position.

In an older child or an adult, raise the head and back on a suitable support as for a hip spica, and apply a plaster shoulder spica to include the arm and hand, with the arm at 90° from the chest, the elbow flexed, and the wrist dorsiflexed. This is the most comfortable position.

CAUTION!
(1) *Don't injure the axillary vessels or nerves.*
(2) *Don't hyper-abduct the shoulder*, as you may injure the brachial plexus.

(4) RELEASE OF ELBOW CONTRACTURES

A large scar may involve the whole flexor surface of the elbow. Make a cautious transverse incision across the fold of the elbow, starting laterally, and avoiding any congested veins. If the whole width of the elbow is involved, extend the incision into healthy tissue on each side. Find a fatty layer and then work gently medially. If you have found the right fatty plane, this should free up the scar tissue. When the incision is complete, divide any deeper strands of fibrous tissue. Fill the large diamond-shaped gap with a medium-thickness split skin graft. Secure it with a tie-over dressing. Immobilize the extended and supinated elbow in a cast which should also immobilize the wrist.

When the wound has healed, apply a cast of the elbow alone in extension for at least 6-12wks. *You are operating for a flexion contracture, so lack of flexion will not be a problem.*

(5) RELEASE OF HAND CONTRACTURES

If the contracture is mild, a dynamic splint may cure it, or at least partially correct the deformity, so that operation will be easier.

If the wrist is hyperextended, divide the scar transversely, and apply a medium-thickness split skin graft: *beware of the median nerve and ulnar nerve and artery!*

If the mcp joints are hyperextended as part of a claw hand, this is a particularly difficult contracture, because the capsules of the joints may need opening up and freeing. Make transverse incisions over their dorsal surfaces, flex them, graft the gap, and splint the hand in the position of function.

If there are flexion contractures of the fingers, incise them transversely maximally *taking care not to damage the digital nerves and arteries*, and fill the gap with a full thickness or a thick split skin graft sutured into place.
For a child, splint the fingers in extension for 3 months, or the contracture will recur. To help the cast stay in place, apply it with the wrist extended. Examine the cast daily at first, and later weekly, to make sure it has not slipped. *For an adult, don't immobilize the extended fingers for >10 days*. Use dynamic splints and night splints.

If there is a very severe finger deformity, you may need to amputate the finger, or arthrodese it in the position of function.

(6) RELEASE OF GROIN, KNEE, ANKLE, AND FOOT CONTRACTURES

Follow the general method, as described above, taking care to extend the incision well beyond the axis of the joint. Tendons may bow-string across the knee and prevent full extension: in this case you will need to make a tenotomy and tendon lengthening (32.8,10).

(b) Difficulties with burns contractures

If you cannot get sufficient release of a contracture in a single stage, release it as much as you can; splint it, leaving it open, and release it further after a few days, then graft it.

If there is an ulcer within a scar, think of squamous carcinoma: excise it with adequate margins and send it for histology (34.5).

MOST SEVERE CONTRACTURES ARE THE RESULT OF POOR CARE

(c) Narrow burns contractures

A Z-plasty is a useful way of releasing a contracture, if it is narrow enough. It is not an easy method, but if your result is not perfect, you can always graft any bare areas that remain. Good results are easier to achieve than with wide contractures which need grafting.

Make a Z-plasty by excising the scar and then cutting 2 flaps in the form of isosceles triangles which share one common limb, and so form a Z. When you extend the limb, the triangular flaps will change their positions spontaneously. Initially, the 2 triangles together form a parallelogram, with its shorter diagonal in the line of the contracture, and its longer diagonal transversely across it (34-4C).

Releasing the contracture and transposing the 2 triangles changes the shape of the parallelogram, so that the new contracture diagonal is the same length as the transverse diagonal was before (34-4D).

The difference in length between the 2 diagonals determines the amount of lengthening in one direction and shortening in the other.

Transposing the triangular flaps increases any extra elasticity that there may be across a scar by at least ⅓, and changes its direction of function.

Expert plastic surgeons find this useful for changing the direction of a facial scar, so that it lies along a Borges line (34-1): if you make one large 'Z', all the transverse shortening and all the tension is concentrated in one transverse diagonal (34-4E,F). But, if you make multiple 'Zs', the lengthening is additive, because all the contracture diagonals are in the same line, but the transverse shortening is spread out over several smaller 'Zs' (34-4G,H). In practice, you will not achieve quite as much lengthening with multiple 'Zs' as you would expect, but it is still a very useful method.

Unfortunately, as most burns usually cause scarring in all directions, there may be no lax tissue available, making a Z-plasty impossible. Where it is suitable, however, it is very effective indeed.

(d) Z-plasty (GRADE 1.4)

INDICATIONS
A narrow contracture of the axilla, elbows, fingers, knee or neck, especially one of the bowstring type, provided the surrounding tissues are reasonably lax and undamaged. If there is no transverse slack tissue to start with, a Z-plasty will not work on its own, but it still may be helpful combined with excision of scar and skin grafting.

A single 'Z' based on the whole length of the contracture. This is useful if, at right angles to the contracture, the tissue is lax enough to allow some shortening in the transverse axis, and the bowstring is reasonably linear, not extending laterally: if this is the case, multiple 'Zs' would be wiser.

Multiple 'Zs' (W-plasty). This is necessary if the available lax tissue is not available at one point, but is spread out along the length of the scar.

SINGLE 'Z'
Use a pen to draw your proposed Z-plasty on the skin; the longer the transverse diagonal, the more length you will gain. Its length will however be limited by the amount of loose tissue available at the sides.

You have 2 ways of choosing the direction of the Zs. Select the best one by drawing equilateral triangles on either side of the central limb, in both of the possible ways. Choose the flaps which:
(1) have the better blood supply,
(2) avoid scarring across the base,
(3) will give the best cosmetic result, and
(4) are likely to rotate most easily.

CAUTION!
(1) Angle the flaps as near to 60° as you can. Use a pre-cut 60° pattern. *The temptation is to make the angles too small: this reduces the length gained.*
(2) Make the sides the same length as the central limb, except that if one flap is scarred, cut it a little longer than the other.
(3) If you are worried about the possible viability of a flap, curve it a little (34-4B).
(4) The tip of a 'Z' is the part most likely to necrose, so make sure you cut it deep enough. If necessary, include some of the underlying scar tissue.

Z-PLASTIES

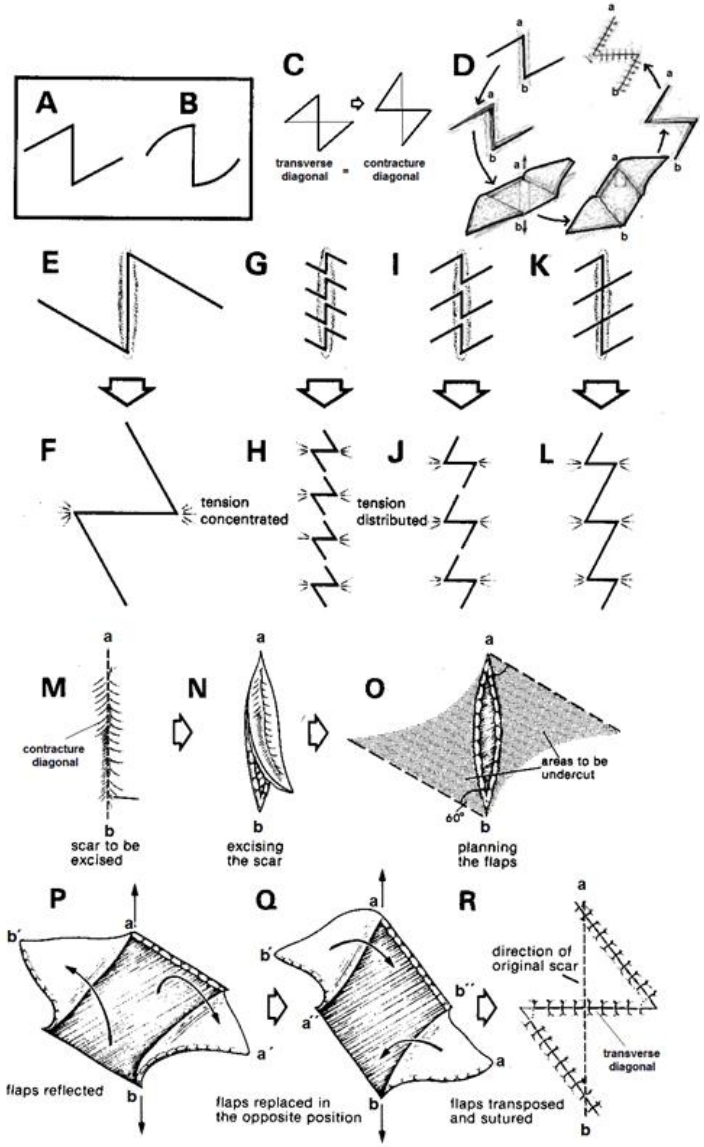


Fig. 34-4 Z-PLASTIES.
A, how flaps are usually cut. B, how to cut them if you are doubtful about their blood supply. C, initial layout of incisions, and how the flaps will transpose. D, final result: the contracture diagonal has lengthened and the transverse diagonal has shortened in equal measure. E,F, single 'Z' showing how the lateral tension is concentrated in a single line. G,H, multiple 'Zs' distributing the lateral tension. I,J, same but with fewer larger 'Zs'. K,L, 'Zs' joined up. M, scar. N, excise the scar. O, plan the flaps. P, raise the flaps. Q, transpose the flaps. R, the flaps in place: note the special suture at the apices.
After MacGregor IA. Fundamental Techniques of Plastic Surgery, Churchill Livingstone 1980 with kind permission.

MORE BURNS CONTRACTURES

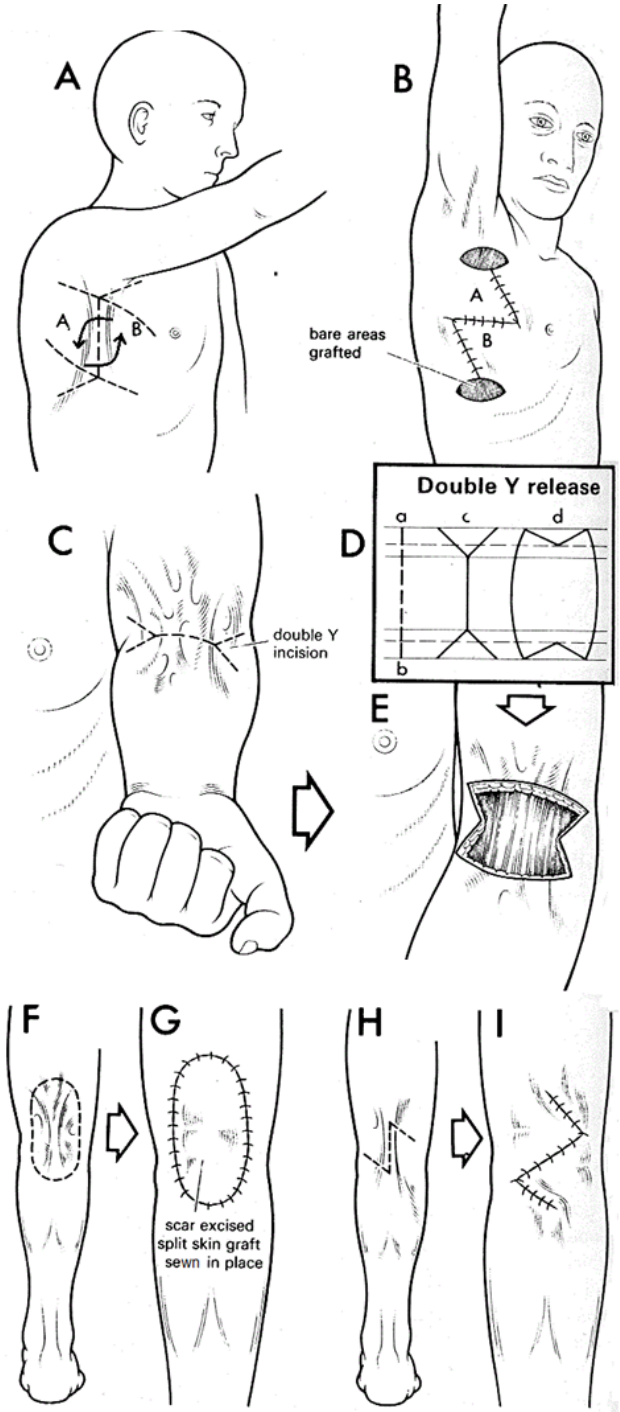


Fig. 34-5 MORE METHODS FOR BURNS CONTRACTURES.
A,B, combining Z-plasties and skin grafting; make the Z-plasty with transverse incisions at each end of it. Open these to leave raw surfaces ready for grafting. C, contracture of the elbow showing a 'double Y' release incision. D, 'double Y' release. This will release the ends of the contracture maximally, without increasing its length: (a-b) the width of the scar, (c) 'double Y' to cover the full width of the elbow, (d) full release of the incision. E, 'double Y' incision ready for grafting. F,G, total excision of a scar with a split skin graft sewn in place. H,I, Z-plasty to treat a narrow popliteal contracture.
By the kind permission of Irving Feller & William C. Grabb, US National Institute of Burns Medicine.

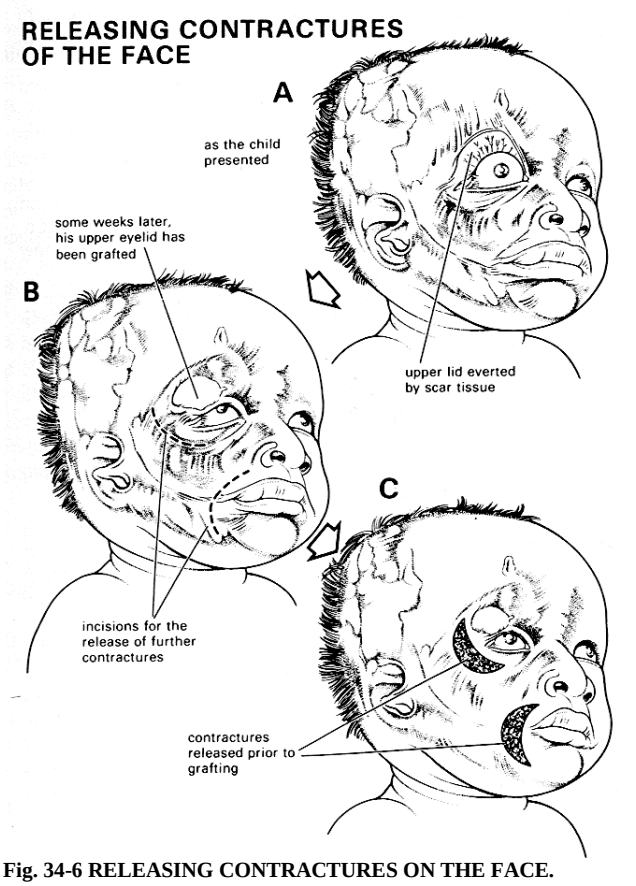


Fig. 34-6 RELEASING CONTRACTURES ON THE FACE.
Make sure your grafts are big enough. These grafts are on the small side, particularly the first one. If you are experienced, you can avoid the 'trap-door' appearance of curved scars by making multiple Z-plasties.
Kindly contributed by Randolph Whitfield II.

34.3 **Sebaceous** (epidermoid) **cysts**

When the mouth of a sebaceous gland is blocked, a cyst results which is filled with creamy yellowish white sebum, into which you may be able to make a dent with your finger. Typically the cyst has a punctum at its centre. Sebaceous or epidermoid cysts are most common on the face, scalp and back, as hemispherical firm or elastic swellings, with no obvious edge, which are adherent to the skin.

Three complications may follow:
(1) A sebaceous cyst can become infected; this makes it enlarge and become red and painful. Recurrent infection makes it adhere to the surrounding tissue, and become more difficult to remove. Often then you need to drain the abscess, and remove the cyst later when infection has resolved.
(2) It can ulcerate, and discharge its contents. The lining membrane which is left can then resemble an epithelioma.
(3) Its contents can escape, and become hard and form a sebaceous horn, which needs excision.

(a) Excision of a sebaceous cyst (GRADE 1.2)
Using LA, incise over the swelling, in the direction of the natural lines of the skin. This is particularly important in the face. *Don't use an elliptical incision*, which may later be difficult to close without tension. Deepen the incision very carefully until you reach the edge of the cyst. Push the points of fine curved scissors between the cyst and the tissue round it, and then open them, so as to define a plane for dissection. Repeat this all round the cyst until it is free, then try to remove it intact with a snip of the scissors.
Press firmly with dry gauze for 2mins to stop bleeding. If any bleeding vessels remain, tie them off. Close the skin, leaving a small Penrose drain (4-14B) in place unless the cavity is completely dry. Remove this at 24-48h.

(b) Difficulties with sebaceous cysts
If the cyst is infected, incise and drain it but *make no attempt to excise it till the infection has settled*.

If the cyst ruptures, try to remove all of its lining by operating gently with fine instruments; if you leave some behind, the cyst is likely to re-form.

If the cyst does not have sebaceous material but hair, it is a DERMOID CYST. You find these at the lines of skin fusion in the embryo, namely in the midline or, more commonly, at the lateral or medial ends of the eyebrow where the maxillary and ophthalmic divisions of the face meet. They may extend internally a considerable distance, so *don't underestimate this lesion*, as you may need to hollow it out of the frontal bone!

If the 'cyst' turns out to be a fatty lump, it is a lipoma: excise it anyway, by shelling it out.

34.4 Skin manifestations of HIV disease

Unfortunately only few of the HIV-related skin conditions (5.6) are amenable to surgical treatment. These are pyogenic granuloma, condyloma (26.6) and Kaposi sarcoma (34.10). If the lesions of *molluscum contagiosum* (which typically look like little navels) are very extensive, you can provide some relief by removing these.

(a) Pyogenic granuloma
(Lobular capillary haemangioma)

Pyogenic granuloma is a misnomer; it can occur anywhere, but is commonest on the face, fingers, or toes, as a soft or moderately firm, dull red, 1cm lump, covered with atrophic epidermis or crusts, and which bleeds easily. This trivial lesion can be misdiagnosed and thought to be a sarcoma (34.15), when all that is needed is simple excision and curettage. Its cause is unknown but may be related to antiretroviral treatment. An antibiotic is only needed rarely if there are signs of spreading infection.

'PYOGENIC GRANULOMA'

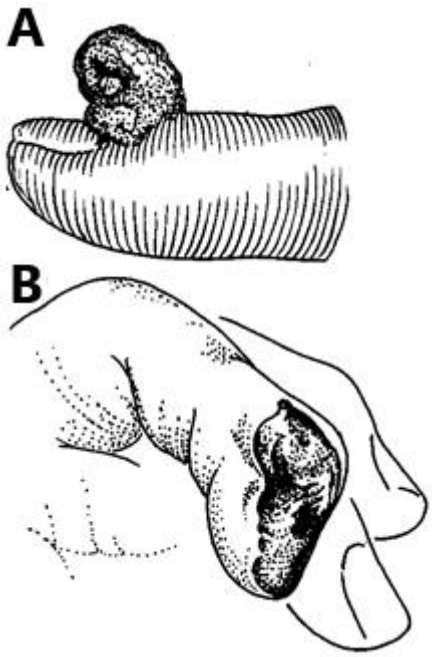


Fig. 34-7 PYOGENIC GRANULOMA (LOBULAR CAPILLARY HAEMANGIOMA).
Excise or curette these lesions. *Don't mistake them for a sarcoma!* If there is any doubt about the diagnosis, take a piece for histology.

(b) Condylomata (Warts)

These can occur anywhere, but are particularly found in the perineal area from sexual contact; even in HIV+ve patients, wounds tend to heal well after excision, even if they are extensive or in the perineum (26.6), *but beware of scarring! Beware also of malignant change.*

34.5 Nodules; basal and squamous carcinoma

You should aim to excise any suspicious skin lump, especially if you think it looks malignant (with rolled everted edges) or might become so, particularly if you can do the operation without disfigurement under LA. *Don't wait till the signs of malignancy are obvious and far advanced!*

(a) Skin nodules

Most of these may ulcerate, and have many causes, some of which are specific to certain areas or people.

Remember many small ulcers are caused by insect or tick bites; these may result in various types of fever, *e.g.* rickettsial typhus or *trypanosomiasis*.

Some ulcers, *e.g.* gouty tophi on elbows, are metabolic in origin, and need medical treatment as well as excision for diagnostic or cosmetic purposes. Many are inflammatory or infective, however, and may have different treatments:

Name	Cause	Agent	Features	Treatment
Bartonellosis (verruga peruana) *(bacillary angiomatosis)*	*B. bacilliformis* *B. quintana, B. henselae*	sandfly in Andes mountains lice; fleas on cats	raised purple nodules on limbs and face, fever, haemolytic anaemia, red berries on skin	chloramphenicol or ciprofloxacin erythromycin or doxycycline
Chromo(blasto)mycosis	fungi spores (Medlar bodies: thick-walled brown sclerotic cells on scrapings + 10% H_2O_2)	thorns in Madagascar, Japan, Amazon and Central America	warty plaque on feet with lymphoedema	itraconazole
Cysticercosis	*Taenia soleum* (pork tapeworm)	ingestion of under-cooked pork	subcutaneous cysts + convulsions, visual loss	albendazole or praziquantel
Dracunculiasis (34.8) (Guinea worm)	*D. medinensis* (Sudan, Chad, Mali, Ethiopia)	contaminated drinking water	papule or cyst with long worm	immersion in water to discharge worm, or slow extraction
Kerion (inflammatory ringworm)	*Tinea* (various)	contact	pustular scalp mass	ketoconazole or griseofulvin
Leishmaniasis (34.7)	*L. braziliensis*	sandfly	itchy red papule, then firm, elastic scaly nodule with softened centre	ketoconozale and/or miltefosine; honey application for early lesions
Leprosy	*Myco. leprae*	long contact	facial, extensor arm nodules and sensory loss	rifampicin, dapsone and clofazimine
Mycetoma (Madura foot, 34.11)	*Actinomycetoma* (Sudan, Somalia, Mexico, India) + *Eumycetoma* spp. (dry areas)	thorn/stone	pustular mass + sinus discharging grains + swelling	trimethoprim itraconazole
Myiasis (cutaneous)	larvae (maggots)	deposition from botfly, tumbu fly, blow fly, or *hypoderma*	itchy nodule with intradermal movements (larva migrans); also in wounds, body orifices	topical oil or jelly and extraction by forceps
Onchocerciasis (34.8)	*O. volvulus* (sub-Saharan Africa, Yemen, Central and South America)	blackfly, near fast-flowing streams; endosymbiosis with *Wolbachia*	itchy non-suppurating nodule on pressure points + hanging groin, and blindness	ivermectin and doxycycline
Sporotrichosis	*S. schenckii*	abrasion	necrotizing ulcerating nodules spread along lymphatics	potassium iodide, or itraconazole *(not in pregnancy)*
Syphilis	*Treponema pallidum*	sexual contact	brown 2° nodule on palm, sole or face	benzathine benzyl-penicillin or doxycycline or erythromycin
Tuberculosis (lupus vulgaris) (Buruli ulcer, 34.9)	*Myco. tuberculosis* *Myco. ulcerans* (West and Central Africa)	inhalation water insects	painless nodule, then ulcerating and scarring nodule quickly ulcerating causing tissue necrosis	ethambutol, isoniazid, rifampicin, and pyrazinamide streptomycin and rifampicin
Tungiasis	*T. penetrans* (Brazil, Madagascar, Nigeria)	flea infestation especially from walking or lying on sand	fibrous painful nodule on foot, or exposed body surface	topical dimeticone, injected into lesions and tetanus prophylaxis

(b) Basal carcinoma

Basal carcinomas are common in areas of white skin (including particularly albinos) exposed to the sun (34-8). Melanin in dark skin protects it from the effects of sunlight. The earliest stage is a raised nodule, but the usual presentation is of an old, or middle-aged, man complaining of a small ulcer on the face or scalp, commonly near the eye, which continues to break down and never really heals. It has a raised rolled edge, grows slowly, and eventually erodes into muscle, cartilage, or bone. Growth is slow and *metastases don't occur*. Prognosis is excellent if you treat lesions early, whether by surgery or radiotherapy.

DIFFERENTIAL DIAGNOSIS includes:
(1) kerato-acanthoma (a solitary firm nodule with central ulceration, initially rapidly growing, then resolving),
(2) skin tuberculosis,
(3) fungal lesions.

EXCISION OF BASAL CARCINOMA (GRADE 1.2)
Excise the lesion with at least 1cm of normal tissue all round it. If you cannot do this, try hard to get help to make an adequate excision because otherwise it will recur and spread extensively. It will not spread to the regional lymph nodes, so there is no need to excise these. Send a specimen for histology. For very small lesions, radiotherapy offers no advantage over surgery, because the diagnosis has to be confirmed by biopsy anyway; for middle-sized or large lesions, it is of some cosmetic value.

(c) Squamous carcinoma

Squamous carcinomas are also common in areas of white skin (including particularly albinos) exposed to the sun. They are rare in dark skins, except as a complication of chronic ulcers (34.9), burn scars, radiation, condylomata in HIV+ve patients, hidradenitis outside the axilla, xeroderma pigmentosum (autosomal recessive sensitivity to UV light) or arsenic exposure (*e.g.* in miners, glass workers or from contaminated groundwater).

Squamous carcinomas develop in adult life or, occasionally, earlier (especially in albinos). They are low-grade tumours which spread to the regional nodes late, and rarely metastasize through the blood. Ulcerating lesions are late stages; typically they have irregular, raised, round, everted edges and indurated bases, which soon become attached to deeper structures, and may erode the bone underneath, causing a pathological fracture.

In theory, prevention is simple: by covering exposed white skin, and treating chronic ulcers and burns to make sure they heal. Unfortunately, albinos from disadvantaged families often face serious discrimination.

RADIOGRAPHS. If the lesion is overlying bone, get a radiograph. A translucent area in the bone under the ulcer shows that bone is being infiltrated. Thickening of the bony cortex and trabeculae, however, indicate secondary chronic osteitis, caused by infection.

SKIN CARCINOMAS

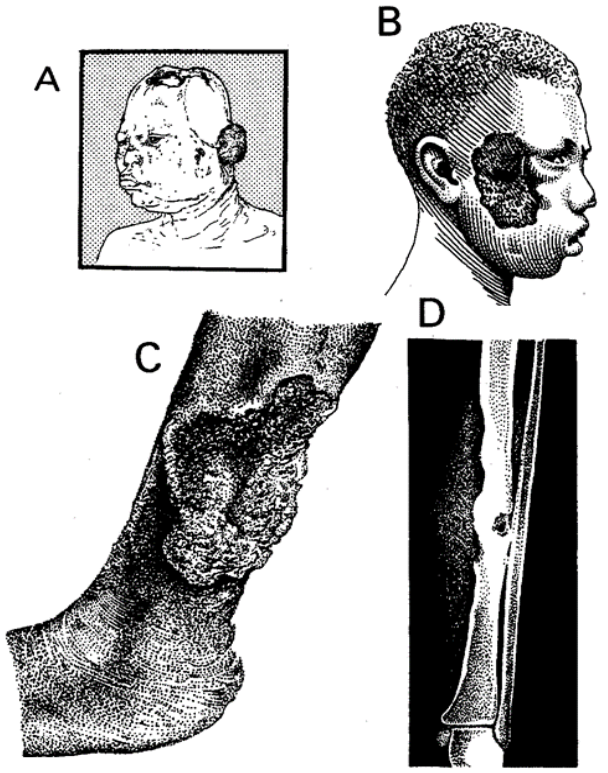


Fig. 34-8 CARCINOMAS OF THE SKIN, squamous and basal carcinoma.
A, this albino has already had several squamous carcinomas. One has eroded the skull; it was successfully excised, and the dura grafted, leaving a large depression. Another large one has now destroyed the ear. B, another albino with a large basal carcinoma (rodent ulcer) of the side of the face. C, a squamous carcinoma in the leg. Note its raised edges. D, radiograph of the same lesion, showing bone destruction and sclerosis.
B, after Bowesman C, Surgery and Clinical Pathology in the Tropics, Livingstone, 1960 with kind permission.

DIFFERENTIAL DIAGNOSIS includes:
(1) A benign chronic ulcer (34.9) – the distinction can be difficult clinically and histologically.
(2) A tuberculous ulcer.
(3) Yaws (34.9).
(4) Syphilis.
(5) Pyogenic granuloma (34.4).
(6) Fungal lesions.
(7) Kaposi sarcoma (34.10).
(8) Amelanotic melanoma (34.6).

EXCISION OF SQUAMOUS CARCINOMA
(GRADE 1.2)
CAUTION! Always confirm the diagnosis histologically first, before starting treatment.
Try to excise a squamous carcinoma with any malignant infiltration around it. So remove at least a 0·5cm margin of macroscopically normal skin around the lesion, and at least a 0·5cm margin underneath it.

If there is a large defect but a satisfactory base, for example muscle, excise any deep fascia and apply a split skin graft. Tie-over sutures are useful to keep the dressing in place.

If the base is not suitable for grafting (if it is connective tissue, fat, tendon, or infected), wait 5-7 days till granulations are satisfactory, and then apply a split skin graft. *(You can store graft wrapped in paraffin gauze in a refrigerator for c.10 days.)*

If bone is exposed, chisel away the cortex until you reach a bleeding surface, wait 7-14 days for granulations to form, and then apply a graft. Occasionally, you can close the defect with either a rotation, a transposition or a myocutaneous flap. If the carcinoma is infiltrating bone so much that the bone would fracture if you remove enough of it to excise the lesion properly, amputate at the first joint proximal to the lesion instead.

Lymph nodes involved by a squamous carcinoma
If the regional lymph nodes are not hard or matted together, leave them. Do a careful follow-up. *Don't do a block dissection prophylactically*, because there is a 10% chance that lymphoedema (34.12) will develop afterwards, and it will not improve the prognosis.

If the nodes are enlarged, and you think that this is caused by secondary infection, use cloxacillin, and wait ≥2wks. If they don't respond, biopsy one, and make a careful follow-up.

If you think that the nodes are involved clinically but are not fixed to deeper structures, **or there is an advanced ulcer-cancer** (most commonly in the groin), perform a block dissection (17.8), and wide excision of the primary, at the same time. The prognosis following wide excision of the primary and block dissection is good.

If nodes in the groin are enlarged and fixed to the femoral vessels, leave them. They will fungate, but there is little you can do about this unless you perform an extended excision with a vascular graft.

If the inguinal nodes become enlarged when they were normal previously, or increase in size after the amputation stump has healed completely, but are still mobile, perform a block dissection.

DIFFICULTIES WITH SQUAMOUS CARCINOMAS
An albino is particularly prone to multiple squamous carcinomas. Advise long skirts or trousers, long-sleeved high-necked shirts, wide hats, tinted glasses, and the avoidance of unnecessary exposure to the sun, especially between 11am and 3pm. The patient must report any new lumps or bumps immediately. Advise the use of the fruit of the sausage tree (*Kigelia africana*): soften the skin of the fruit with 10% urea or 2% salicylate, and use this as a protective cream. *Keep a careful follow-up of your albino patients:* they may be ostracized socially.

Use lotions with high protective factor (30+ or 50+): they are quite expensive, so instead you can use zinc oxide on the lips as this blocks the sunlight. You may be able to abort pre-cancerous lesions with topical 5% fluorouracil. Otherwise, treat squamous carcinoma in the same way as above.

If the lesion is bleeding profusely, apply hydrogen peroxide or 10% formalin soaked onto gauze, avoiding contact with normal skin.

34.6 Melanoma

In a black patient, malignant melanomas arise only from non-pigmented parts of the skin: the soles of the feet (most commonly), the palms of the hands, the nail beds, and the mucosa. In a white patient, a melanoma can arise anywhere, usually in a pre-existing naevus, especially after long exposure to sunlight, commonly on a man's trunk or a woman's legs, or, rarely, from the choroid plexus of the eye. In all skin colours, malignant melanomas only occur after puberty; most are pigmented, but a few are amelanotic. They may occur at the junction of depigmented and pigmented skin in severe cases of vitiligo.

They spread:
(1) by local infiltration, usually horizontally at first, or as satellite nodules, but later vertically into the deeper tissues,
(2) to the regional lymph nodes; deposits may also grow in lymphatic channels on the way there,
(3) through the bloodstream.

Treatment is by wide surgical excision; there is no effective radiotherapy or chemotherapy. Amputation may be necessary to obtain clearance.

(a) Prevention
(1) Excise any elevated mole (pigmented lesion) >0·5cm in diameter which shows any sign of growth, colour change, bleeding, ulceration or itching.
(2) *Don't expose white skins, including those of children and especially albinos, to sunlight.*

(b) Differential diagnosis includes a benign naevus, a pigmented seborrhoeic wart, a squamous papilloma or carcinoma (34.5), a capillary cavernous haemangioma (33.12), pyogenic granuloma (34.4) and Kaposi sarcoma (34.10).
N.B. Histologically, the diagnosis can be difficult.

Suspect that a black patient has a melanoma if there is:
(1) Any growing dark lesion on the soles of the feet, on the palms of the hands, or in the nail beds, particularly on the big toe. The commonest site is at the junction of the deeply and lightly pigmented areas on the hands and feet.
(2) A deeply pigmented lesion on the sole of the foot, >2cm in diameter, whether or not it is ulcerated.
(3) An ulcerated lymph node in the groin, with dark areas showing through the skin, or in the base of the node.

MELANOMAS

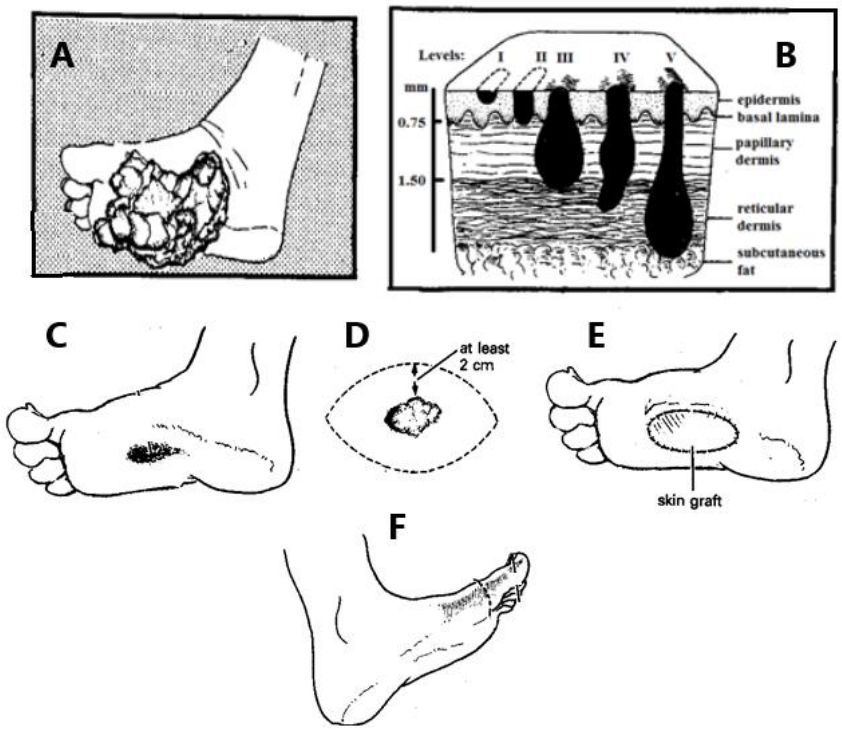


Fig. 34-9 MALIGNANT MELANOMAS.
A, if a melanoma presents as late as this, amputation has no immediate advantage over local excision and grafting but skin-grafting the sole may not be very satisfactory in the long term. B, Clark's levels of invasion: Level I does not penetrate the basement membrane. Level II extends into the papillary dermis. Level III extends to the reticular interface. Level IV invades the reticular dermis. Level V penetrates the subcutaneous fat. Compare this with Breslow's staging level. C, excise a melanoma with a margin of ≥2cm of normal skin round it. D, if it presents sufficiently early, excise it and E, graft the wound. F, amputate a toe for a subungual melanoma.
B, after Grabb WC, Smith JW, Plastic Surgery. A Concise Guide to Clinical Practice. Little Brown, with kind permission. C-E, after Seigler HF in Rob C, Smith R. Operative Surgery. Butterworth 1981 p.221 with kind permission.

Suspect that a white patient has a melanoma if:
(1) any previously existing pigmented mole enlarges, weeps, scabs, bleeds, itches, ulcerates, becomes darker, or produces a dark surrounding halo.
(2) any pigmented lesion keeps growing progressively. Be especially suspicious if it is >1cm, with an irregular border, surface, or pigmentation.
(3) there is a rapidly growing brownish fleshy ulcerated skin tumour, even if it is pale (it may be amelanotic).

(c) Staging malignant melanoma
Prognosis depends on the size of the tumour and depth of infiltration of the excised specimen macroscopically. Breslow's level is more accurate: I ≤0·75mm, II 0·76-1·5mm, III 1·51-2·25mm, IV 2·26-3·0mm, V >3mm. Survival drops drastically from level III.

(d) Excision of malignant melanoma (GRADE 1.3)
If you suspect that a lesion is a melanoma, but have not previously biopsied it, excise it with a margin of at least 1cm of normal tissue all round for each mm of tumour thickness, with preferably more on the proximal end. Remove all the underlying subcutaneous tissue and deep fascia. If the bed that remains is suitable, graft it immediately. Take a split skin graft from the opposite limb, *not the limb bearing the melanoma*.

Prophylactic block dissection of the regional nodes probably does not help. However, you can inject 5ml of blue dye around the primary lesion, and then explore the groin 20mins later, and remove a blue-tinted *sentinel* node. If this is visibly black on section or contains melanoma on histology, a block dissection *is* indicated. Otherwise follow up regularly, so that if the regional nodes enlarge, you can perform a block dissection.

If there is local infiltration, and spread to the regional nodes, make a wide local excision, and a block dissection of the regional nodes (usually inguinal, 17.8). If there is growth in the intervening lymphatics (for example in the neck), excise these in continuity. It is doubtful if this improves survival, but it does remove deposits which may ulcerate.

If there is already widespread dissemination, there is nothing you can do, except provide terminal care (37.1).

If wide local excision is not possible without amputation, as for example under the big toe or a nail, amputate well proximal to the lesion.

(e) Histology. If possible, send the whole specimen for examination. If this is impractical, cut and orientate it for the pathologist to make it possible to ascertain the depth of penetration, and the margins of normal tissue excised in the vertical and horizontal planes; fix the specimen on card and label the card "anterior/posterior", "medial/lateral", "superior/inferior".

34.7 *Leishmaniasis*

The female sandfly is the vector for transmitting the flagellate *protozoa Leishmania* from rodents, particularly gerbils, or domestic animals, especially dogs, to humans and between humans themselves. Different types of *Leishmania* give rise to specific diseases: normally cellular immune responses prevent a second infection, but with HIV disease widespread infestation often occurs. Deforestation has exposed many millions of non-immune people to *Leishmania*, which is now endemic in 88 countries.

There are 4 forms of *leishmaniasis*: cutaneous, mucocutaneous, visceral and post-kala azar dermal types.

They are caused by specific *Leishmania:* 90% of cases of the cutaneous form occur in Afghanistan, Brazil, Iran, Peru, Saudi Arabia and Syria; 90% of the mucocutaneous form occur in Bolivia, Brazil and Peru; 90% of the visceral form occur in Bangladesh, Brazil, India, Nepal and Sudan. Post-kala azar dermal *leishmaniasis* occurs after treatment for the visceral disease.

(a) Cutaneous *leishmaniasis* (Oriental sore)

After an incubation period of days to months from the time of the sandfly bite, an itchy red papule develops on exposed parts of the body, especially the face. This then produces a firm elastic scaly nodule with a shallow, well-defined, punched-out central ulcer having yellow-red granulations at its base. Spontaneous healing occurs after 3-12 months depending on the species of *Leishmania*. If there is HIV disease, the nodules spread extensively on the skin, and often cause visceral involvement.

(b) Mucocutaneous *leishmaniasis*
(Espundia, Chiclero ulcer, uta)

Nodules tend to appear at the mucocutaneous junction of nose and lips; later after months or years, they may spread by lymph or bloodstream to the nasopharynx, palate, uvula, larynx and the airways. This results in destruction of these tissues, with resulting gross facial deformities needing plastic reconstruction (29.18).

(c) Visceral *leishmaniasis* (Kala azar)

Dissemination of *amastigotes* throughout the reticulo-endothelial system results in fever, weight loss and massive splenomegaly. There is often also lymph node enlargement and hepatomegaly, particularly in children where the skin becomes darker ('kala azar' means, in Hindi, the black sickness). Visceral *leishmaniasis* is particularly common in HIV+ve patients.

(d) Tests. Fix a smear or aspirate from a nodule or ulcer, from a lymph node, or from the spleen (after checking the clotting and bleeding times) in methanol, and stain it with Giemsa. You should see many *amastigotes* (Leishman-Donovan bodies) which are rounded 2-3μm bodies inside macrophages.

(e) Treatment
Although nodules heal, they take long and do so with scarring; if there are many, the disfigurement is significant. Single-dose IV liposomal amphoteric in B 3mg/kg followed by oral miltefosine 2·5mg/kg for 7 days gives >95% cure rates. *Antimonials were standard treatment but may cause pancreatitis or cardiac angina*. Drug resistance and toxicity are very common with HIV+ve patients; relapse is frequent after 1 month to 3yrs. Repeated relapses may occur. Ketoconazole and the application of honey are useful for early cutaneous lesions.

34.8 Guinea worm infestation (dracunculiasis, dracontiasis) and *onchocerciasis*

(a) Guinea worm infestation
This occurs when an encysted subcutaneous filarial worm is released when the skin becomes wet or ulcerates; gravid females release larvae which are swallowed by the crustacean cyclops (water fleas 1-2mm in size). When man, cat or dog swallows fresh water from a well infested with these cyclops, the larvae are liberated by digestive juices, migrate through the intestinal wall to areas of loose connective tissue, and finally after c.1yr the females lodge as fertilized worms under the skin of the legs, or more rarely the arms. They are 60-90cm long.

It was endemic in Saudi Arabia, Iraq, Central Asia, Nigeria, and Sudan, but now remains mainly in South Sudan, Chad, Mali and Ethiopia, especially in areas of conflict. Eradication has been effective by treating drinking water with the effective organophosphate larvicide, *temephos* (1ppm), and filtering it with polyester cloth.

When the worm emerges from the skin, it causes intense itching and oedema. Scratching may encourage secondary infection. When the affected part is put in water to soothe the discomfort, the female worm contracts at the base of the ulcer and releases thousands of first-stage larvae, so contaminating the water further.

If a guinea worm presents on the skin, *don't try to dissect it out*, because severe sepsis usually follows. Instead, carefully wind it round a matchstick, and be prepared to take 3wks in doing so, leaving the stick with its coil of worm under a dressing, and pulling out a little more each day. If the worm is broken, milk out the larval fluid. Treat with a topical antiseptic and tetanus toxoid as an adjunct to mechanical removal. If you can extract the worm before it emerges, you will prevent contamination of water resources. Applying topical metronidazole or thiabendazole cream eases the irritation and reduces risk of infection.

If a large rubbery cystic mass develops, usually on the trunk, and typically on the back near the angle of the scapula, distinguish a guinea worm cyst (a low-grade encapsulated abscess) from a lipoma:
(1) A guinea worm cyst often has a small scar on its surface.
(2) On contraction of the muscles, you can see that a lipoma is usually superficial, and a guinea worm cyst deep.
(3) Aspirate the mass with a wide needle. A guinea worm cyst usually contains sterile pus. If necessary, dissect out the mass, taking care not to injure surrounding structures. There is no easy plane of cleavage.

If the cyst becomes infected, it may be impossible to distinguish it from pyomyositis (7.1) or septic arthritis (7.17), especially of the knee: you will have to drain these in the usual way.

ENCYSTED GUINEA WORMS

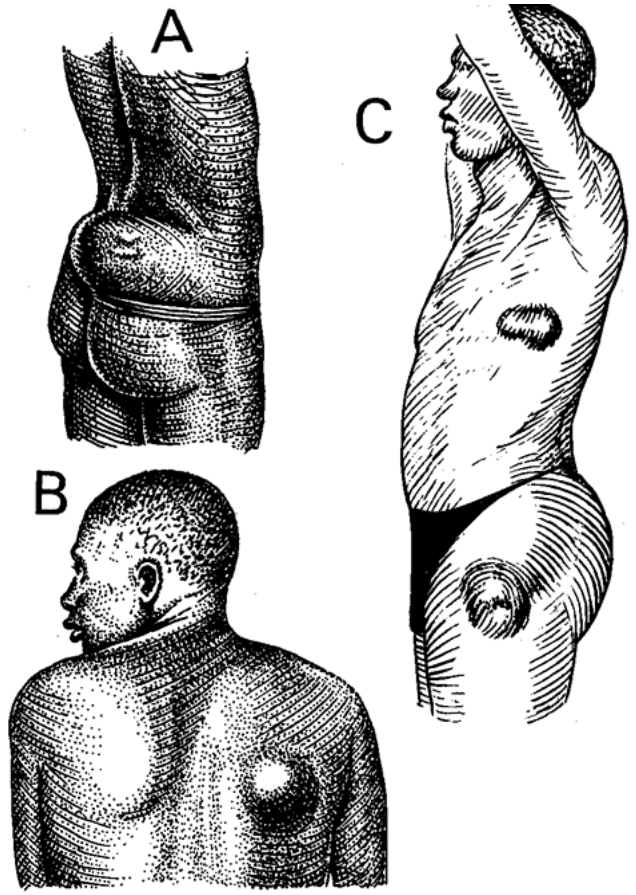


Fig. 34-10 ONCHOCERCAL NODULES AND ENCYSTED GUINEA WORMS.
Both are easy to remove surgically. A,B, encysted guinea worms are commonly found on the trunk, particularly over the inferior angle of the scapula and the crest of the ilium. C, onchocercal nodules are usually found over the iliac crests, trochanters, sacrum, knees, shoulders or head.
Partly after Bowesman C. Surgery and Clinical Pathology in the Tropics, Livingstone 1960 with kind permission.

(b) ***Onchocerciasis*** results from infestation by a filarial nematode, *Onchocerca volvulus*, which comes to live in subcutaneous nodules or free in the skin. The female worm is 35-70cm long and twists itself inside a capsule; the fibrous nodules so resulting may exist for up to 15yrs. The worm itself produces millions of *microfilariae* which invade skin and connective tissue where they are ingested by biting blackflies and develop into infective larvae. Bites in Africa tend to be low on the body from flies breeding by fast-flowing streams; in South America bites are on the head from flies breeding in small hillside streams.

It is endemic in 36 countries, especially in sub-Saharan Africa, but also common in Brazil, Venezuela and Yemen, affecting 18 million individuals, 99% of whom are in Africa.

Most *microfilariae* die within the skin and cause itchy popular dermatitis, which becomes lichenified, atrophied and depigmented; in the eye keratitis and chorio-retinitis (28.7) result. Regional lymphadenopathy is a common consequence. Co-infection with loiasis is common.

Tests. Take at least 2 skin snips and place these for 1-24h in the well of a microtitre plate full of saline; under low-power microscopy, you will then see *microfilariae* migrate out of the skin. If they don't appear, cover the plate with clingfilm and re-examine after 24h.

Treatment. Treat with ivermectin once every 6 months according to weight:

15-25kg	26-44kg	45-64kg	65-84kg
3mg	6mg	9mg	12mg

This treatment may need to continue for up to 10yrs to cover the lifespan of the worm, as the drug kills *microfilariae* but not the adult worm. Add doxycycline 100mg bd for 4wks to eradicate *Wolbachia*, which is usually present as well.

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If a patient from an endemic area has firm onchocercal nodules, 2-3cm diameter on bony prominences, especially over the iliac crests, trochanters, sacrum, knees, shoulders, or head, remove them under local anaesthesia, and examine for an encysted worm.

If an inguinal adenolymphocoele develops (hanging groin, 18-5), you might need to excise it if it becomes very uncomfortable.

If an inguinal hernia develops, repair this bearing in mind the tissues will be weak (18.4).

34.9 Tropical ulcer

These are full-thickness necrotizing bacterial infections of the skin, arising through a puncture wound or dirty laceration. Spreading cellulitis (6.22) with spirochaetes *Treponema vincentii* and fusiform organisms (both are penicillin-sensitive) causes dermal gangrene and skin breakdown, which is enhanced by further infection, trauma or solar exposure. Their characteristic distribution (34-11D) is probably explained by their origin from infected cuts from the sharp grasses of bush paths.

An acutely painful ulcer begins with a raised, thickened, and slightly undermined edge. This ulcer grows rapidly for several weeks. A bloody discharge covers the grey slough on its floor, the skin around it is dark and swollen, and muscle, bone, and tendon occasionally lie exposed in its base. After about a month, the pain, swelling, and discharge improve, and it either heals or becomes chronic, resembling any other longstanding indolent ulcer. Osteomyelitis is rare, but a reactive periostitis may in time raise an ulcer above the surrounding skin. Sometimes its edge is thickened and everted, and resembles a carcinoma.

It is very similar to pyoderma gangrenosum, found in adults on the shins but in children on the head and neck, associated with inflammatory conditions such as Crohn's disease, and which typically recurs in sites of trauma. This responds rapidly to prednisolone 1mg/kg od and then azathioprine 50mg od whilst tapering off the steroids.

TROPICAL ULCERS

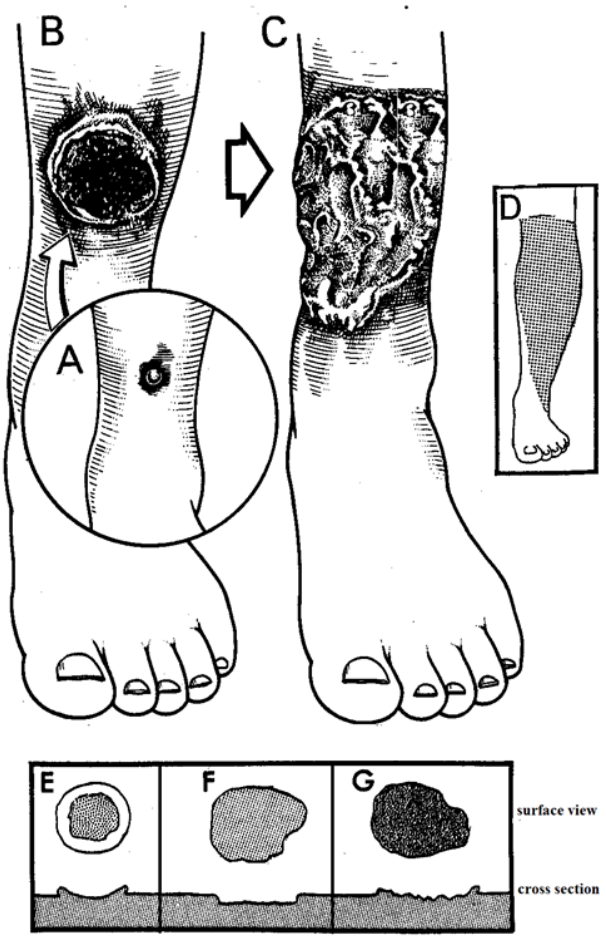


Fig. 34-11 TROPICAL ULCERS.
A, earliest stage of a tropical ulcer: a pustule containing *Treponema vincentii* and fusiform bacilli. (Commonly, however, the early stage is just a small cut.) B, pustule rupturing to form an acute ulcer. C, chronic ulcer showing the exposed tendons. D, characteristic site of tropical ulcers anteriorly on the lower leg, becoming more lateral lower down. E, acute tropical ulcer, with an everted oedematous edge, and a dirty slough covering much of its base. F, chronic ulcer, with an edge which is not raised, and a uniform avascular base. G, note difference with a squamous carcinoma, with its everted edge, and irregular base.

(a) Differential diagnosis

The site of a tropical ulcer is its most important diagnostic feature. Distinguish chronic non-specific ulcers from those with specific cause, *e.g. mycobacteria*, spirochaetes, protozoa, fungi, carcinoma or repeated trauma.

Suggesting tuberculosis: a ragged, shallow ulcer, with bluish overhanging edges. Its base is less vascular and more fibrous.

Suggesting a Buruli ulcer: an otherwise fit child, or young adult, suffering from a huge ulcer with deeply undermined edges, anywhere on the body, not necessarily on the feet and lower legs.

Suggesting leprosy (32.2): a trophic ulcer from pressure necrosis, usually on the anaesthetic sole of the foot.

Suggesting anthrax: a red papule with a ring of vesicles forming a black eschar associated with significant oedema, from contact with infected animals especially carcasses, often with extreme toxicity. Other causes may be cowpox or monkeypox!

Suggesting diphtheria (desert sore): a ragged ulcer with an overhanging edge and a grey-brown base, persisting if there is also HIV disease. There may be cardiac effects and a neuropathy.

Suggesting yaws: a child with heaped hyperkeratotic ulcers on moist surfaces, especially soles and hands.

Suggesting syphilis: an ulcer appearing 6wks to 6 months after a chancre.

Suggesting *leishmaniasis* (34.7): a reddish, sharply defined, indurated ulcer around a sandfly bite.

Suggesting mycetoma (34.11): a painless swelling on the foot with sinuses discharging grains.

Suggesting herpes (5.6): ulceration along one or more dermatomes in an HIV+ve patient.

Suggesting carcinoma (34.5): a heaped-up, irregular, longstanding ulcer with rolled everted edges.

Suggesting varicose ulcer (35.1): a brownish eczematous sloughy ulcer above the medial malleolus associated with varicose veins, deep vein thrombosis, and obesity.

Suggesting sickle cell disease: a chronic ulcer over the lateral malleoli in a sickle cell patient.

CAUTION! The macroscopic differentiation of a chronic tropical ulcer from a specific or malignant ulcer (34.5) can be difficult when there is no extension into the surrounding tissues. Be sure to take a biopsy before you perform any radical surgery.

MOST GRAFTING SHOULD BE DONE AT THE ACUTE OR SUBACUTE STAGE

(b) Treatment for acute tropical ulcers

Debride the ulcer and get it clean with daily soaks, and honey, ghee, banana, pawpaw or zinc-medicated dressings (2.10). When it is clean, usually within 7 days, and if it is >5cm diameter, perform a split skin graft. *Don't use topical antibiotics.*

Smaller ulcers will heal without grafting. If the granulations are abundant, scrape the base of the ulcer with a scalpel. There is no need to anaesthetize it; there are no nerves in granulation tissue, so this does not hurt, provided you avoid the epithelium. Scraping does not improve the 'take', but it does reduce fibrosis under the graft later and so makes it more stable. Control bleeding with hot packs.

If possible, apply the graft as a single sheet, which has been meshed to allow the escape of exudate and blood, or, less satisfactorily, apply it as patches or pinch grafts. Immobilize the affected part, and expose the graft on the 5th day, or earlier if it is smelly.

Then soak off the dressing slowly to avoid removing the graft at the same time. It may need covering for another week. The donor site should have healed in 10 days.

(c) Treatment for chronic tropical ulcers

If an ulcer is deep fibrotic and on the lower leg, measure the ulcer carefully over a period of follow-up (with photographs if possible); apply platelet-rich fibrin (from centrifuging venous blood) as a thin layer weekly with fine forceps, elevate the limb and enforce rest in bed, though encouraging movement in the other limbs! *Take especial care of pressure points, the patient's skin elsewhere, and the condition of the feet, including the toenails.*

If there is spreading secondary infection, use cloxacillin and apply a thick absorbent dressing 2cm thick with a firm but not over-tight bandage, carefully applied to avoid wrinkles and folds. *If the dressing is soaked, it needs to be changed as the discharge will cause further skin erosion.* A vacuum dressing will help remove excess fluid and slough: this can be economically locally made using low-pressure (125mm Hg) suction machines.

Split skin grafts don't take on longstanding fibrotic ulcers. These cause longstanding morbidity, and may become malignant. If the base of the ulcer is suitable, and is not too deeply fibrosed or over bone or tendon, excise and graft it as a single procedure.

Alternatively, apply a tourniquet (3.4), and excise the ulcer. Cut away all avascular scar tissue, until you reach a raw, bleeding surface; if necessary, use an osteotome to remove any dead bone. Apply hypochlorite, or a dry dressing, to the ulcer bed, cover it with gauze, cotton wool, and a bandage, and release the tourniquet. After 5-10 days, when the base of the ulcer is covered with suitable granulation tissue, prepare a myocutaneous flap (34-12).

IPSILATERAL THIGH-TO-LEG FLAP (GRADE 3.2)

Check that the peripheral arterial supply in the distal leg is good. Plan the operation meticulously, measuring the size of the flap and making sure it will cover the ulcer when the knee is flexed. Use a piece of cloth as a template, and cut it the right size, and then mark the skin with indelible ink (34-12A). Infiltrate the flap area with dilute lignocaine/adrenaline solution. Debride the ulcer thoroughly to produce fresh clean granulation tissue.

THIGH-TO-LEG *TENSOR FASCIA LATA* FLAP

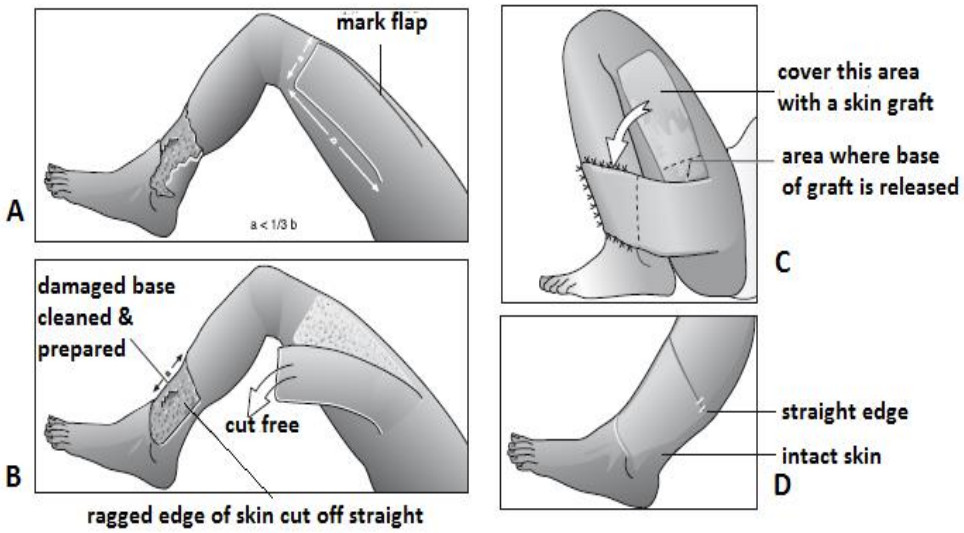


Fig. 34-12 IPSILATERAL THIGH-TO-LEG *TENSOR FASCIA LATA* FLAP.
A, *chronic* distal leg ulcer with flap marked out on the thigh, where a<⅓b. B, during transfer. C, flap sutured prior to immobilization. D, end result after division of flap.
After Badran HA. Plastic Surgery in Kamel R, Lumley J. (eds) Textbook of Tropical Surgery, Westminster 2004 p.722 Fig 163.1.

Raise the flap using the blood supply to the ilio-tibial tract which comes from the lateral femoral circumflex artery. Take the skin attached to the *tensor fascia lata* muscle from the level of the pubic tubercle anteriorly along a line just lateral to the anterior superior iliac spine, and posteriorly along a line down from the greater trochanter. The ilio-tibial tract lifts off the *vastus lateralis* and you can then swing the flap in the direction you want.

With the knee firmly flexed, attach the flap over the ulcer (34-12C) with 3/0 interrupted nylon sutures. Put a split skin graft on the lateral thigh defect. Dress the wounds, and immobilize the knee in plaster.

After 5 days expose the grafted area, and redress the wound, putting on a tight crepe bandage so that the patient cannot extend the knee. After 3wks, divide the flap, allowing enough tissue to be sutured to the recipient site, and suture the proximal portion to its donor site.

CAUTION! Don't perform this operation if there is peripheral ischaemia, or *if the patient has significant arthritis:* the knee may become permanently stiff after 3wks of immobilization. Make sure the ulcer site is free of infection before you attempt such a flap.

You can use a flap from the opposite leg but immobilizing both legs for a cross-leg flap is difficult.

(d) Buruli ulcer occurs in areas near rivers and stagnant water, especially where there has been environmental change, *e.g.* due to mining. It is found in West and Central Africa, India, China, Indonesia, Australia (where koalas and possums carry the disease), Mexico, Peru and Bolivia; aquatic insects may transmit the causative organism, *Mycobacterium ulcerans*, which causes necrosis of skin and deep fascia through the action of cytotoxins.

Commonly a child <15yrs presents with a painless, small, well-demarcated, indurated swelling, attached to the skin, but not to deeper tissues. It is almost always single and on the limbs, and is often near a joint, although the site is variable. There is little pain or tenderness, little or no fever, and the regional lymph nodes are not enlarged. The lesion grows, the skin over it desquamates creating a plaque, becomes pigmented, and then breaks down to form a chronic expanding ulcer with a necrotic base and edges which may be undermined 5-15cm. Secondary infection occurs, and a foul slough forms. Satellite ulcers may appear, but metastatic spread is rare. Some ulcers remain unchanged for weeks; others cover much of a limb, or the trunk, in a few weeks. Untreated, they result in scarring and severe contractures.

DIAGNOSIS is suggested by the appearance of the swelling and the ulcer, the absence of lymph node enlargement, and the failure to respond to tropical ulcer therapy.

SPECIAL TESTS. Look for AAFB in the ulcer base; send material for culture. *M. ulcerans* grows on media used for *M. tuberculosis*, but only at 33ºC. Mycolactone produced can be detected by PCR.

TREATMENT. Early on, use rifampicin and streptomycin or, better, clarithromycin for 4-8wks; healing continues after completing the course of treatment. If the lesion is ulcerated, control secondary pyogenic infection with antibiotics, and irrigate with warm water. Excise all diseased tissue, and when granulations are healthy (c.3-4wks), re-examine the wound for residual TB, and if clear, cover the wound with a skin graft.

(e) Desert sore (diphtheritic ulcer) occurs in dry desert climates, starting as a painful vesicle leaving a raw and tender ulcer with a grey-brown base. Later this becomes punched out with an undermined edge. The organism, *Corynebacterium diphtheriae*, produces an exotoxin which can give rise to myocarditis and peripheral neuropathy. Use benzylpenicillin and immunize contacts with diphtheria toxoid.

(f) Yaws (framboesia) occurs amongst poor children living in unsanitary and overcrowded conditions in warm, humid tropical climates. It is caused by *Treponema pertinue*, which is transmitted by direct contact through clothing and insects. A painless swelling which occasionally ulcerates, the 'mother yaw', starts at the site of the contact with concurrent regional lymphadenopathy. This heals spontaneously after 3-6 months when the infection spreads to moist areas of the skin, *e.g.* soles, palms and the back of the knees. These usually ulcerate, become infected, and may destroy joints and bone. Use a single oral dose of azithromycin 30mg/kg.

(g) Herpetic ulcers occur characteristically in HIV+ve patients following the acute episode of the herpes zoster reaction. One or more dermatomes may be involved. Acyclovir 800mg x5 od for 1wk is useful early in the eruption. Post-herpetic neuralgia may be severe.

(h) Hidradenitis suppurativa (Verneuil's disease) or **pyoderma fistulans sinifica** (fox-den disease) may have a genetic predisposition. These result in localized skin sepsis arising from apocrine glands (so don't develop in children), especially in the axillae, groins, perineum, back of the neck and under the breasts. The result is quite marked skin thickening, fistulae and multiple skin bridges, but the disease does not extend below the fascia. *Be careful to distinguish this from necrotizing fasciitis* (6.23). You will achieve a lasting cure only by excising the whole affected area and leaving the wound to granulate.

34.10 Kaposi sarcoma

Kaposi sarcoma (KS) starts as a proliferation of lymphatic endothelial cells which transform into a sarcoma; the stimulus is infection with the herpes virus type 8, transmitted by saliva, promoted by immunosuppression. The HIV tat protein also promotes KS.

(a) Presentation is in 4 main types (in decreasing order of frequency):
(1) By far the most common, HIV-related KS in men and women, with symmetric lymphadenopathy, oral purplish patches or plaques, pulmonary infiltration and skin nodules (though these are often absent). The nodules don't occur in areas subject to sustained pressure (*e.g.* the soles of the feet or under dental prostheses). Ultimately lesions lead to woody hard oedema and ulceration.
(2) Endemic (HIV-ve) African KS usually of younger men, with indolent nodules on the limbs preceded by hot oedema, ultimately resulting in widespread infiltrating and visceral involvement.
(3) Iatrogenic KS as a result of immunosuppressive drugs for cancer or connective tissue disease, where if the drugs are withdrawn, KS regresses, often completely.
(4) Classical KS of elderly men usually of Arabic, Jewish or Southern European descent, with indolent nodules on the limbs that may regress spontaneously.

(b) Diagnosis
Plaques and nodules are so typical that skin biopsy is rarely necessary in the presence of HIV disease.
N.B. Biopsy of oedematous skin may produce a non-healing wound.
Lymph node excision biopsy may be useful (17.1). All patients suspected of KS must have an HIV test.

(c) Treatment
Chemotherapy (doxorubicin, vincristine, or bleomycin) gives a dramatic response, but there is early relapse in HIV+ve patients unless they also have ARV therapy (5.8). Radiotherapy is useful for isolated limb lesions, but supplement it with chemotherapy. Early KS may not warrant treatment, which may cause serious side-effects; in HIV+ve patients, co-existent pulmonary disease is common: you usually have to treat this first.

KAPOSI SARCOMA

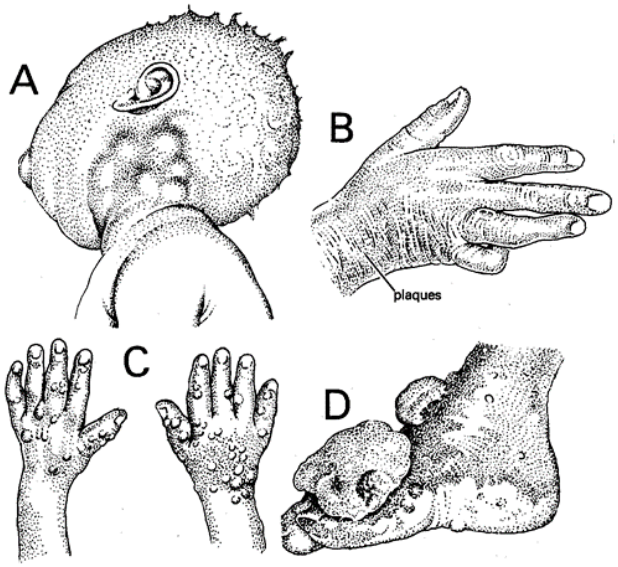


Fig. 34-13 KAPOSI SARCOMA (KS).
A, lymphadenopathy in children. B, infiltrating type. C, nodules (more often seen on the leg). D, large cauliflower-like lesions.

Amputation (35.3) can relieve much distress if a heavily affected limb is ulcerated, septic and useless; *don't do this, though, if oedema extends up to the groin. Beware of bleeding* (especially if there is thrombocytopenia): use a tourniquet.

34.11 Mycetoma (Madura foot)

Mycetoma is a chronic progressive granulomatous inflammation caused by implantation of filamentous fungus-like bacteria (*streptomyces*, *actinomyces*, *nocardia* etc), or the true fungi (*eumycetes*, particularly *madurella* and *leptosphaeria*, *exophiala*, *pyrenochaeta*, *scedosporium*), through cracks in the skin caused by sharp objects, especially thorns, splinters or stones. Typically it occurs on the foot, but may affect the hand, particularly in those working in the fields in arid zones with short rainy seasons especially in latitudes between 15ºS and 30ºN.

It begins slowly to form a circumscribed, rubbery or cystic, *painless* lobulated mass. If it is on the sole, pressure flattens it into a disc. Sinuses appear, and occasionally discharge granules. As one sinus heals, more appear, and become secondarily infected, but this secondary infection does not extend deeply. By the time that 5yrs have elapsed, the whole foot is swollen, and covered with open sinuses and scars. Progression of the disease is faster with *actinomyces* than *eumyces.*

Differentiate this from botryomycosis (6.4), which also produces 'sulphur granules' but which arises from staphylococcal infection.

MYCETOMA

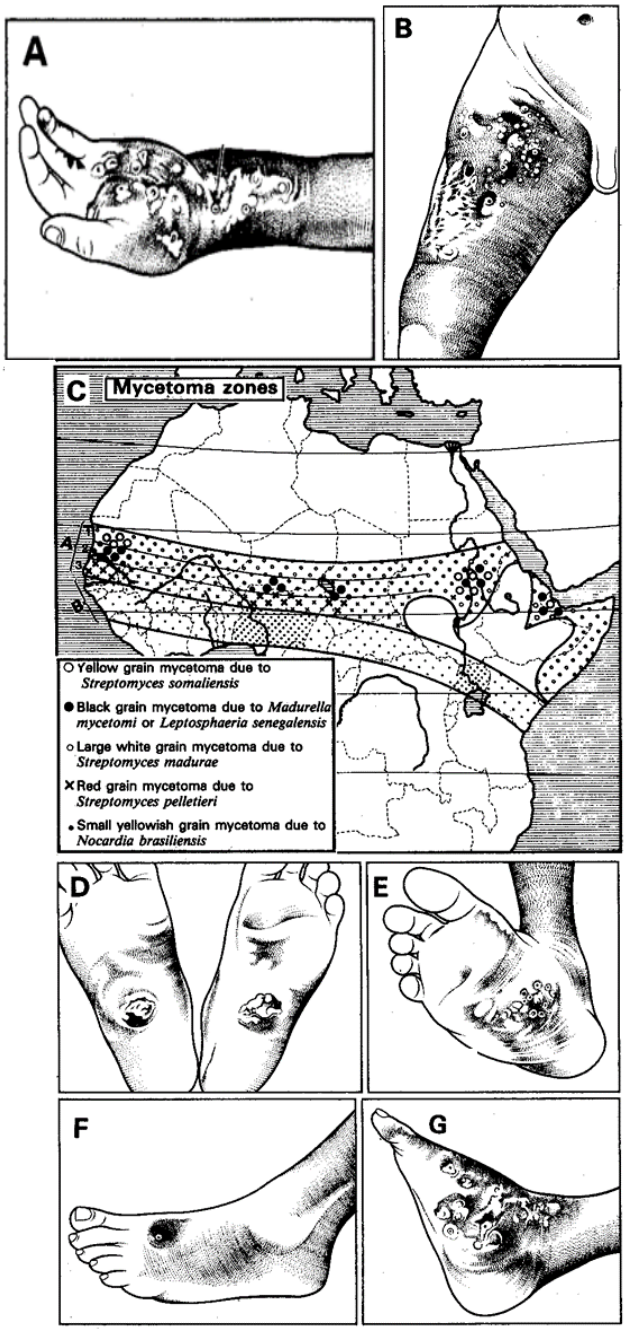


Fig. 34-14 MYCETOMA.
A, mycetoma of the hand, spreading through the carpal tunnel into the forearm (unusual). B, advanced mycetoma of the thigh 20yrs after infection had begun in the foot. C, endemic and sporadic mycetoma zones in Africa. D, early black grain mycetomas of the soles of both feet, showing flattened disc-shaped swellings. This is the typical early lesion but simultaneous involvement of both feet is rare. E, more advanced lesion. F, mycetoma of the dorsum of the foot. This may be part of a dumb-bell lesion extending from the sole between the metatarsals. G, diffuse mycetoma of 10yrs' duration; still painless and the sufferer was still working.
After Crockett DJ. Mycetoma. Tropical Doctor 1973;3(1):28-33 with kind permission.

The primary site of infection is usually in the subcutaneous fat; it may spread beyond the plantar or palmar fascia, which form a natural barrier. It may then spread in the deep fat between the tendons, along the lumbrical canals, and even through the carpal tunnel, up into the forearm.

The bone may be invaded relatively early, still without causing pain, and is rapidly destroyed. *Mycetomas never regress spontaneously*.

Regional lymph nodes are usually not enlarged, but may be affected by secondary sepsis and occasionally by lymphatic spread of mycetoma.

(a) Diagnosis. Try to find the granules, because without them, all a pathologist can say is that there is a granulomatous infection with multiple micro-abscesses. The colour and size of the grains can give a clue to the cause: *Actinomyces pelletieri* produces small red grains, *Streptomyces somaliensis* medium-sized yellow, *Actinomyces madurae* large white, *Nocardia brasiliensis* small yellow, *Madurella mycetomi* and other *eumyces* large brown-black. If you cannot see the grains with the naked eye, get an aspiration specimen for cytology. You should see filaments with x40 magnification.

(b) Radiographs. Once the periosteum is breached, the tarsal and metatarsal bones are rapidly destroyed. New bone in the walls of abscesses forms buttresses projecting outwards at angles to the shaft of a long bone. The centre of an infected bone has a honeycomb appearance, and a good film shows tiny cystic areas of bone destruction, each the site of a micro-abscess. Differentiate this from an osteosarcoma or tuberculosis.

(c) Ultrasound. This shows typical thick-walled cavities with no acoustic enhancement, with the grains giving numerous bright hyperreflective echoes, especially with *eumyces.* These are less distinct with *actinomyces*.

(d) Differential diagnosis
Actinomycosis produces yellow granules but occurs very rarely on the foot.
Cutaneous B-cell lymphoma may look identical, and equally chronic.

(e) Treatment
For ***actinomyces***, use trimethoprim. Rifampicin can replace streptomycin and sulfadoxine-pyrimethamine (Fansidar), and ciprofloxacin can replace cotrimoxazole in resistant cases.
For ***eumyces***, try ketoconazole 400mg od, for 1yr, but the chance of cure is much lower.
For ***nocardia***, use cotrimoxazole with amikacin for 2wks. Otherwise, try griseofulvin 500mg od.
Curette and drain any low-grade abscesses that form.

If a lesion is localized, and is confined to the soft tissues, excise it and repair the defect with a split skin graft. *Take great care not to rupture the capsule of eumyces* as you will otherwise transfer the fungus to adjacent areas, and recurrence will be inevitable.

Excise a margin of healthy tissue with *actinomyces* as its border is ill-defined. *Always* use a tourniquet.to produce a bloodless field. Flood the operative field at the end of the operation with iodine to minimize risks of contamination.

If bone is involved, or will be, Syme's amputation (35.7) is appropriate provided you can clear the disease adequately.

If there is a lesion of the hindfoot with severe bone and joint destruction, perform a below-knee amputation (35.6).

If there is a lesion of the hindfoot with minor bone involvement, and without severe destruction of its joints, you will probably have to amputate, but wait until the foot becomes a real nuisance. Make sure there is no response to medical treatment.
Always combine surgery with long-term medical treatment for at least 6 months.

CAUTION!
(1) Mycetoma is painless, so *don't amputate a limb until the patient is quite convinced that the limb is of no use*.
(2) Follow the patient up carefully, and make sure he reports any involvement of the inguinal nodes. When he does, this is an indication for urgent amputation and block dissection of the nodes (17.8).

34.12 Elephantiasis

If there is gross generalized swelling of the leg, arm, or scrotum, or if a woman has a similar swelling of her breast or vulva, the condition is known colloquially as elephantiasis. Usually, this is due to longstanding lymphatic obstruction. Occasionally, it is due to venous obstruction, but this is seldom gross enough to need surgery. Distinguishing between lymphatic and venous obstruction can be difficult. Oedema due to lymphatic obstruction becomes firm quite rapidly, but early cases may show pitting. Oedema due to venous obstruction becomes solid late, and eventually reaches a stage where it fails to pit.

In most areas, the causes of lymphatic obstruction (lymphoedema), in decreasing order of frequency, are:
(1) Tuberculosis
(2) Repeated lymphangitis due to lymphatic obstruction, usually from *streptococci*, filariasis or podoconiosis (34.13)
(3) Malignant glands in the groin, or less often the axilla
(4) Kaposi sarcoma
(5) Block dissection of the glands, usually for carcinoma
(6) Congenital lymphatic hypoplasia (Milroy's disease).
Other causes include:
(7) Chronic fungal infections
(8) *Onchocerciasis* (34.8)
(9) Lymphogranuloma venereum.

Filariasis due to infection in tropical and subtropical regions with *Wuchereria bancrofti* and less often to *Brugia malayi* or *B. timori* is restricted by the prevalence of the insect vectors but its geographical range spans a population of c.1 billion people in 80 countries, mainly in remote rural or poor peri-urban areas. *B. malayi* and *B. timori* give rise to lymphoedema below the knees; *W. bancrofti* to the whole leg, arm, breast, scrotum, or vulva.

Elephantiasis due to advanced podoconiosis (whether it has reached the 'mossy foot' stage or not) responds fairly well to surgery. Elephantiasis due to filariasis is difficult to treat surgically (34.14).

(a) Differential diagnosis
Suggesting venous oedema: rapid onset, pitting of the skin with ulceration, varicose veins, medical causes (*e.g.* heart failure, hypoproteinaemia, nephritis, cirrhosis).

Suggesting filariasis (34.14): scrotal involvement, oedema which starts at the most dependent part for each site and moves upwards: below the malleoli for the leg, the fundus for the scrotum, the foreskin for the penis, and the dorsum of the hand for the arm. Microfilariae found in blood films.

Suggesting podoconiosis (34.13): a barefooted patient from a podoconiosis area; worse on one leg than the other; below-knee swellings most marked distally. Symptoms are the first evidence of disease (in filariasis they are the last), and include burning of the lower legs at night, with persistent itching of the 1st and 2nd toe clefts, and plantar oedema of the forefoot. No microfilariae in the blood, and a chronic warty thickening of the lower legs ('mossy foot').

Suggesting chronic non-specific lymphangitis: some source for it, such as a tropical ulcer (if the swelling is in the lower leg). Acute recurrent attacks of lymphangitis. Enlargement of the nodes draining the swollen area only: these may be large and firm, or small and fibrotic. Lines of hyperpigmentation on the skin indicating previous lymphangitis. A lymph node biopsy showing fibrosis and non-specific inflammatory changes.

Suggesting tuberculosis (17.4): chronic enlargement of many superficial nodes (inguinal, axillary, and cervical); a history of prolonged illness in the past, with fever and enlarged nodes, some of which discharged for long periods; multiple sinuses, or the scars that follow their healing, especially over the lower end of the vertical chain. Involvement of an entire leg from toes to groin, or an entire arm or a woman's breast. A +ve lymph node biopsy confirms the diagnosis; if tuberculosis is no longer active, only non-specific fibrosis may be seen.

Suggesting lymphogranuloma: also produces sinuses, but these are usually confined to the superior group of nodes, over the medial part of the inguinal ligament.

Suggesting malignant disease: firm mass in the groin or axilla; typical purplish raised lesions of Kaposi sarcoma.

N.B. The site involved indicates the probable cause:

Breast and arm, or vulva	tuberculosis, filariasis
Scrotum or vulva	filariasis
Lower leg	filariasis, podoconiosis

(b) Treatment
If you are in doubt, try the appropriate specific treatment for tuberculosis or filariasis. Lymphoedema due to tuberculosis nearly always settles with anti-TB therapy, unless it is diagnosed very late. Antibiotics are unlikely to influence chronic non-specific inflammation. Check the HIV status.

34.13 Podoconiosis

Podoconiosis ('dust in the feet': non-filarial endemic elephantiasis) is found in fertile highland volcanic regions in Uganda, Tanzania, Cameroon and Sao Tome, and Central and South America and Indonesia due to alkaline red clay soils whose silicates penetrate the skin and are taken into the lymphatics. It only involves the legs, presenting as bilateral asymmetrical swelling of the feet and lower legs. This causes the lymphatics to fibrose and obstruct, and the femoral nodes to enlarge.

This in turn makes the legs and feet swell, and progress through stages which are described as 'water bag', 'rubbery', and 'wooden' (34-15). Finally, the leg becomes hyperkeratotic, 'mossy', and nodular. Lymph may ooze through the skin, which may be secondarily infected by fungi or bacteria. The disease may progress steadily, or there may be a succession of acute episodes which resolve incompletely. Villagers in endemic areas are often able to recognize the early stages.

'Mossy foot' can also describe other diseases causing multiple excrescences on the feet, notably *chromoblastomycosis* (34.5).

Elevation, elastic stockings, and long leather boots help in the earlier stages, but once the 'wooden' stage has developed, the only treatment is surgical. If you see a patient early, persuade him to wear boots or shoes which will minimize further progression. The main preventive measure is wearing fully protective shoes rather than sandals or open shoes from childhood.

Podoconiosis is disfiguring, and may result in chronic disability, so treatment is important. Unfortunately, on presentation, the lymphatics are often incurably blocked, so that medical treatment is ineffective.

Surgically, you can:
(1) Compress the leg of a 'soft' case by intermittent compression.
(2) Excise individual nodules over the toes.

(a) Early diagnosis. After a long day's work in the fields, or a long walk, one of the feet becomes swollen, and feels tense. The lymph nodes are enlarged and firm.

PODOCONIOSIS

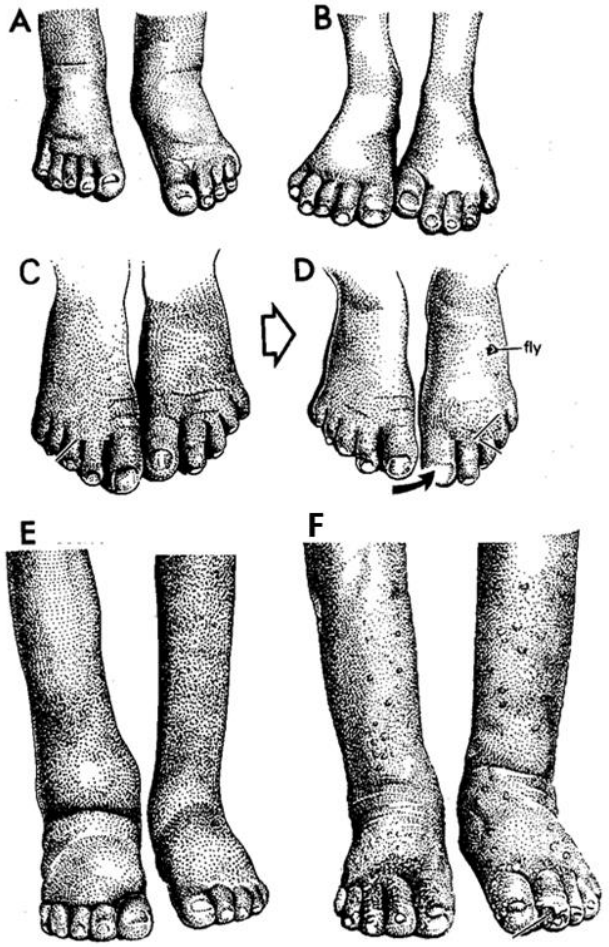


Fig. 34-15 DIAGNOSING PODOCONIOSIS.
A, 'block toes'. Early oedema of the left forefoot affecting the plantar aspect of the metatarsal pad, as well as the toes, which appear rigid, as if they were wooden and nailed on to the forefoot. They may be lifted off the floor by plantar oedema, and lack the usual curve of normal toes. B, 'knocking' big toes on walking, due to splaying of the forefeet as the result of deep oedema at the level of the metatarsal heads. C,D, increased skin markings, which become more evident if the toes are compressed. Flies, attracted by exuded lymph to an otherwise clean foot, are characteristic. E, wet 'waterbag' foot, which is readily reduced by compression or elevation; its skin is soft, and you can pinch it off the bone. F, dry 'wooden' type, which cannot be reduced by compression or elevation; it is hyperkeratotic, and often nodulated. *After Price EW, Pre-elephantiasic stage of endemic nonfilarial elephantiasis of lower legs: 'podoconiosis'. Tropical Doctor 1984; 14(3):115-9 with kind permission.*

Try to recognize the following early stages:
(1) 'Burning leg' is a burning sensation in the lower leg, from in front of the medial malleolus to behind the medial condyle of the knee, sometimes extending upwards into the thigh. The femoral nodes may be tender. Pain is usually worst at night, and is relieved by uncovering the leg. Each episode usually affects the same leg, and the second leg does not usually become involved until the first one shows clear signs of disease. Although the burning area of the leg may be tender, few patients seek help at this stage.
(2) 'Itchy foot' is a persistent localized pruritis, usually on the dorsum at the base of the first or second toe clefts, or below the middle malleolus. Thickening of the skin (pachydermia), from constant scratching, is a common presentation.

When the toes start swelling, the itchy area precedes the upper level of the swelling, and indicates progression of the disease.
(3) 'Block toes' (34-15A) lack their normal curves, and look wooden and rigid, as if they were nailed on the forefoot. 'Splayed forefoot' is a widening of the forefoot, and separation of the toes, which gives the foot a spatula-like appearance, on one or both sides (34-15B). It is due to deep oedema between the metatarsal heads. The skin is unusually resistant to being lifted by your fingers.

TREATING PODOCONIOSIS

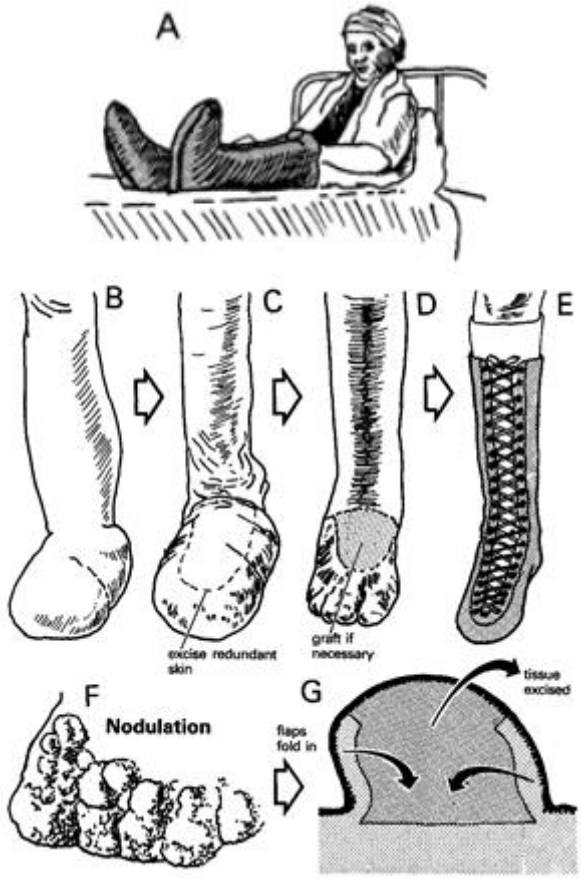


Fig. 34-16 TREATING PODOCONIOSIS.
A, intermittent compression in use (the foot is raised during treatment). B, 'waterbag' foot before decompression. C, same foot after initial reduction of swelling of the lower leg. D, removal of excess skin nodules over the toes. E, leg protected in a boot. F, nodulation. G, how to excise a nodule.
After Price EW, Pre-elephantiasic stage of endemic nonfilarial elephantiasis of lower legs: 'podoconiosis'. Tropical Doctor 1984; 14(3):115-9 with kind permission.

Plantar oedema is asymmetrical (unlike cardiac or renal oedema). Press with your thumb on the sole over the head of the first metatarsal. You may see mild lymphatic oozing, tiny blebs of lymph, or an unusual number of flies attracted to it.

The forefoot shows an excessive deposit of keratin on the dorsum at the base of the first or second toe cleft. The clefts themselves usually remain normal, even in advanced disease with increased skin markings at the base of the first toe cleft and running longitudinally rather than laterally (as is normal). Compressing the toe (34-15D) shows the marks more clearly.

(b) Early treatment
(1) Raise the foot of the bed to the height which relieves the discomfort; a hammock is suitable.
(2) Apply ankle-length elastic socks before rising in the morning; or apply wide (10cm) one-way stretch elastic bandages; crepe bandages are inadequate.
(3) Protect the skin of the feet from the soil, preferably in shoes. Treat any other conditions present.
(4) Use pH-neutral soap.
(5) Start to reduce the size of the swellings with intermittent compression (34-16A-C).
(6) Advise the wearing of stockings and boots (34-16E).

(c) Excision of foot nodules. Excise these for aesthetic reasons, or to make wearing shoes easier (34-16D). They have no sensory nerves, so you can remove them without anaesthesia.

CAUTION! Don't try to remove the femoral nodes. *Don't try to remove redundant tissue unless rigorous barriers are in place to prevent contact with the soil.* Recurrent swelling is more painful and debilitating than the original disease.

N.B. The difference in treatment between podoconiosis and filariasis is that the first is caused by contents of the soil, which are hard to avoid without moving home and work. The second is caused by a vector, mosquitoes, which can, at least in theory, be avoided.

Although the long-term consequence is blockage of the lymphatics, surgery has therefore more to offer in filariasis than in podoconiosis.

34.14 Filariasis

Filariasis is a parasitic infection which causes considerable morbidity to c.120 million people in 80 countries of the tropics and subtropics. *Wuchereria bancrofti* is the cause in >90% of cases, but *Brugia malayi* or *B. timori* occur in Asia. Larvae ingested by *Culex*, *Anopheles*, *Aedes* or *Mansonia* mosquitoes are deposited on the skin of humans, and microfilariae then migrate to the lymphatics *via* the insect bite.

(a) Symptoms start within a few weeks to months of infection, as fever, lymphangitis, tender lymphadenopathy, erythema, and oedema. The inguinal, epitrochlear, and axillary nodes are commonly involved. Attacks may be repeated every few months. There may also be secondary bacterial infection.

In males, the spermatic cord, epididymis and testes are often involved, resulting in painful recurrent attacks of epididymo-orchitis (27.3), which may be followed by suppuration in the scrotum.

Synovitis, arthritis and asthmatic-type attacks also occur.

(b) Chronic effects are the result of lymphatic obstruction, commonly in the retroperitoneum. This can produce:
(1) Lymphoedema, which may progress to gross hypertrophy of the subcutaneous tissues (elephantiasis: 34.12) affecting the whole leg and scrotum (*W. bancrofti*) or below the knee (*B. malayi* or *timori*), or the arm, breast, or abdominal wall (less common). No known treatment will reverse these changes, although doxycylene may arrest the progression by treating co-existent *Wolbachia.*
(2) Hydrocoele (27.24), which is very common in areas of filariasis, usually with chronic epididymo-orchitis.
(3) Vulval lymphoedema, which is very uncomfortable and embarrassing, and may become enormous. This causes obvious sexual dysfunction, extreme mental anguish, and may result in urinary retention, and severe difficulty in walking. Surgery is complicated and needs an expert.
(4) Varicocoele (27.37).
(5) Lymphocoele of the spermatic cord, which may be diffuse, resembling a varicocoele (34-17E), or encysted (34-17F). Beware: *it may exist with an irreducible inguinal hernia* (34-17G)!
(6) Lymphatic varix (hygroma). This is a soft cystic lymph-filled swelling in the axilla, neck, or groin.
(7) Chyluria (27.37), due to rupture of dilated lymphatics into the urinary tract.
(8) Chylous ascites, due to rupture of dilated lymphatics into the peritoneal cavity.

(c) Special tests
(1) The microfilariae of *W. bancrofti* and *B. malayi* are usually present in Giemsa-stained thick blood films taken between 10pm and 2am.
(2) Puncture an enlarged node, or lymphatic varix, with a needle, and look for microfilariae in the small volume of fluid you aspirate.
(3) Fluid aspirated from an infested hydrocoele is amber-coloured and has vacuolated mesothelial cells, fibrin, old blood clots, cholesterol crystals and 'calcium dust': these findings associated with epididymitis are highly suggestive of filariasis in an endemic area. You can test this fluid for filaria by ELISA or PCR tests, if they are available. Microscopic examination of the fluid rarely reveals the filaria.

(d) Ultrasound of dilated lymphatics can show viable adult worms 'dancing'. The hydrocoele fluid shows a mottled echogenicity. Measure skin thickness by ultrasound at the malleoli to monitor progression of the disease.

(e) Treatment
Use doxycycline 100mg bd for 6wks, which also treats symbiotic *Wolbachia.* Add a single-dose albendazole 400mg, and then a single dose of ivermectin (at dose according to weight, 34.8) after 4wks.
(Although both albendazole and ivermectin have limited effects on adult worms, doxycycline, by eliminating *Wolbachia*, will remove the microfilariae.)

N.B. Diethylcarbamazine is no longer recommended especially in areas endemic for onchocerciasis or loiasis, because of the potentially fatal reactions. *Likewise the provocation test* (for sampling blood) *is not advised.*

SOME FILARIAL LESIONS

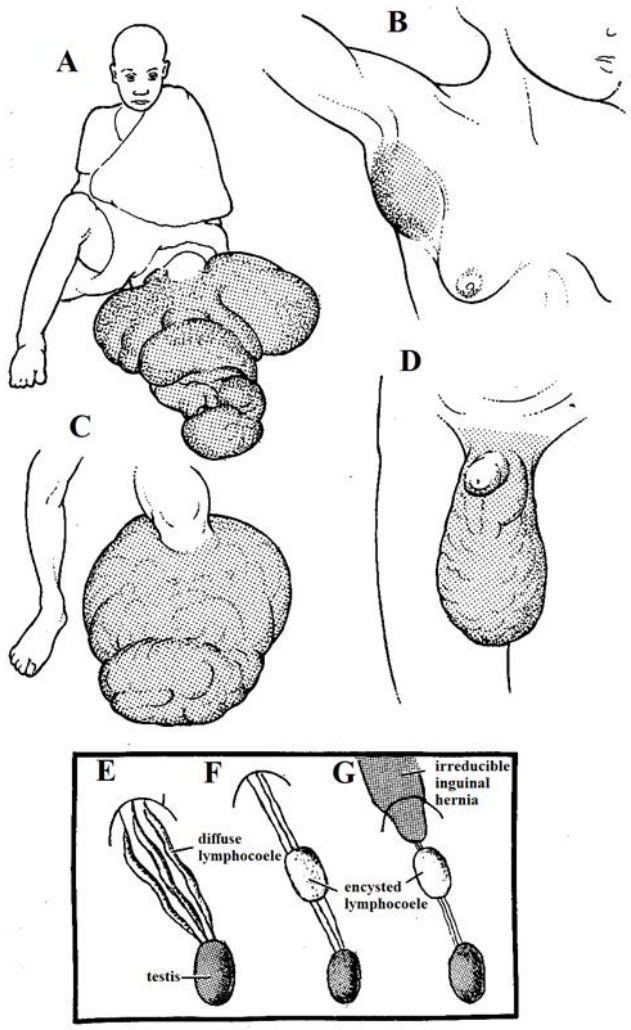


Fig. 34-17 SOME FILARIAL LESIONS.
A, extensive filarial involvement of the leg. After the operation she could walk without support. B, East African woman with an axillary swelling; needle puncture showed that this was a lymphatic varix. C, this filarial mass required amputation; after the operation the patient weighed only half as much as he did before. D, elephantiasis of the scrotum with involvement of the groin nodes, but without involvement of the penis. E, lymphocoele of the cord. F, encysted lymphocoele. G, strangulated hernia with an encysted lymphocoele. *After Mahadevan R, Surgical aspects of filariasis. Tropical Doctor 1978;8(1):28-35 with kind permission.*

Reduce lymphoedema by prolonged firm bandaging; then prevent further swelling by supporting the tissues permanently. Intermittent positive pressure methods, as with podoconiosis (34.13), reduce the oedema very effectively. A patient previously unable to walk may thus be able to do so.

Alternatively, insist on bed rest and bandage the leg with crepe bandages from the foot upwards, using sponge rubber to protect the tissues from too tight bandaging. Remove the bandages every day, and replace them a little tighter. When you have reduced the swelling, fit a graduated compression stocking, which will be useful despite the discomfort in hot, moist climates.

If the disease is advanced, mobilize the oedema fluid by initial elastic compression, and then consider surgery; this is not easy. The operation is a 2-stage process; excise half the circumference of the swollen tissues at a time. This ensures that skin flaps of adequate thickness retain a blood supply.

The deep fascia must be included in the excision to allow drainage through the deep muscle compartments of the leg. Afterwards continue with the use of elastic compression stockings.

N.B. The Charles operation, where only the oedematous subcutaneous tissue is removed, and the fascia covered with skin graft, *results in inevitable worsening of lymphoedema distally, and is not recommended.*

34.15 Sarcomas

These include rhabdomyosarcomas, fibrosarcomas, liposarcomas, synovial sarcomas, dermatofibrosarcomas, histiocytomas, neurofibrosarcomas, epithelioid sarcomas and angiosarcomas. They all arise from mesenchyme, are commonest from the 2nd to the 4th decades, and vary considerably in malignancy. They spread by local infiltration, and lymphatic spread is usually late but may be present in up to 10%. In less differentiated tumours, blood dissemination may occur early, especially to the lung.

Treatment is mainly surgical. The results of *radical* local excision are at least as good as very radical surgery involving amputation. Sarcomas are relatively radioresistant. Chemotherapy is an expensive supplement to surgery, and is not nearly so effective as with lymphoma or nephroblastoma.

Distinguish these tumours from cutaneous metastases especially from breast, lung, prostate, pancreas, thyroid or kidney cancers.

Make sure that if you take a biopsy, you can include your incision in subsequent surgery to remove the tumour, so as to remove the biopsy track. Many of these sarcomas need wide excision; this may however only be possible by amputation. *You must never 'shell out' a sarcoma*: there is no proper capsule, and recurrence will be inevitable. If you are going to operate, make sure you can excise the tumour with a margin of normal tissue.

N.B. Don't try to excise such a tumour unless you know the full extent of its spread.

(a) Rhabdomyosarcoma

This is the commonest soft tissue sarcoma in those <15yrs. It occurs more often in males than in females, usually at 5-25yrs, in the trunk and limbs, head and neck. (It also occurs in the orbit, bladder, and testis.)

Rhabdomyosarcomas probably arise from embryonic mesenchymal tissue and are fairly malignant: they spread locally quite rapidly.

(b) Fibrosarcoma

This usually arises from the muscle sheath or periosteum of the thigh, lower leg, or back. The patient, who is usually 30-50yrs, presents with a firm to hard mass which is usually painless in its early stages. Fibrosarcomas are moderately malignant, and spread by local infiltration.

A CHONDROMA AND A SARCOMA

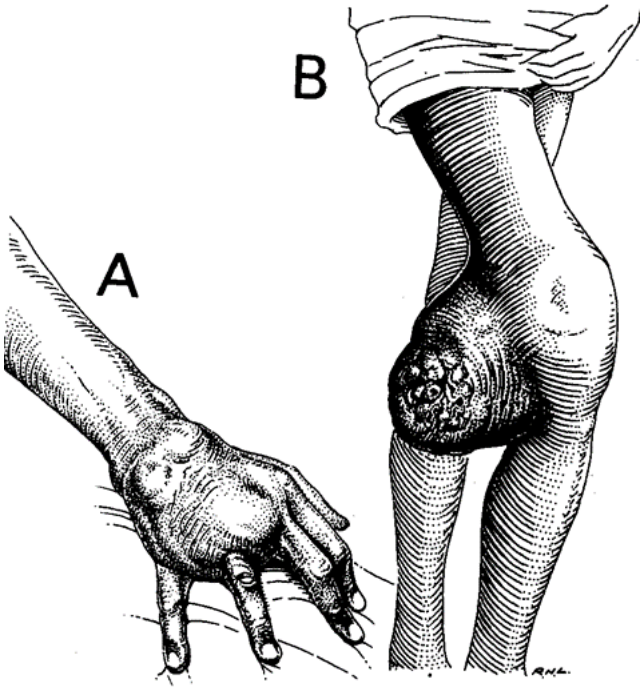


Fig. 34-18 A CHONDROMA AND A SARCOMA.
A, chondroma of the 4th metacarpal. B, advanced sarcoma of the lower end of the right femur.
After Bowesman C, Surgery and Clinical Pathology in the Tropics, Livingstone, 1960 with kind permission.

(c) Liposarcoma

Typically, this occurs in the soft tissues of the thigh, retroperitoneum, or mesentery, in patients of 30yrs or older. There are basically 4 histological types: well differentiated (hard to distinguish from a lipoma), mixed (commonest), round cell (most malignant), and pleomorphic. Low-grade varieties (mixed and well differentiated) tend to recur locally; round cell ones (rare) metastasize early.

A lipoma (34.3) is probably in fact malignant if:
(1) it forms a steadily growing mass in the thigh or abdomen, or recurs after excision,
(2) it has reddish necrotic areas on its cut surface, with cysts instead of plain fatty tissue,
(3) it is difficult to shell out of the surrounding tissues.

In the thigh, distinguish a liposarcoma from pyomyositis by aspiration of pus in the latter.

(d) Synovial sarcoma

This presents as a solid swelling like a ganglion; because of its position next to tendons, excision is usually inadequate and recurrence common. Make sure you use a tourniquet when removing these tumours and use a meticulous technique.

(e) Dermatofibrosarcoma 'protuberans'

This starts as an intradermal plaque and extends slowly over many years into surrounding tissues, especially in patients of 30-50yrs. Classically it has an hourglass shape, with one nodule pressing inwards and one outwards, hence 'protuberans'. Excision results in recurrence in over 50% of cases, so make sure there is a wide (3-5cm) margin of normal tissue, especially in surgery for the second time.

(f) Malignant fibrous histiocytoma

This usually occurs on the extremities arising in subcutaneous tissue or in the fascia, where the prognosis is worse with c.30% having metastatic disease. It probably arises from fibroblasts. Wide excision with 3cm margins including adjacent fascia and muscle is necessary.

(g) Neurofibrosarcoma (Malignant schwannoma)

This develops from neural sheath tissue often in longstanding neurofibromas in Von Recklinghausen's disease (neurofibromatosis type 1), where the chance of malignant transformation is 15%.

34.16 Pressure sores (Decubitus ulcers)

It takes <1h to produce ischaemic changes in the skin from a pressure point; this is usually owing to a bony prominence bearing the patient's weight, but it might also be an area of skin compressed by a tight plaster. The 1st grade is redness (difficult to see on pigmented skin); then there is bluish discolouration from bruising and later pallor from ischaemia. The 2nd grade is skin blistering, abrasion, and ulceration. The 3rd grade is deep ulceration with undermining of adjacent tissue. The 4th grade is necrosis, which is often deeper than you might imagine, with osteitis and subsequently pyoarthrosis.

Pressure sores occur in:
(1) the paralyzed,
(2) the severely debilitated patient,
(3) those with neuropathy.

Damage to the skin from pressure is made worse by contamination with pus, urine or faeces; with septic sores or incontinence, huge sores can develop within 48h (54.11).

Obviously it is best to prevent this disaster happening: in hospital this should be possible with dedicated nursing, turning a patient regularly every 2h *day and night*.

However, patients may come to you from elsewhere with pressure sores, expecting a miracle.

You will not succeed with surgery for pressure sores if you cannot provide the dedicated nursing these patients need; avoid pressure on the suture lines!

N.B. You will not get a pressure sore to heal on an HIV+ve patient without antiretroviral therapy (5.8).

(a) Special tests

Screen for HIV and diabetes. Check the Hb level. Take skin snips if you suspect leprosy. Take pus swabs. If you suspect osteomyelitis, X-ray the underlying bones.

(b) Treatment

(1) **Psychological care and routine nursing** of the paraplegic is essential; if this is not available, *don't embark on difficult surgery*. Make sure pressure is kept off the sore, and the patient is turned every 2h. Hammocks, sheepskin and inflatable mattresses are all helpful but do not substitute for nursing care.

(2) **Correct the nutritional deficit**; these patients need high-protein, high-calorie diets even just for healing.

(3) **Clean and dress the sores**, initially twice daily, and treat HIV disease and diabetes. Transfuse blood if the Hb is <70g/L. *Don't use antibiotics unless there is spreading cellulitis*.

(4) **Debride** the pressure sore: a small ulcer often hides a large area of necrosis. You have to be radical with the removal of ischaemic tissue, otherwise sepsis will continue and necrosis will extend. The aim of getting successful skin cover is to remove the pressure point, so *don't be afraid to remove bony prominences*, especially if they too are ischaemic or septic.

N.B. An initial debridement on a paraplegic needs no anaesthetic (because of the sensory loss), but beware blood loss, which will not be compensated by peripheral vasoconstriction. *Sudden hypotension may be catastrophic!* Use sedation or a light anaesthetic for subsequent debridements and flaps, if you need to move the patient intra-operatively.

For a sacral sore, **divert urine and faeces** by catheterization and a temporary sigmoid colostomy (11.5). Though this adds considerably to the burdens of the patient, it significantly eases nursing care, and avoids contamination of the sore. *It is very disappointing to see a successful graft or flap destroyed by sepsis!*

(5) **Obtain skin cover** *when the wound is clean*, nutrition and anaemia corrected, HIV treated and diabetes controlled. Good, lasting skin cover is rarely possible by simple skin grafting, although this may be a useful preliminary to a flap procedure. The type of flap depends largely on the site of the pressure sore.

It is best to use a piece of cloth as a template for the flap; mark out the size and shape of the flap on the skin and cut it always a little larger to allow for errors of calculation. For pressure sores, it is usually not possible to fashion an advancement flap (where you loosen the base of a flap to give it greater stretch). You will need to use a transposition (34-20) or rotation flap (34-21). Triangulate the defect to be excised, making 2 equal sides longer than the base (34-19).

Such a flap takes the skin and underlying subcutaneous tissue, but *not* muscle. However, the blood supply of a myocutaneous flap, such as the *tensor fascia lata* (34-12), *gracilis*, *latissimus dorsi* and *pectoralis major* flaps, is much more reliable and makes them worth learning from an expert.

N.B. Be careful when you prepare the flap that you don't damage its arterial supply.

PLANNING A LOCAL FLAP

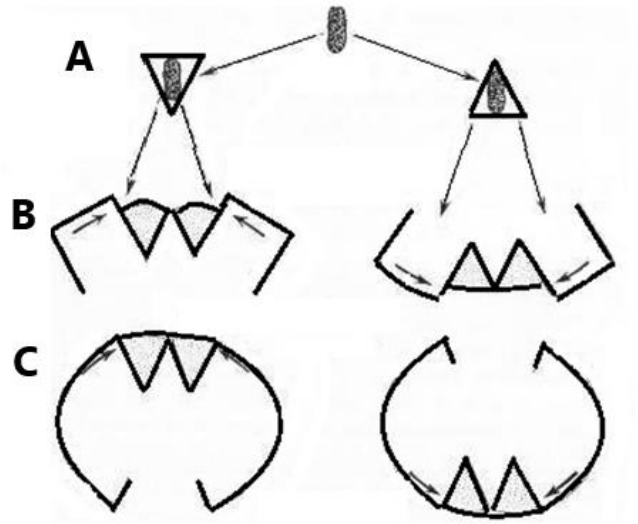


Fig. 34-19 STEPS IN PLANNING A LOCAL FLAP.
A, Step I, triangulate the defect, and select the direction of its base. B, Step II, select whether a transposition or C, rotation flap is best, and select on which side of the defect the flap should be constructed. *After MacGregor IA. Fundamental Techniques of Plastic Surgery, Churchill Livingstone 1980 p.139 Fig 4.28.*

Make sure, when you make a transposition flap, that you make it approximately square (*its length must never be greater than its base*) and that there is adequate length of the flap (longer than the triangular defect) so that it pivots around a point on its base furthest from the defect (34-20).

DESIGN OF A TRANSPOSITION FLAP

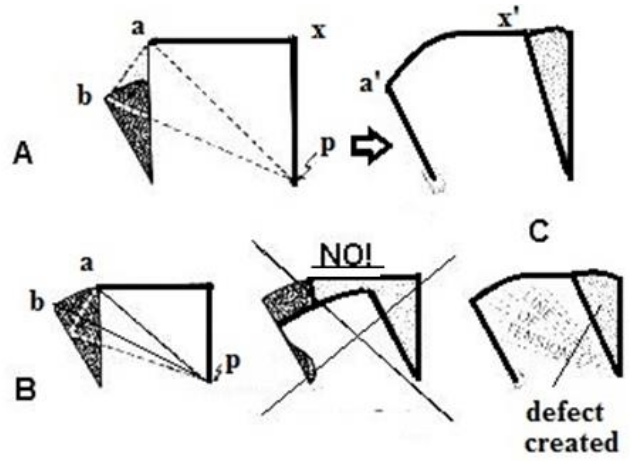


Fig. 34-20 DESIGN OF A TRANSPOSITION FLAP.
A, a correct design where pa=pb; a moves to a' and x to x' (p is the pivot point). B, an incorrect design where pa<pb and transfer will only be achieved with tension. C, lines of tension; close the defect created primarily or with a skin graft.
After MacGregor, IA. Fundamental Techniques of Plastic Surgery, Churchill Livingstone, 1980. p.140 Fig 4.29.

When you make a rotation flap, the bigger the flap size, the less tension there will be; make a 'back-cut' (34-21) along the diameter of its circle as this will allow some lateral movement as well. Close the defects created primarily, or secondarily with a skin graft if there is not enough laxity to close without tension.

For both types of flap, use subcutaneous suction drains post-operatively.

DESIGN OF A ROTATION FLAP

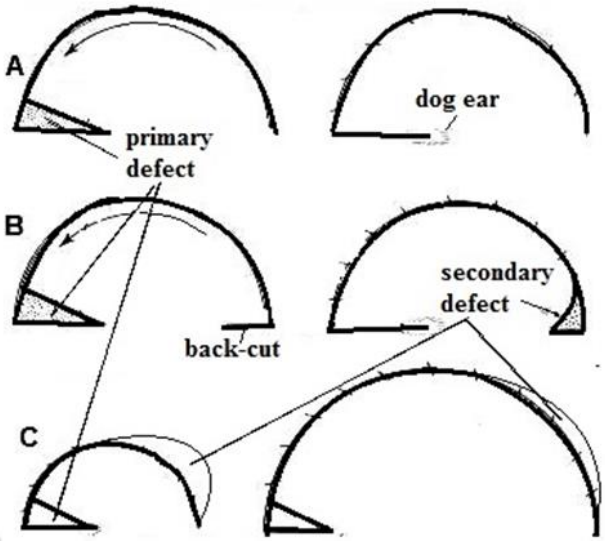


Fig. 34-21 DESIGN OF A ROTATION FLAP.
A, without back-cut making a dog ear. B, with back-cut making a secondary defect needing closure. C, difference on tension with flap size: the area of the secondary defect is the same with both flaps.
After MacGregor, IA. Fundamental Techniques of Plastic Surgery, Churchill Livingstone 1980 p.137 Fig 4.27.

If there is a sacral sore, use an INFERIORLY-BASED BUTTOCK ROTATION FLAP (GRADE 2.5). If the patient is paraplegic, use a unilateral rotation flap or bilateral flaps which will give you more cover (34-22).

SACRAL PRESSURE SORE REPAIR

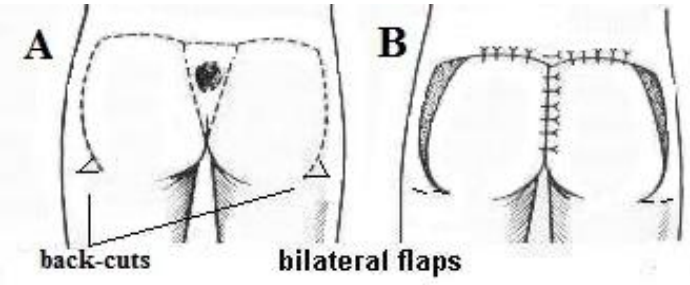


Fig. 34-22 SACRAL PRESSURE SORE REPAIR.
A, marking the flaps, with back-cuts. B, secondary defects, which you can usually close primarily.
After MacGregor, IA. Fundamental Techniques of Plastic Surgery, Churchill Livingstone 1980 p.230,232 Figs 7.2,4.

If there is a trochanteric sore, use a LATERALLY-BASED POSTERIOR THIGH TRANSPOSITION FLAP (GRADE 2.5).

You can make the blood supply of the flap more reliable if you incorporate the ilio-tibial tract (the *tensor fascia lata* muscle) into the flap, thus making it a myocutaneous flap. (Its length can then be up to 3x its base.) Remember to cut off a wedge of protruding femoral trochanter (34-23C).

TROCHANTERIC PRESSURE SORE REPAIR

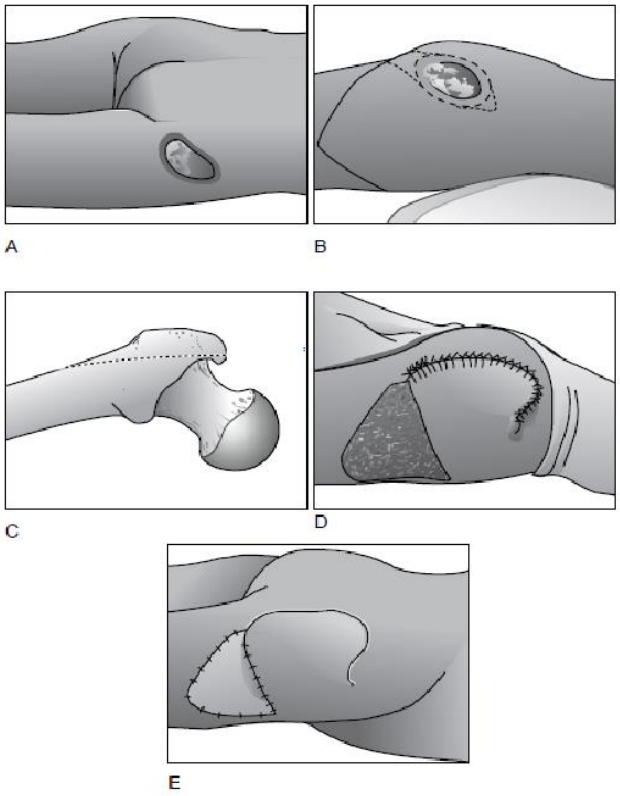


Fig. 34-23 TROCHANTERIC PRESSURE SORE REPAIR.
A, outline the sore. B, mark out the transposition flap on the lateral upper thigh. C, excise a lateral wedge of trochanter. D, transfer the flap by rotating it clockwise. E, skin graft the remaining defect.
After MacGregor, IA. Fundamental Techniques of Plastic Surgery, Churchill Livingstone 1980 p.231 Fig 7.3.

If there is an ischial sore, use a MEDIALLY-BASED POSTERIOR THIGH TRANSPOSITION FLAP (GRADE 2.5).

ISCHIAL PRESSURE SORE REPAIR

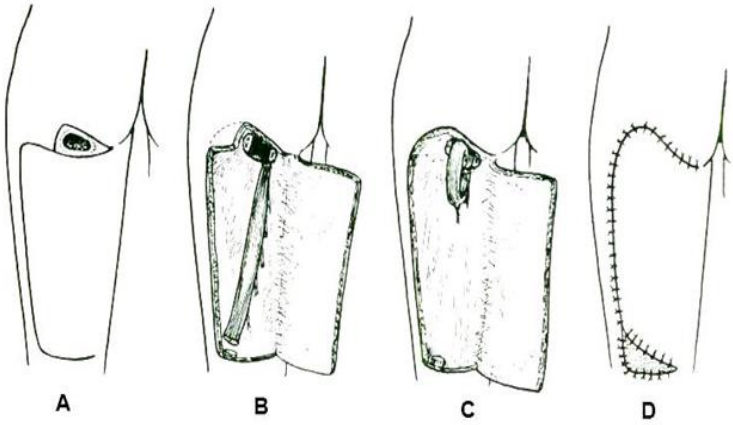


Fig. 34-24 ISCHIAL PRESSURE SORE REPAIR.
A, mark the transposition flap. B, expose the hamstring muscle with the flap prepared. C, divide the hamstring muscle and roll its distal end into the cavity left by excising the ischial tuberosity. D, close the wound, and graft the small defect inferiorly.
After MacGregor, IA. Fundamental Techniques of Plastic Surgery, Churchill Livingstone, Edinburgh 1980. p.233 Fig 7.5.

Flex the hip when you plan the flap: *ensure suture lines don't rest on pressure points*. Make a very broad-based flap, and excise the ischial tuberosity; this leaves a dead space which, in a paraplegic, you can fill by dividing the lower end of the hamstring muscle, dividing the lower half of its perforating vasculature, and rolling it upwards into the defect (34-24C). Then transpose the flap superiorly to cover the defect.

N.B. If you use a *tensor fascia lata* flap, the blood supply is more assured, but make the flap longer.

HEEL PRESSURE SORE REPAIR

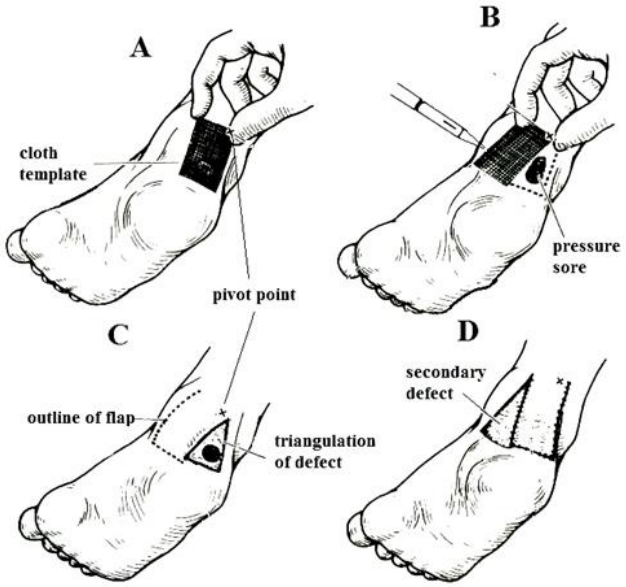


Fig. 34-25 HEEL PRESSURE SORE REPAIR.
A, make a cloth template of the flap. B, outline the flap, noting its pivot point, and triangulate the defect. C, cut the flap. D, transpose the flap and graft the residual defect.

If there is a foot sore, examine the insensitive foot carefully (32.11). For an uncomplicated heel sore, use a SUPERIORLY-BASED POSTERO-MEDIAL TRANSPOSITION FLAP (GRADE 2.5).

Mark the flap out carefully, triangulate the defect, and transpose the flap. Graft the residual defect.

N.B. Don't take skin from the ball of the heel: it is specialized for walking!

If the Achilles tendon is involved in the sore, remove its necrotic parts; sacrifice it in a paraplegic.

If the foot is hopelessly infected, perform an amputation (35.3).

35 Vascular surgery

35.1 Varicose veins

ANATOMY AND PHYSIOLOGY

There are four categories of leg vein, and they all have valves which stop blood flowing downwards away from the heart.

(1) **Long and short saphenous veins** run superior to the deep fascia, and are usually deep to the fibrous layer of the superficial fascia (35-1). They have numerous valves, the most important of which is the femoral valve, in the long saphenous vein, just before it penetrates the deep fascia to join the femoral vein. The femoral valve prevents blood from the femoral vein flowing back into the saphenous vein.

(2) **Superficial collecting veins** are tributaries of the saphenous veins. They lie between the skin and the fibrous layer of the superficial fascia. These have valves, but they are poorly supported by connective tissue.

(3) **Deep veins** accompany the arteries, and run among the muscles deep inside the leg. These have important valves.

(4) **Perforating veins** pass through the deep fascia, joining the superficial collecting veins to the deep veins. Their valves direct blood into the leg. The most important of these perforating veins are just behind the medial border of the tibia.

Standing at rest, the superficial veins on the dorsum of the foot support a column of blood that reaches to the right heart. While the leg muscles are relaxed, this blood flows through the perforating veins, into the deep veins inside the leg. On walking, the contractions of the leg muscles squeeze the blood from the deep veins up towards the heart. This cycle of contraction and relaxation reduces the pressure in the superficial veins, and prevents varicosities.

However, if the valves of the deep perforating veins are incompetent, blood from inside the leg is pushed out at high pressure into the unsupported superficial collecting veins. This distends them, and makes them varicose. The increase in venous pressure makes capillary pressure increase, which results in tissue oedema, and leakage of fluid into the tissues, hence tissue oedema. This fluid is rich in albumin and so infection is a real risk, especially as the nutrition of overlying skin becomes impaired.

If the valves which guard the long and short saphenous veins are incompetent, the blood in the femoral and popliteal veins can flow downwards, into the saphenous veins, and make them varicose.

The aim of surgery is to stop blood flowing backwards through veins with incompetent valves.

Varicose veins are the result of failure of the valves in the venous system, which takes 2 forms:

(1) Primary: the valves of the saphenous system fail, while the deep veins of the legs remain normal; the symptoms are usually mild, and the legs rarely ulcerate.

(2) Secondary (post-thrombotic): the deep veins, or the communicating veins between the superficial and deep systems, have had their valves destroyed by thrombosis; ulceration is more common, and treatment more difficult.

Varicose veins are generally associated with Western lifestyles; obesity and low-fibre diets play a role. They are unsightly and cause aching and cramps, a scaly, itchy, varicose eczema, swelling of the legs, and ulceration; occasionally they bleed. Symptoms may bear little relationship to their size and extent. If they are primary, the swelling usually only involves the feet and ankles, and resolves completely overnight. If they are secondary, the lower legs may be swollen all the time.

Make sure the pain is due to the varicose veins (relieved on lying down, worse at the end of a day's standing), and not due to (invisible) ischaemia, arthritis of the hip or knee, a prolapsed intervertebral disc, or *meralgia paraesthetica* (32.17) which can be there at the same time as the (visible) varicose veins. Swelling of the legs may co-exist with varicose veins, but is usually due to another cause, *e.g.* heart failure or lymphoedema.

Very occasionally varicose veins are the result of an **arterio-venous fistula**: you should be able to hear a bruit and feel a thrill over the fistula. The veins may be enormous. Occlusion of the fistula by pressure will, however, make them disappear.

VARICOSE VEINS

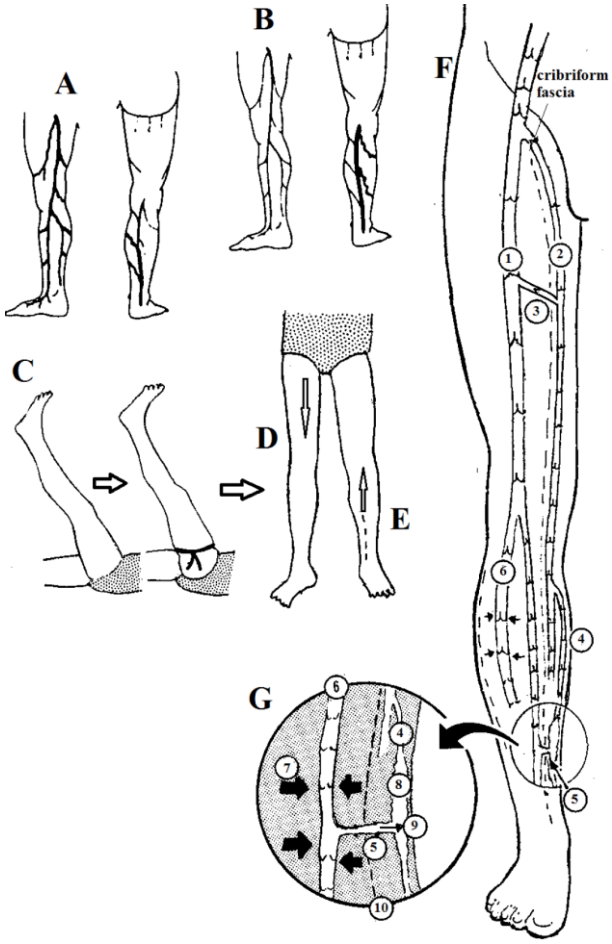


Fig. 35-1 VARICOSE VEINS: ANATOMY.
A, varicosities of the long saphenous system. B, varicosities of the short saphenous system. C, Trendelenburg test for the long saphenous vein: lay the leg supine and then raise it. Apply a venous tourniquet just below the saphenous opening. Stand the patient up and release the tourniquet. D, if the femoral valve is incompetent, the veins fill immediately from above. E, if it is normally competent, they fill slowly from below. F, anatomy of the leg veins; the long saphenous enters the femoral vein through the cribriform (deep) fascia. G, close-up view of a varicosity, and an incompetent perforating vein connecting it with the deep venous system. (1) femoral vein. (2) long saphenous vein, passing through the cribriform fascia. (3) mid-thigh perforating vein. (4) superficial collecting vein. (5) perforating vein with its valves destroyed. (6) deep veins of the leg. (7) muscular forces compressing the deep veins. (8) varix in a superficial collecting vein. (9) blood forced through a perforating vein with an incompetent valve. (10) superficial fascia.
After Ellis H, Calne RY. Lecture Notes on General Surgery, Blackwell Science, 10th ed 2002 p.93 Fig 12.1, with kind permission.

BE SURE THAT THE VALVES OF THE DEEP VEINS ARE COMPETENT BEFORE YOU TIE THE SUPERFICIAL VEINS

ANATOMY OF VARICOSE VEINS

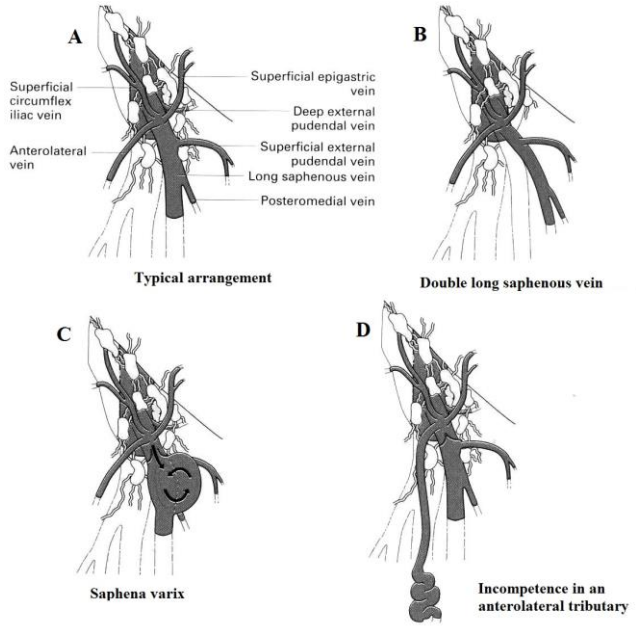


Fig. 35-2 VARICOSE VEINS: GROIN ANATOMY.
Variations in the anatomy: A, typical arrangement. B, double long saphenous vein. C, saphena varix. D, incompetence in an anterolateral tributary.
After Morris, PJ, Malt RA. Oxford Textbook of Surgery, OUP, 1994 p.550 Figs 51a-c.

(a) Examination. Examine the patient standing in a good light. Feel the veins. If he is obese, percuss the course of the saphenous veins. Examine the peripheral pulses. Feel for a thrill in the vein above as you tap it below, and listen for a bruit of a (rare) arterio-venous fistula.

If there is ulceration, thick induration, and marked hyperpigmentation, the valves of the deep veins are almost certainly incompetent, and the varicose veins are secondary. Otherwise they are probably primary.

Perform the Trendelenberg test:

To test the competence of the perforating veins and the valves of the greater saphenous system, lay the patient supine, raise the leg, and massage the veins proximally to empty them. On the upper thigh apply a rubber tourniquet, tight enough to compress the veins. Then ask him to stand and move the forefoot up and down, so as to actuate the calf muscle pump. Inspect the varices for 30secs, and then remove the tourniquet (35-1C,D,E).

If the veins gradually fill from below on standing, **and continue to fill gradually from below when the tourniquet is released**, *the valves in the veins of the legs are normal*.

If the veins fill rapidly from below, the varices are being filled from the deep veins, and *the valves of the perforating veins are incompetent*.

If blood flows rapidly into the long saphenous vein from above, after removing the tourniquet, the *long saphenous valve is incompetent*.

If finger pressure on the upper part of a thigh varicosity controls blood flow, the *anterolateral tributary is incompetent, bypassing the main long saphenous valve* (35-2D).

To test the competence of the valves of the short saphenous vein, lie the patient flat and apply 2 tourniquets, one above the knee to occlude the long saphenous vein and another just below the popliteal fossa to occlude the short saphenous vein. Ask the patient to stand up, leave the upper tourniquet on, and remove the lower one. If the blood flows immediately into the short saphenous vein from above, the *short saphenous valve is incompetent.*

To find the sites of major incompetent perforating veins:
(1) Look for visible and palpable 'blowouts' of subcutaneous veins.
(2) Feel for circular gaps in the deep fascia below the visible or palpable 'blowouts'.
(3) Repeat the tourniquet test at each level above a 'blowout', to see if this prevents the 'blowout' appearing.

(b) Special tests. Doppler ultrasound is easy: pressure on the vein above causing a rush flow below indicates incompetence. *N.B. Don't perform a venogram with contrast*: you may cause the thrombosis you want to avoid!

(c) Diagnosis
Suggesting primary varicose veins: usually start at 15-25yrs. No incompetence of the perforators shown by the test above. Incompetence demonstrated by back-flow on release of the upper thigh tourniquet (long saphenous), or just below the popliteal fossa (short saphenous).

Suggesting secondary varicose veins: obesity, multiple pregnancies, or a pelvic tumour; a history of venous thrombosis, an older age, less obvious veins partly hidden by eczema, fat necrosis, or ulceration.

(d) Non-operative treatment
INDICATIONS
(1) Minor symptoms.
(2) Uncertainty whether symptoms are really due to varicose veins.
(3) Untreated HIV disease.

METHOD
Encourage weight loss if appropriate, frequent walking, avoiding prolonged standing and sitting, and raising the leg when seated. Try to fit graduated compression stockings from the distal metatarsals to thigh or calf (depending on whether the long or short saphenous system is affected).

CAUTION! Make sure that the stockings are not too tight to give a tourniquet effect: however, *they are no use if too loose!* Ordinary elastic bandaging is not as good but better than nothing.

N.B. They are uncomfortable in hot climates though!

SCLEROTHERAPY OF VARICOSE VEINS

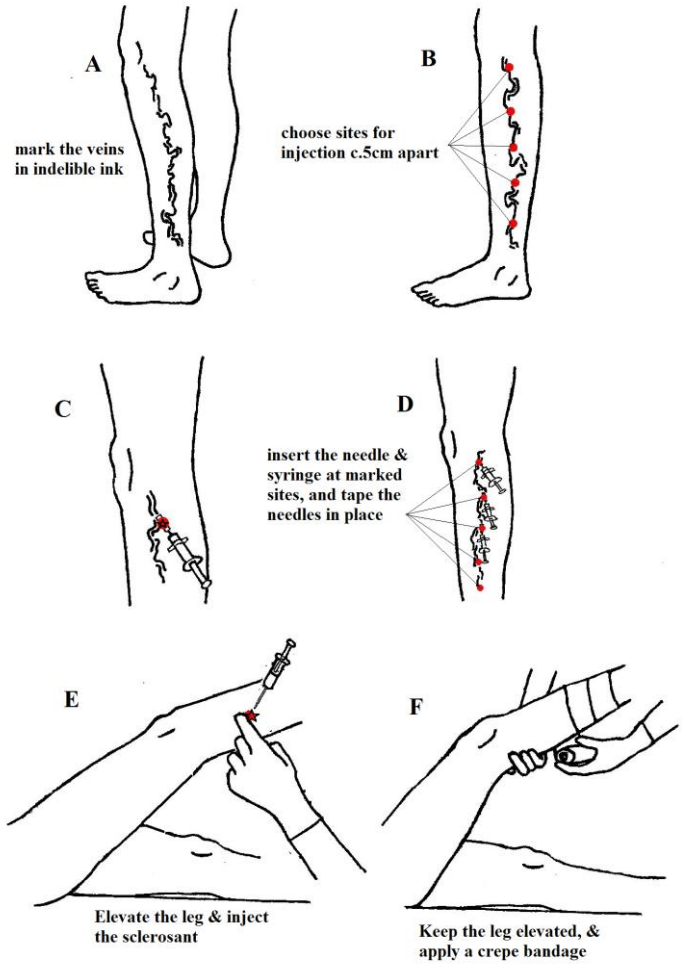

A
mark the veins in indelible ink
B
choose sites for injection c.5cm apart
C
D
insert the needle & syringe at marked sites, and tape the needles in place
E
Elevate the leg & inject the sclerosant
F
Keep the leg elevated, & apply a crepe bandage

Fig. 35-3 SCLEROTHERAPY.
A, mark the visible varicose veins. B, choose maximum 5 injection sites c.5cm apart: mark these in a different colour ink. C, insert a needle and syringe and check that the needle is correctly in the vein by withdrawing a spot of blood, then tape needle and syringe to the skin. D, tape further needles and syringes to the skin. E, elevate the leg and inject the sclerosant, starting from the site nearest the foot, and apply pressure. F, apply crepe bandage up to last injection site, and continue the injections.
Kindly contributed by George Poulton.

(e) Sclerotherapy (GRADE 1.2)

INDICATIONS

(1) The cosmetic treatment of small primary varicose veins.
(2) Incompetent perforating veins without an obvious incompetent major valve.
(3) Varicose veins which persist or recur after stripping.

CONTRAINDICATIONS

(1) An incompetent major valve.
(2) Large varicosities.
(3) Gross obesity (it is difficult to maintain compression).
(4) Deep venous thrombosis.

EQUIPMENT. 5 small syringes fitted with fine needles and filled with 0·5ml of 3% sodium tetradecyl sulphate, 5% ethanolamine oleate, or 0.5-2% polidocanol (especially as a foam). 2 different colour marking pens, strapping, gauze, crepe bandages and graduated compression stockings.

CAUTION! Anaphylaxis is rare but may occur, so have hydrocortisone and adrenaline to hand.
The veins must be almost empty when you inject, and be kept empty so that their walls adhere, so careful bandaging is critical. *If you inject sclerosant into an artery, you may cause extensive gangrene, so don't inject around the ankle.*

METHOD

You need an assistant. Ask the patient to stand up; observe, palpate, and percuss the veins; mark them with a permanent marking pen. Then ask him to lie down, elevate the foot, and feel the course of the veins for gaps in the fascia (sites of incompetent communicating veins). Mark these with a pen of a different colour. Press with the tips of your fingers on as many of these gaps as you can, and, still pressing, ask him to stand. Remove your lowermost fingers first. If removing your finger from a gap in the fascia immediately causes the vein to fill, that gap is the site of an incompetent perforating vein. If it does not fill, there was no perforator in it. The sites where pressure controls the filling are the best sites for injection. Inject the lowest sites first.

Ask the patient to sit on a couch with the affected leg over the edge of the bed so that the vein fills, insert the mounted needle at the marked sites c.5cm apart, and aspirate only as far as the transparent hub to be sure you are in the vein, and strap the syringes securely to the skin; then empty the vein by raising the leg above the horizontal. Isolate the segment to be injected by pressing with your fingers above and below it, and inject 0·5ml of sclerosant. Apply a pressure pad over the injection site to keep the vein empty, and apply a crepe bandage up to that site. Then move up to the next site, and repeat the process until all your chosen sites have been injected. *Don't inject >5 sites*. Leave a gap in the bandaging for knee flexion.

N.B. You might find butterfly needles easier.

Apply a graduated compression stocking over the bandage and immediately encourage walking for 1h, and thereafter for 5km daily. Advise elevation of the legs as much as possible. Remove the bandages for a wash after 1wk. Then re-apply the bandages od, with the leg raised, for at least 3wks in order to maintain pressure without interruption. If the bandages become loose, reapply them. Further injections for missed or recurrent veins may be necessary: wait 3 months before doing this.

If there is severe pain after the injections, take off the bandages with the leg elevated and look for skin necrosis or gangrene. Use analgesics and encourage walking if there are no untoward effects.

(f) Long saphenous vein ligation, stripping and avulsions for varicosities (GRADE 2.5)
Determine whether there is long sapheno-femoral or short sapheno-popliteal incompetence. Make sure the deep venous system is patent. Wait till after delivery if the patient is pregnant. If a patient is on oral contraceptives, she should stop them one month before operation.

EQUIPMENT. STRIPPER, for varicose veins, Nabatoff, in sterilizer case, complete with 3 metal olives, cable and handle. (Some strippers have olives that can be attached at either end.)
N.B. A sterilized scooter clutch wire works as well!

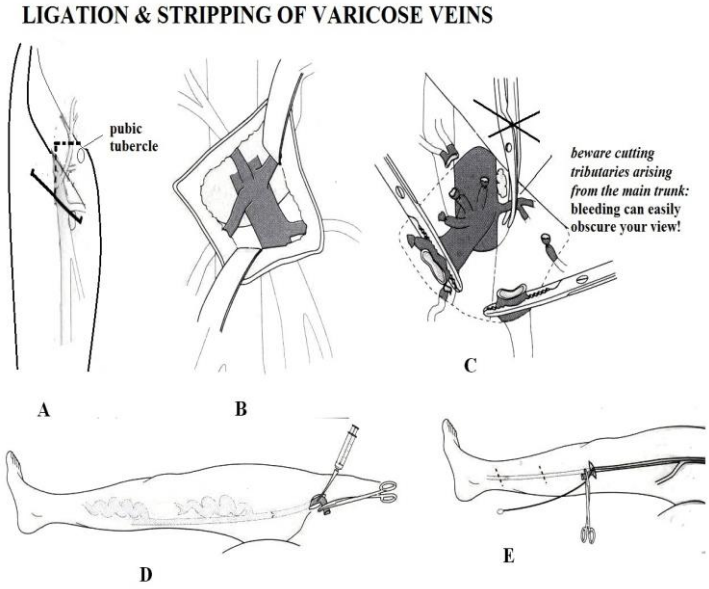


Fig. 35-4 VARICOSE VEINS: LIGATION & STRIPPING.
A, incision 2cm lateral and below pubic tubercle. B, expose the saphenous vein and tributaries (note the medial axis of the long saphenous vein compared to the femoral). C, divide the saphenous vein and tributaries. D, syringe test to prove incompetence. E, pass the stripper through the groin downwards.
After Morris PJ, Malt RA. Oxford Textbook of Surgery. OUP 1994, p.551-2 Figs 51e(i,iii),f(iv,vii),g(ii),h(ii).

PREPARATION
Scrub the groin thoroughly with betadine and make sure the legs are well washed beforehand. With the patient standing, mark the vein to be operated on, and all its tributaries and dilations, using a permanent marking pen. Also find and mark the perforating veins, using the finger-pressure method described above.

FOR THE LONG SAPHENOUS VEIN, lay the patient supine with a 30º head-down tilt with the feet apart. Support the heels on foam cushions.

Make a 5cm oblique incision 1-2cm below and parallel to the inguinal ligament, centred a finger-breadth medial to the pulsating femoral artery (35-4A). Deepen the incision, until you reach the superficial fascia. Proceed carefully using non-toothed dissecting forceps, spreading the fatty tissues gently with scissors to expose the saphenous vein (35-4B).

You may find the superficial external pudendal artery in the way, running over or under the long saphenous vein; make no hesitation in ligating and dividing it if it is in the way. You often come to tributary veins of the saphenous first before the vein itself. Tie and divide all these tributaries (35-4C). Then dissect out the groin end of the saphenous vein and demonstrate the sapheno-femoral junction as the vein dips down through the cribriform fascia (35-1F).

Tie the saphenous vein securely with two ligatures as close to its entry to the femoral vein as you can, making sure no further tributaries enter above your ligature. Clamp the saphenous vein a suitable distance from the femoral vein with haemostats and divide it.
N.B. There may be a ***saphena varix***, a dilated saccule below the incompetent valve; you will need carefully to get above this to ligate the vein satisfactorily.

CAUTION! **If you encounter much bleeding**, *don't clamp blindly with haemostats*, or you may damage the femoral vein, or even the femoral artery. Instead, apply pressure, and raise the foot of the table. Get the suction ready. After 3mins pressure you can usually find the bleeding point and control it, with either a haemostat or a fine silk stitch.

Then mobilize more length of the saphenous vein down the leg; place an untied ligature round it and, holding one edge of the vein carefully with dissecting forceps, release the haemostat. If there is too much bleeding from the open vein (because the patient was not put in head-down tilt), re-apply the haemostat further down the leg. Introduce a wide-bore cannula into the lumen of the vein, and inject 20ml of saline; no resistance should be found, and the varicosities should be shown to bulge (35-4D). If this is the case, introduce the stripper (one with a suitably-sized olive) into the vein and tie the ligature to prevent it falling out (35-4E).
N.B. Make sure you have put the stripper inside the saphenous vein and not the femoral*!*

Release the haemostat, if applied, and manipulate the stripper down the leg, by twisting and turning it. You may guide its passage by external manipulation as well. Try to get it to mid-calf position; *don't try to go as far as the ankle where nerves are close to the vein and may be damaged by avulsion*. Sometimes the stripper gets stuck because of the tortuosity of the varicose vein; in this case you can make a small incision over where the stripper has reached, dissect out the vein and manipulate it further by direct vision. You may have to open the vein between ligatures to do this successfully.

When the stripper has reached its destination, make a 2cm incision over the olive, dissect out the vein, pass looped ligatures underneath the vein at two sites, tie the lower ligature tight and divide the vein. Extract the olive from inside it, and tie the ligatures round the stripper and vein.

Now make 0·5cm-long incisions through the skin over the various tributaries marked in indelible ink. Select prominent remaining varicosities, and by spreading forceps raise a loop of varicose vein by gentle blunt dissection. Follow it as carefully as you can in each direction and when you have exposed as much length of vein as you can, pull it out. Get your assistant to press on the site to stop the bleeding. There is usually no need to tie the vein unless it is large or perforates the deep fascia. Close the small skin incisions with one suture.

N.B. Don't go as far as the ankle with avulsions.

Now raise the leg high and slowly pull the stripper attached to the vein out from the groin. Keep the leg high for a few minutes afterwards to reduce bleeding. Examine the groin wound for bleeding. Examine the avulsed vein for its length to make sure you have extracted it *in toto*; make sure the olive is still attached to the stripper. Close the remaining wounds, and bandage the leg firmly.

(g) Difficulties with stripping varicose veins

If the varicosities don't bulge with the syringe test, incompetence may be in the anterolateral tributary (35-2D): dissect this out, and do the test again. Pass the stripper down this vein.

If the stripper does not pass below the knee, make an incision over the marked route of the varicosed saphenous vein at a suitable point, and introduce another stripper in the same way from below, so that the 2 strippers meet. You can then try to manipulate the proximal stripper upwards, following withdrawal of the first one, or simply pull out each stripper in turn.

If the olive becomes detached, palpate where it has dislodged, and make a 3cm incision over it. Dissect down and remove it; bleeding may be troublesome, so have suction ready.

ANATOMICAL VARIATIONS OF THE SHORT SAPHENOUS

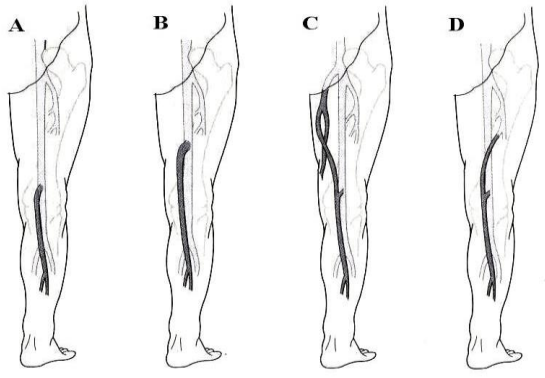


Fig. 35-5 ANATOMICAL VARIATIONS OF THE SHORT SAPHENOUS VEIN.
A, normal level of the sapheno-popliteal junction. B, high level termination. C, upward extension to join the posteromedial tributary of the long saphenous vein. D, upward extension deep to the profunda femoris vein.
After Morris PJ, Malt RA. Oxford Textbook of Surgery, OUP, 1994, p.553 Fig51j.

FOR THE SHORT SAPHENOUS VEIN, you need either spinal anaesthesia or GA and intubation. Use at least 2 assistants to turn the patient by a log roll and lay him prone with the feet apart, and the knees slightly flexed. Put pillows under the chest and pelvis, and make sure the neck is supported, and the abdomen can move freely. It is best to put the arms flexed above the head.

CAUTION! If your team is not familiar with GA in the prone position, don't attempt it!

Make a transverse incision across the middle of the popliteal fossa and deepen it through the deep fascia to expose the short saphenous vein (which lies deep to it). Dissect it out, ligating its tributaries, and trace the knee end down into the popliteal fossa, and doubly ligate it close to its communication with the popliteal vein.

CAUTION! The anatomy of the short sapheno-popliteal junction is notoriously variable (35-5). *Don't attempt to strip the short saphenous vein* as it is closely accompanied by the sural nerve, and avulsion may well result in anaesthesia of the lateral foot and calf. Also, there may be short *and* long saphenous incompetence! However, select prominent remaining *superficial* varicosities outside the deep fascia, dissect these out through 0·5cm incisions, and avulse them.

POST-OPERATIVELY, keep the legs elevated at 30º for 24h. Encourage walking as soon as possible for 1h daily. Leave the pressure bandage applied at operation for 1wk, then remove the sutures. Advise wearing bandages for a further 2wks.

If varicose veins recur, try sclerotherapy if the varicosities are limited. Recurrence may occur when some tributaries of the saphenous vein or a double vein are not ligated; to re-explore the sapheno-femoral junction is quite hazardous because of scarring, though, and mapping of the veins by Doppler is usually necessary.

(h) Complications of varicose veins

(1) VARICOSE ULCERATION

N.B. Most lower leg ulcers in the tropics are chronic tropical ulcers (34.9) and varicose ulcers due to incompetent perforators are less common. If the ulcer is not typically the result of varicose veins, consider alternative causes.

A varicose ulcer is usually on the lower ⅓ of the leg, especially just behind and above the medial malleolus. It may be of any size and shape, its edges are usually brown and eczematous, and it has red granulations under the slough on its base. The patient is usually obese. Progressive fibrous atrophy of the subcutaneous tissues of the lower leg ('inverted bottle leg') and liposclerosis precede ulceration. There are gross varicose veins of many years' standing with incompetent deep perforating veins in 50% of cases.

TREATMENT
Be prepared for a long haul! Insist on bed rest and apply frequent sterile water soaks until the ulcer is clean and oedema has gone. Deslough the wound, and when clean, apply betadine or zinc oxide paste.
Only use antibiotics if there is associated cellulitis (6.22). *Don't use topical antibiotics.*
Advise cleaning the ulcer once or twice daily, and coating the ulcer weekly with platelet-rich fibrin, and sleeping with the foot of the bed raised.
When dressings are no longer cumbersome, apply graduated compression stockings from the base of the toes to the thighs. Recent ulcers (<3 months) will often heal this way, but a graduated compression stocking should be worn for life, despite the discomfort in the heat.

If the ulcer will not heal or recurs, there is no history of deep vein thrombosis and you can be sure of the venous abnormality, check for malignant change (characterized by firm nodules) and take biopsies. Otherwise, treat the varicose veins by operation to reduce orthostatic venous pressure on the skin. Try honey, ghee, pawpaw or banana dressings (2.10). Then think of skin grafting if the ulcer surface is granulating well (34.9).

(2) HAEMORRHAGE
If varicose veins bleed, haemorrhage can be alarming. Elevate the leg and apply pressure to the bleeding vein. If bleeding persists or recurs, take the patient to theatre to expose and isolate the vein and ligate it formally.

(3) SUPERFICIAL THROMBOPHLEBITIS
If an indurated line develops along the course of the vein, advise that it will usually be gone in a month. Use cloxacillin 500mg qds and ibuprofen 400mg tds for 1wk. There is almost no danger of pulmonary embolism.

35.2 Peripheral gangrene

Inadequate blood supply leads to gangrene of the peripheries; strictly speaking, gangrene implies digestion of dead tissue by anaerobic bacteria. This may occur as a primary process in gas gangrene due to *clostridia* (6.24), or more usually secondarily to ischaemia. If there is little subcutaneous fat, and no oedema, the skin becomes cold and waxy, haemoglobin diffuses out of the veins, discolouring the skin purple and then black, and ischaemia results in shrinking of the tissues (mummification or dry gangrene). There results a clear line of demarcation between viable and dead tissue, and the dead part sometimes simply falls off, leaving the stump to heal. At the junction between live and dead tissues, however, organisms do survive, as is the case with the more common scenario of fatty oedematous tissues, particularly found with diabetes mellitus. The result is a mass of infected, necrotic, smelly, partially destroyed tissue, known loosely as wet (or moist) gangrene.

Underlying causes are numerous, and can be:
(1) Sepsis with diabetes mellitus (causing a combination of vasculopathy and neuropathy).
(2) Peripheral ischaemia due to arterial disease (usually because of cigarette smoking), HIV or syphilitic vasculitis, arterial emboli, vascular injury (including injection of barbiturate or sclerosant into an artery, 35.1), and vasospasm due to cold (*e.g.* in trench foot) or, rarely, an accessory cervical rib.
(3) Compartment syndrome due to burns, crush injury, snake bite especially with inappropriate tourniquet use, too tight plaster of Paris, fibrous stricture (*e.g.* ainhum, auto-amputation of the 5th toe) or an acute venous thrombosis.
(4) Septicaemia (from any cause) resulting in simultaneous venous and arterial thrombosis, especially in neonates, HIV+ve patients, and the malnourished.
(5) Necrotizing fasciitis (6.23) or gas gangrene (6.24): these produce a toxic combination of (3) and (4).
(6) Multi-organ failure.

(a) Examination
The diagnosis of gangrene is usually obvious; unfortunately many patients present when gangrene is already established and all you can do is amputate. You therefore need to know where and how to do so.

Make sure ischaemia is established: you may still save toes, feet, fingers or arms if you release an eschar, decompress a compartment syndrome, or simply *slowly* warm up a cold periphery.

Make sure you document all the peripheral pulses (including thrills and bruits), examine for capillary return on the toes or fingers (should be <1sec) and for sensation, and check a random blood glucose and HIV screen (and also VDRL if available). Look for xanthelasmata at the inner canthus of the eyes, indicating hyperlipidaemia, as well as the tell-tale signs of nicotine-stained fingers.

Measure the ratio of the ankle to the brachial systolic pressure (significant occlusion exists if it is <0·85) but this may be unreliable if arteries are calcified as in diabetes or renal failure. If you have a Doppler ultrasound probe, this gives greater sensitivity than the finger and can give very useful objective information about flow rates. However, you may not be able to tell where the occlusion lies, and if there is a stenosis, whether there is a more significant stenosis more proximally placed.

Similarly if you compare the p_aO_2 taken with a pulse oximeter at the big toe and the thumb, you can get an idea of the degree of relative hypoxia: this is significant if the ratio is <0·6; a reading of <20mm Hg in the lower leg demonstrates significant ischaemia.

(b) Vascular reconstruction
It is, sadly, often too late with many patients to consider this. However, with minor areas of gangrene or ischaemia, a patient will do better if you can arrange a successful revascularization of the limb and perform a minor amputation, rather than a major amputation *without any improvement in vascular supply*.

If you see a patient with claudication at <200m, or rest pain, try to refer him before gangrene sets in. You can relieve much pain (and peripheral inflow) with epidural analgesia.

35.3 Amputations in general

(a) Introduction

The decision to amputate is always a drastic one. There is, obviously, no going back, so think about remaining function. This will depend on many factors: the patient's determination, cultural obligations, residual pain, the presence of continuing infection, the healing of the stump, the level of the amputation, the prostheses and the physiotherapy available. Generally speaking, a disarticulation away from sepsis is less likely to become infected than amputation through the bone.

Some prostheses may be of little use in the patient's particular circumstances, and a simple hook or peg leg may be preferable to a sophisticated gadget even if he does get one.

The rule in the arm is to maintain as much length as possible: every cm is useful. For the leg, the crucial element is walking or, failing that, crawling. If the heel is preserved, a patient should be able to use the foot to walk.

In a perfect stump:
(1) The scar is not exposed to pressure.
(2) The skin slides easily over the bone.
(3) The skin is not infolded.
(4) There is no redundant soft tissue.
(5) There is no protruding spur of bone.
(6) The stump is painless.
(7) The wound has healed by first intention.
(8) The skin has good sensation.
(9) The shape of most should be conical.

So try if possible to make sure:
(1) you divide the bone where its cross-sectional area is greatest and so gives broad support,
(2) the whole surface of the cut bone is capable of weight-bearing,
(3) the skin of the stump is centred on the stump, firmly attached, and adapted to weight-bearing,
(4) the stump end is bulbous so the prosthesis does not slide off.

Deciding where to amputate can be difficult. Mention where, why, and how you plan to amputate. Make sure you have properly counselled the patient, the family or any relevant decision-maker and obtained consent for amputation. This may be a decision that is taken after considerable discussion in the family or clan; *don't force them into agreement against their own judgement*, otherwise they will not cooperate.

Mention the possibility of a higher amputation if complications ensue, and mobilize well post-operatively. If the patient is going to take a long time to recover, say so. Discuss any alternatives. Mention the stages of the road to rehabilitation. If you focus on this (going back to work, or walking), you are much more likely to engender enthusiasm about the future. Think of what position the patient is likely to find himself in 12 months' time. Get help from peer groups of amputees.

If you have to operate in an emergency, and you cannot obtain consent because the patient is unconscious and there are no family members present, but you think the amputation is life-saving, get a second opinion, a witness to document this, and take a photo of the limb to be amputated.

AN AMPUTATION IS THE START OF A LIFELONG TREATMENT

N.B. There may be elaborate rules for burying the amputated limb.

An emergency amputation for sepsis or crushed limb may save someone from the jaws of death!

(b) Considerations in leg amputations

You will need to amputate a leg much more frequently than an arm. The lower in the leg you amputate, the greater the chance that the patient will walk again afterwards, especially in the elderly. The longer the bone stump, the less effort is required when walking. Energy expenditure and oxygen consumption increase as the level of amputation rises in the limb: for an above-knee amputation, 65% extra work is needed.

However, the lower in the leg you amputate, the greater the chance that the tissue through which you cut will not be viable. So, feel the pulses carefully and take measurements to assess the degree of ischaemia (35.2): *don't perform a below-knee amputation if you cannot feel a popliteal pulse.* If the tissues have poor bleeding and the muscle is purple at operation, abandon this level and cut more proximally.

The leg must have a prosthesis which will bear weight. There are a limited number of these, and the stumps for them are standardized. So always perform one of the standard leg amputations. There are three technological grades of prosthesis; of these, the third is not necessarily the worst.

WHERE TO AMPUTATE

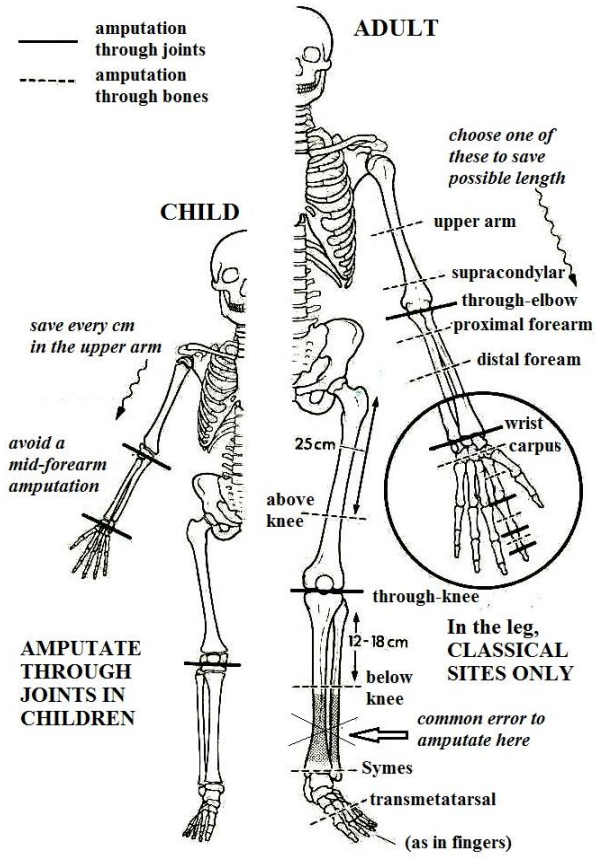


Fig. 35-6 AMPUTATION SITES.
In the arm, save every centimetre in the child's upper arm, and at the joints so as not to affect growth; choose the longest feasible amputation in the adult arm; in the leg, amputate at the classical sites only.

A patient might have:
(1) A sophisticated modern prosthesis costing >US$300.
(2) A simpler modern prosthesis costing US$30, such as the Jaipur foot (35-23A) developed by the BMVSS organization, which a mechanic can mend (www.jaipurfoot.org).
(3) A traditional prosthesis, such as a pylon, a peg leg (35-23B) or elephant boot.

Don't despise the last; when well made, they last longer than any of the others, and are better than a modern prosthesis for working in the fields. Remember that the patient may be used to sitting on the floor rather than on a chair, and so his prosthesis must take this into account. To this end, the Jaipur prosthesis is most suitable. It does not require any shoe: amputees can walk barefoot, or use a shoe. It is made of waterproof material, so that amputees can walk in wet and muddy fields. It permits enough foot dorsiflexion and other movements necessary to walk on uneven surfaces.

A leg prosthesis can:
(1) have a cup to bear weight on the sides of the stump, in which case the scar should be at the end.
(2) bear weight on the end of the stump, in which case the scar should be posterior.
(3) have a modern total contact socket in which the position of the scar is unimportant.

Limb-fitting centres vary in their scope and preferences, so visit your local one and find out what they like. A good prosthetist can fit any well-constructed stump with a prosthesis.

Work in good collaboration with your physiotherapist and prosthetic supplier.

CONSERVE EVERY CENTIMETRE IN THE ARM; DO A STANDARD AMPUTATION IN THE LEG

(c) Considerations in children

The epiphyses play pivotal roles in growing limbs, so try to preserve them. Disarticulation, especially at the knee, will preserve the epiphyses. But aim for a below-knee amputation whenever possible. Children´s skin is much more elastic and it tolerates more tension to close a stump. You might even tolerate skin defects of a stump, as they heal quicker the younger the child is. Amputating through the mid-thigh or foream causes the bone to stop growing, but below-knee and above-elbow amputations produce excessive growth, needing revision later.
The technique of periostal flap cover over the shaft stump might prevent stump bony overgrowth.
If there is no good follow-up, amputate through a joint and avoid complications of bony overgrowth.

(d) Considerations in upper limb amputations

Save as much of the length of the arm as you can, because the patient will probably get no prosthesis. If possible, disarticulate the elbow.
If you can leave a reasonable length of humerus, it can be used to hold things by gripping them against the chest. If you have to amputate very high up, even a very short stump will preserve the outline of the shoulder.
N.B. In some cultures, amputation of the right hand makes you a social outcast!

AMPUTATION EQUIPMENT

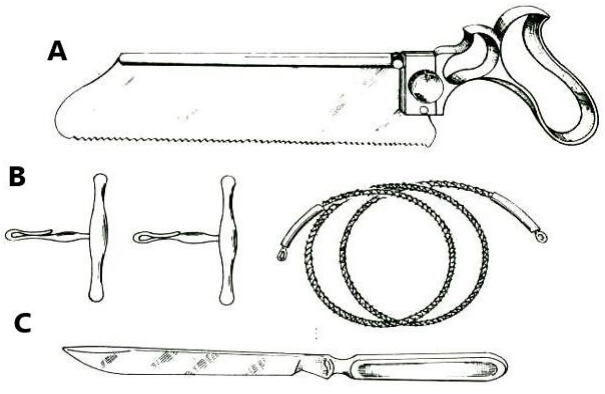


Fig. 35-7 EQUIPMENT FOR AMPUTATION.
A, hand saw. B, Gigli wire and holders. C, sharp long amputation knife. *N.B. If necessary, you can use any sterilizable saw or domestic knife.*

SAW, amputation, with hinged back, 230mm, with spare blades. The back of the saw stiffens it during the early part of the cut, but can be hinged back later to let the saw pass through.
SAW, Gigli, with a pair of handles and 30cm blades. A Gigli bone saw is a piece of wire with sharp teeth on it which you pull to and fro between two handles. Use it to cut bone in awkward places.
KNIFE, amputation, Liston 180mm. If you don't have an amputation knife, sharpen a long kitchen knife and use that.

N.B. An electric saw is a luxury: keeping strict sterility is difficult.

(e) Indications

(1) Gangrene
(2) Chronically infected limb with osteomyelitis
(3) Advanced soft tissue or bone malignancy
(4) A useless limb, such as affected by severe contractures or polio
(5) A gross congenital malformation
(6) An irretrievably damaged limb (60.1).

(f) Methods for amputations

(1) ANAESTHESIA
Relaxation is unnecessary. Ketamine is the preferred anaesthetic. You can use spinal anaesthesia in a haemodynamically stable patient. Spinal and plexus anaesthesia seem to prevent post-operative phantom pain, and are preferable in ischaemia. If a patient is conscious, premedicate him heavily so he cannot hear his bones being sawed through!

(2) TOURNIQUET
Use a tourniquet and exsanguinate a limb by elevating it and applying an elastic bandage from the distal to the proximal end, *except when you are amputating for ischaemia*. *Don't exsanguinate a limb if there is sepsis or malignancy in it!*

N.B. You may have difficulty identifying viable muscle if the tourniquet remains in place.

(3) TECHNIQUES OF AMPUTATION
FLAPS FOR AMPUTATIONS
When you perform an amputation, you are making 2 myoplastic flaps. The classical 'fish mouth' flaps must be long enough to cover the bony stump, but not too long that their blood supply is inadequate or they make a lumpy end. If the flaps are equal, the resulting scar will sit at the end of the stump, but if one is slightly longer than the other, the scar will be on the front (if the posterior flap is longer) or the back (if it is shorter). Aim to place the scar where it is not going to be pressed on. In the leg, its site depends on the kind of prosthesis envisaged: end-bearing, side-bearing, or total contact. In the lower arm and leg, transverse scars are better than antero-posterior because they don't get drawn up between the two bones. In the hand and the foot, place it dorsally. Higher up the arm the scar can be anywhere.
A 'dog ear' at the corner of a wound usually resolves.

The long posterior flap technique is the standard for the below-knee amputation (35-24A) especially in ischaemia. *Equal anterior and posterior, or lateral flaps are liable to fail.*
The skew flap is also good, but more difficult (35-24C).

N.B. The guillotine amputation has no flaps; use it only in special circumstances (60.4).

Decide where you are going to saw the bone (the point of section) and plan the flaps in relation to that point. Place the angle of the fish mouth at the site of bone section. Mark the flaps out carefully with a permanent marker.

THE GEOMETRY OF 'FISH MOUTH' FLAPS

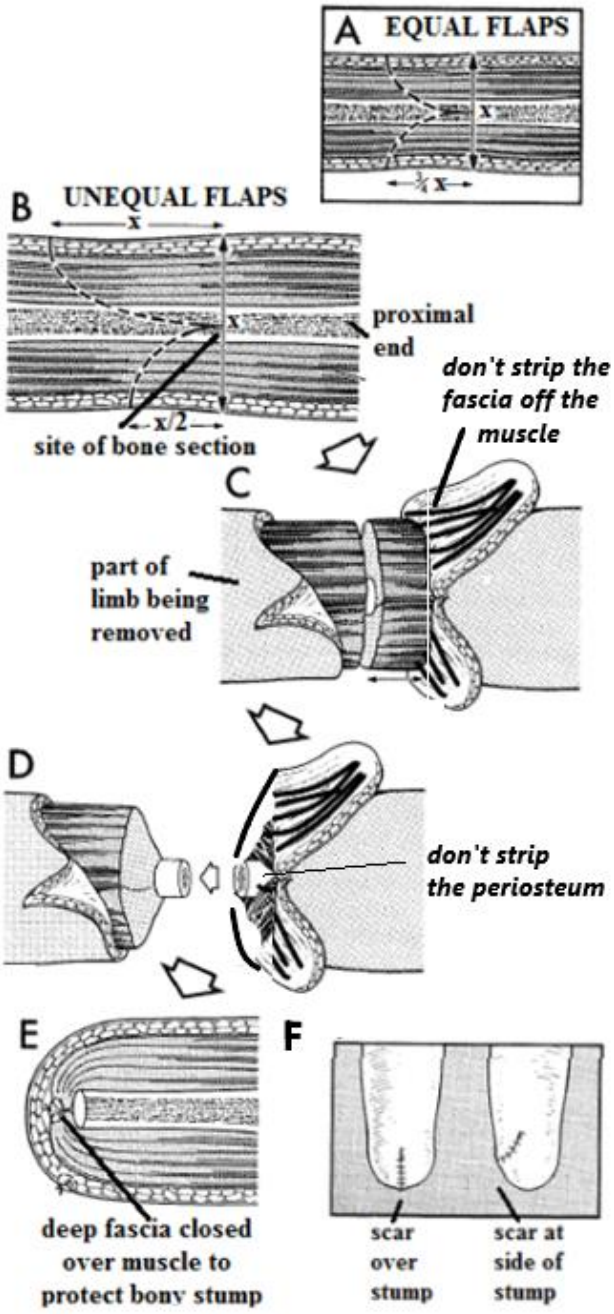


Fig. 35-8 'FISH MOUTH' FLAPS.
Together, the flaps should be 1½ x the diameter of the limb. A, either make them as 2 equal flaps, each ¾ of the diameter of the limb, or B, make 1 flap equal to the whole diameter and the other flap equal to ½ of it. C, reflect the skin with the deep fascia and muscle a little distal to the bone section. D, reflect the periosteum only 1-2cm so you can saw the bone cleanly: *don't strip the periosteum off the bone.* E, close the deep fascia over the bony stump, protecting it with muscle. F, position the scar depending on the type of flap you use.
Kindly contributed by Peter Bewes.

For equal flaps, make the length of each flap equal to ¾ of the diameter of the limb (35-8A), or ½ the circumference.
For unequal flaps, make the longer flap equal to the diameter of the limb, and the shorter one equal to ½ its diameter (35-8B).

N.B. As a general rule, the combined length of both flaps should equal 1½ times the diameter of the limb at the site of the bone section.

CAUTION!
(1) Start by making fish mouth flaps long. You can always trim them if they are too long later, *but you cannot lengthen them if they are too short*.
(2) Cut them round, *not pointed*.
(3) *Make sure the scar is not at the end of the stump if the limb will carry the pressure of a prosthesis.*

Cut the flaps as far distally as you can, so that you can refashion them later. Cut through the skin down to the deep fascia, and reflect this up with the skin as part of the flap. The flaps will need to be of enough length to meet over the end of the stump (35-8E). Minimize trauma to the flaps: handle them with stay sutures rather than with forceps, particularly in diabetics.

CUTTING MUSCLES DURING AN AMPUTATION
Muscles always contract after you have cut them. So cut them transversely c.8cm distal to the site of bone section (35-8C). Leave them a little longer if you are using delayed primary closure, because they will shrink.
Use a long sharp amputation knife or kitchen knife to cut the muscles straight down to the bone. *Don't use a scalpel* which makes many small cuts and leaves shreds of injured muscle.
Make sure you have released the tourniquet when checking the muscles.

If the muscles look unhealthy when you cut them, abandon the operation at that site, and amputate more proximally. Healthy muscle is a nice bright red, and has a good capillary ooze. Ischaemic muscle is a dark bluish red, bleeds little or not at all, and contracts feebly.

If the cut ends of the muscles bleed furiously, apply packs for 5mins and a tourniquet. If the amputation is very high, you may have to expose the main artery higher up.

CONTROLLING BLEEDING DURING AN AMPUTATION
Early in the operation, find the major arteries and veins. Tie them separately with double transfixion ligatures (3.2). Then cut the vessels between these ligatures. Later, after you have released the tourniquet and removed the limb, tie the remaining smaller vessels. *Don't use diathermy.*

CAUTION! Don't rely on digital pressure over the main vessels to control bleeding.
Bleeding and muscular contraction are useful signs that the muscle is not ischaemic: if these do not happen on cutting muscle, amputate more proximally. *Don't use bone wax.*

CAUTION!
(1) If you don't use a tourniquet, try to find and tie the major vessels *before* you cut them.
(2) *Don't use a clamp:* if it slips, there will be massive bleeding.
(3) Careful haemostasis of the stump is essential. If a clot forms, it is easily infected.

CUTTING NERVES DURING AN AMPUTATION
Don't crush, ligate or inject nerves: a painful neuroma will result, especially in the fingers. Instead, very gently pull each nerve into the wound, cut it cleanly with a knife, then let it retract above the amputation site. The sciatic nerve is accompanied by an artery which may bleed profusely, so tie the artery off carefully, separately from the nerve.

SAWING BONES DURING AN AMPUTATION
Reflect the myoplastic flap proximally for 1-2cm on a limb to expose bare bone. For large bones, use a sharp saw with well-set teeth, or a Gigli wire saw (35-7). Steady it and draw it across the bone a few times to start with. When it has made a good groove in the bone, saw steadily, cooling the bone with water.

Ask an assistant to hold the limb to steady it, and maintain a steady smooth movement to prevent the saw locking in the bone and splitting it. Finally, remove any spikes with bone forceps, and bevel any protruding edges with a coarse rasp.

N.B. Use a bone cutter for phalanges.

CAUTION!
(1) *Only reflect the periosteum proximally a very short distance* (35-8D), because the bone under it will die, and a ring sequestrum will form.
(2) *Don't damage the surrounding muscle with the saw*. Retract the muscle well out of the way with a towel wrapped round the limb (35-9), then saw.
(3) Bone dust from the saw acts as a foreign body, so wash it away.

COVERING THE STUMP WHILE YOU SAW

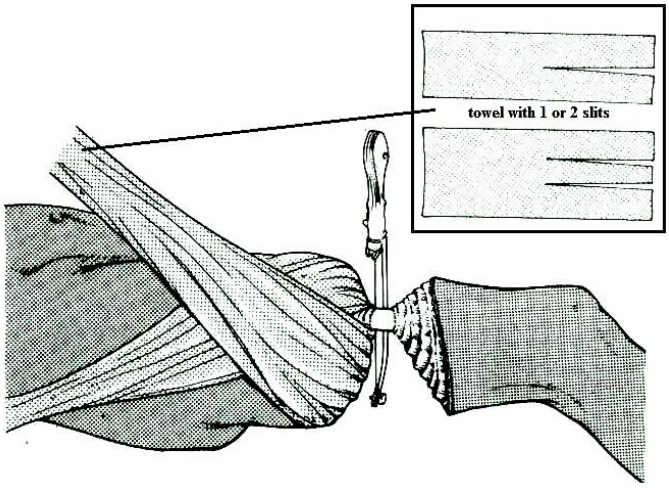


Fig. 35-9 COVER THE STUMP WHILE YOU SAW.
The towel with 2 slits in it is for the forearm and lower leg. The central flap goes between the bones.

DEALING WITH FAT DURING AN AMPUTATION

If the limb is very fat, cautiously remove as much subcutaneous fat as is necessary. *Don't remove too much, especially near the edges of the flap*, or it may necrose. Learn to design flaps so that they come together accurately without dog ears; if they do form, leave them, they will soon disappear. *Don't excise them,* otherwise you may end up with a wound that is too tight to close!

CLOSING THE WOUND AFTER AN AMPUTATION

Release the tourniquet, if present, and control all bleeding before you start closing the flaps. Make sure haemostasis is meticulous. *Don't use diathermy.* Check that the flaps meet without tension. *If they don't, cut more bone off.*

Suture the cut ends of the fascia securely together over the cut end of the bone, so that the muscle cushions it, and is well able to move over the stump. Cut the muscle long but *don't leave so much muscle that the stump becomes bulbous.*

Suture the skin and deep fascia separately using interrupted monofilament 3/0 sutures without leaving gaping areas between them, and without tying them tight. Dress the stump firmly, *but not too tightly.* If there is much oozing, insert a closed drain for 24-48h.

Avoid using an open drain, as you risk introducing infection this way. *This may be disastrous in ischaemic tissues.*

Elevate the arm or hand. Plaster of Paris over a leg stump will make it more comfortable and its weight will tend to prevent hip flexion contracture.

Change the wound dressings only if they are smelly or soaked. Remove sutures after 7 days for the hand and arm, and 14 days for the leg.

Plan delayed primary closure on the 5th post-operative day. You may have to shorten the bone so you can close the flaps without any tension.

POST-OPERATIVE CARE AFTER AN AMPUTATION

Elevate the limb after amputation. Make sure the limb or finger is exercised from the 1st day. Provide adequate analgesia. Mobilize the patient early; if you can fit a temporary prosthesis before a definitive one to allow exercises, do so. You will thereby avoid the development of pressure sores (34.16).

As soon as a lower limb stump is healing well, bandage it. For the leg, suture two 15cm crepe bandages end-to-end. For the arm, use one 10cm bandage. Roll the bandage tightly, then wind it round the stump. Apply more tension to the end of the stump than to its base, or it will become bulbous. *Don't use adhesive strapping on the skin*, or you may tear the skin off the stump.

Prepare the leg stump for a prosthesis; use an elastic bandage 8-10cm wide for a below-knee, and 10-15cm wide for an above-knee stump. Wrap the bandage from distal to proximal (knee or groin) with more pressure distally than proximally (35-10A,B). Overlap the layers by ½ the width of the bandage at each turn, keeping it smooth and free of wrinkles. Even if there is a small wound on the stump, start the bandaging, and teach the patient how to use the stump.

This will hasten the change of the stump from a bulky cylinder to a narrow cone, and exercises will strengthen the remaining muscles. After a lower leg amputation, *e.g.* learning to kick a large rubber ball about is very therapeutic. *Don't allow a flexion contracture of the knee or hip to develop.*

If there is already a tendency to flexion, keep the knee in a backslab or cast until full mobilization.

BANDAGING A STUMP

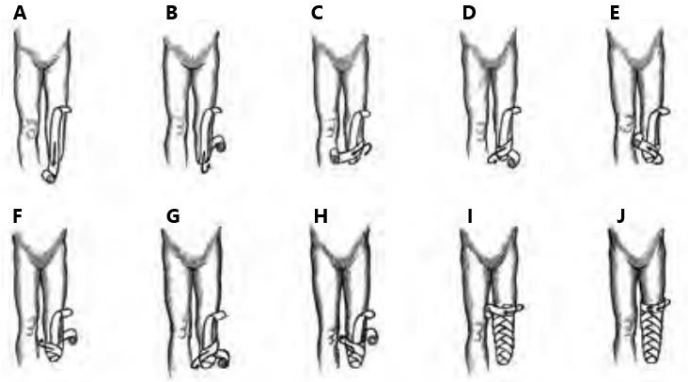


Fig. 35-10 STUMP BANDAGING.
A,B, wrap the bandage from stump to knee (for a BK amputation). C,D, then pass around the tip more tightly than higher up. E-J, make sure the bandage is smooth and causes no wrinkles.
After Giannou C, Baldan M, Molde, Á. War Surgery, ICRC Geneva 2013.

(g) Difficulties with amputations

If the stump bleeds some time after the operation (reactionary haemorrhage), return to theatre, explore the wound, tie the vessels, leave the wound open and close it later when it is clean.

If the stump bleeds some days later (secondary haemorrhage), this is likely to be serious. Apply a tourniquet. Explore the wound to find the bleeding point(s). If you cannot find them, wash the wound with hydrogen peroxide. In desperation, pack the wound with dry gauze, and remove it 48h later.

If the stump becomes infected, open the wound in theatre, irrigate it and debride dead tissue. You may still be able to save the situation if there is no further ischaemia. *Always consider delayed primary suture if there is an increased risk of infection.*

If a persistent sinus develops in the stump, explore it; you may find a piece of necrotic tendon, or an area of osteomyelitis. Another possibility is a stitch sinus. If the offending suture might be securing a vessel, *don't remove it until you have tied the vessel higher up*. Explore the stump, remove all dead and dying tissue, and pack it ready for secondary closure.

If the flaps break down, you probably cut them too short or closed them too tight. Wait until granulation tissue is clean and ready and then apply a skin graft.

The final quality of the skin over the stump will be worse than it would have been if the flaps had survived, and it may break down later. Alternatively, you may have to amputate more proximally.

If a patch of gangrene forms in a flap, be careful, it may hide a larger area of necrosis underneath. Return to theatre. Debride any necrotic tissues; you may need to amputate again more proximally, especially if the limb is ischaemic.

If there is spreading sepsis or gas gangrene, amputate more proximally immediately, through the shoulder or hip if need be, and leave the wound open.

If a prosthesis cannot be fitted, you have probably designed the stump wrong. The reasons include:
(1) bone adherent to the scar,
(2) a spicule of bone sticking out through the skin,
(3) a flexion contracture in a below-knee or above-knee amputation,
(4) too short a stump.
Get advice from your rehab technician as to what is the best way forward.

If the stump is painful:
(1) you may have damaged the nerves or left them too long, allowing a neuroma to form and stick to the scar,
(2) the bone may be too long in relation to the flap,
(3) there is a haematoma or infection in the wound, or
(4) the tissues are ischaemic.

35.4 Arm and hand amputation

(a) Mid-humeral amputation (GRADE 2.5)

If you can provide a prosthesis, *don't amputate through the lower 4cm of the humerus*, because it will be difficult to fit. Remember that the brachial artery lies quite superficially, and is overlapped medially by the biceps.

PREPARATION
Abduct the arm to about 80° on an arm board. Place a block under the arm just proximal to the amputation site. Apply a tourniquet as high as you can. Note the time.

Start proximally at the site of bone section, and mark out equal anterior and posterior skin flaps. Make the length of each flap ¾ of the diameter of the arm at the site of section (35-11). Find, doubly ligate, and cut the brachial artery and vein just above the site of section.

Find, gently pull and cut the radial, medial and ulnar nerves so that their ends retract well above the stump. Cut the anterior muscles 1·5cm distal to the site of section.

Cut the *triceps* 4cm distal to the site of section or free its insertion from the olecranon. Preserve the *triceps* fascia and muscle as a long flap. Retract the periosteum 1-2cm to expose clean bone and saw it cleanly. Rasp the end of the humerus smooth.

Bevel the *triceps* to make a thin flap, reflect it anteriorly over the end of the humerus, and suture it to the anterior muscle and fascia. Release the tourniquet, control bleeding and close the stump (35.3).

(b) Supracondylar amputation

Make equal fish mouth flaps proximal to the site of bone section. Find, clamp, tie, and cut the brachial artery and vein just proximal to the site of section. Cut the median, ulnar, and radial nerves clean at a higher level so their ends retract well above the stump.

Cut the muscles in the anterior compartment of the arm 1.5cm distal to the site of section. Free the insertion of the *triceps* tendon from the olecranon. Preserve the *triceps* fascia and muscle as a long flap.

If there is any hope of an elbow prosthesis, reflect this flap proximally and cut the periosteum all round the humerus at least 4cm above the elbow joint to allow room for the elbow mechanisms of the prosthesis.

If there is no hope of an elbow prosthesis, leave as much bone as you can. Saw across the humerus at the level you choose, and rasp its end smooth. Trim the *triceps* tendon to make a long flap, carry it across the end of the bone, and suture it to the fascia over the anterior muscles.

ABOVE-ELBOW AMPUTATION

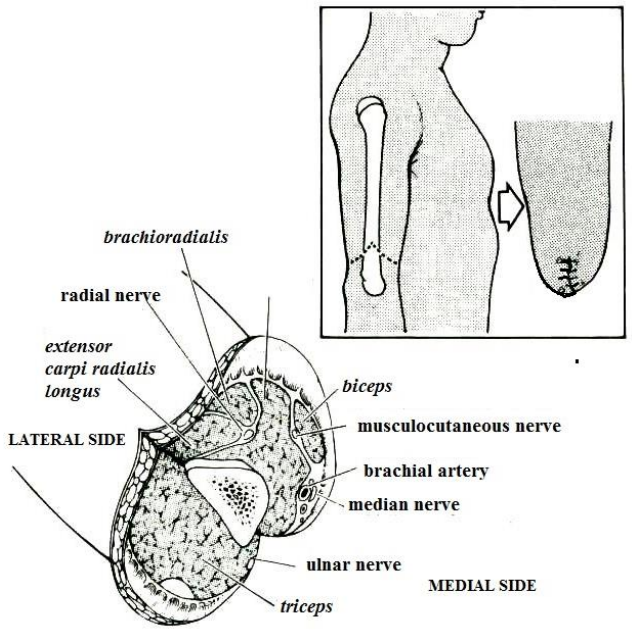


Fig. 35-11 SUPRACONDYLAR AMPUTATION.
If you leave a patient with a reasonable length of humerus, it can be used to hold things by gripping them against the chest.
After Rob and Smith with the kind permission of Graham Stack.

(c) Elbow disarticulation (GRADE 2.5)

Make equal anterior and posterior skin flaps. Start at the level of the epicondyles and curve the posterior flap 2·5cm distal to the tip of the olecranon. Bring the anterior flap just distal to the insertion of the *biceps* tendon. Reflect the flaps to the level of the epicondyles.

Start on the medial side. Find and divide the bicipital aponeurosis. Free the origin of the flexor muscles from the medial epicondyle and reflect it distally to expose the neurovascular bundle on the medial side of the *biceps* tendon. Tie and cut the brachial artery just above the joint. Gently pull the median nerve and cut it proximally. Find the ulnar nerve in its groove behind the medial epicondyle and cut it proximally in the same way. Free the *biceps* tendon from the radius, and the *brachialis* tendon from the coronoid process of the ulna.

Find the radial nerve in the groove between *brachialis* and *brachioradialis*, pull it, and cut it proximally. On the lateral side of the elbow, cut the extensor muscles 6·5cm distal to the joint, and reflect their origin proximally.

Cut the *triceps* tendon near the tip of the olecranon. Cut the capsule on the front of the joint, complete the disarticulation, and remove the forearm. Leave the articular surface of the humerus intact. Reflect the *triceps* tendon anteriorly and suture it to the tendons of the *brachialis* and *biceps*.

Make a thin flap from the extensor muscles, reflect it medially and suture it to the remains of the flexor muscles on the medial epicondyle.

MID-FOREARM AMPUTATION

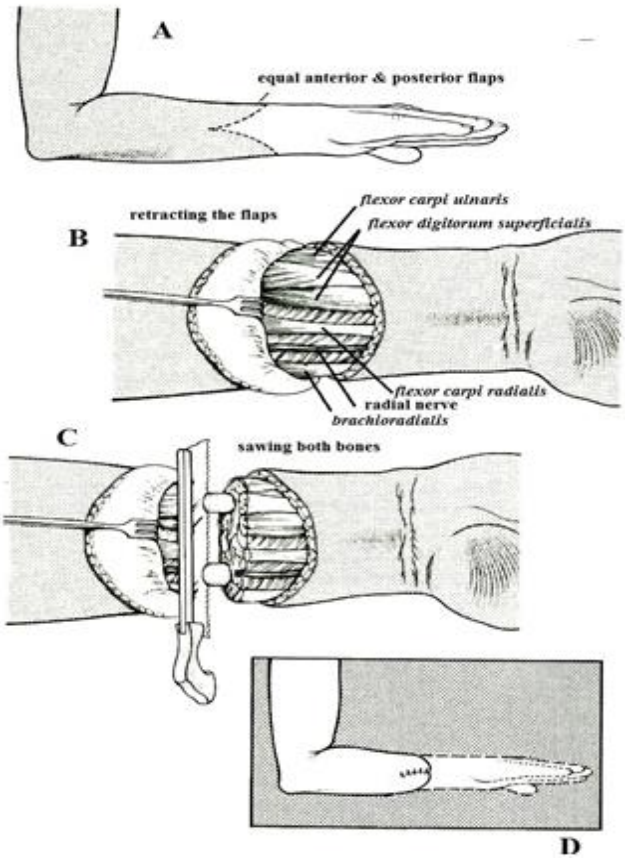


Fig. 35-12 FOREARM AMPUTATION.
Preserve as much length as you can. An elbow with even a short length of forearm is better than none. A, use equal flaps. B, reflect the flaps with the deep fascia. C, having divided the nerves and muscles, peel off the periosteum 1-2cm off the radius and ulna, and saw them cleanly through. D, final view.
After Rob C, Smith R, with the kind permission of Graham Stack.

Suture the muscle mass to cover the bony prominences and exposed tendons at the end of the humerus. Put sutures through the periosteum when necessary. Release the tourniquet, control bleeding and close the stump as in 35.3.

(d) Below-elbow and distal-forearm amputations (GRADE 2.5)

Abduct the arm on an arm-board or side-table, and place it supine. *If you cut the flaps with the arm prone, they will later be twisted.* Try to preserve as much length as possible.
If there is enough good skin, make equal anterior and posterior flaps (35-12A), as long as ½ the diameter of the forearm at the amputation site. If skin is scarce, make the best flaps you can.
Reflect the skin flaps with the deep fascia to the site of section (35-12B). Clamp, tie and cut the radial and ulnar arteries just above this site.
The radial and ulnar nerves run on the outside of their arteries, and the median nerve under *flexor digitorum profundus*; pull these nerves down gently, and cut them proximally.
Cut the muscles transversely distal to the site of section, so that they retract above it. Trim away all excess muscle. Saw the radius and ulna (35-12C) and smooth their cut edges. Suture the muscles closed over the bony stump. Release the tourniquet, control bleeding and close the stump (35.3).
If possible, amputate through the metacarpus or wrist, rather than higher up. Ischaemia is an exception. The circulation in the distal forearm is easily compromised, so if the arm is ischaemic, an amputation higher up the forearm may be better than one lower down.
If you have to amputate through the wrist, it may later be possible to make an 'alligator mouth' out of the 2 forearm bones (Krukenberg's operation), so that there is something to grip with.
Antero-posterior flaps are better than lateral ones, because the scar cannot retract between the bones.

(e) Wrist disarticulation (GRADE 3.1)

Make a long palmar and a short dorsal flap. Start the incision 1·5cm distal to the radial styloid, extend it distally towards the base of the first metacarpal.

Carry it across the palm, and then proximally to end 1·5cm distal to the ulnar styloid (35-13A,B). Make a short dorsal flap by joining the two ends of the palmar incision over the dorsum of the hand. Bring the dorsal flap distally level with the base of the middle metacarpal.

If skin is scarce, vary the design of the flaps. Reflect the flaps proximally with the underlying fascia to the wrist joint. Clamp, tie and cut the radial and ulnar arteries just proximal to the joint. Extend the incision proximally between *pronator teres* and *brachioradialis*, so that you can divide the median, ulnar, and radial nerves proximally. (If a neuroma forms here, it will be far from the scar.)

WRIST DISARTICULATION

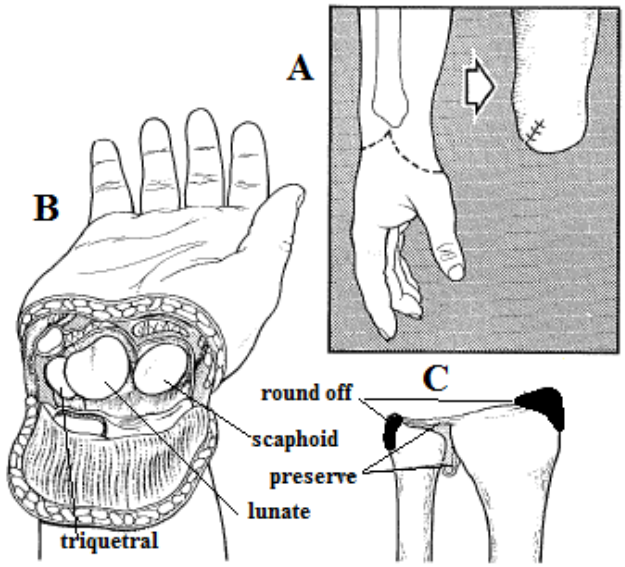


Fig. 35-13 DISARTICULATING THE WRIST.
A, make a long palmar and short dorsal flap. B, capsule of the wrist divided. C, round off the radial and distal styloids, and preserve the distal radio-ulnar joint and the triangular ligament.
After Campbell WD, Edmonson AS, Crenshaw AH (eds). Operative Orthopaedics. CV Mosby 6th ed 1980 with kind permission.

Cut all tendons just proximal to the wrist and let them retract into the forearm. Cut round the capsule of the wrist joint and remove the hand. Saw or nibble off the radial and ulnar styloids. Rasp the raw ends of the bones smooth and round. Release the tourniquet, control bleeding and close the stump (35.3).

CAUTION!
Don't injure the radio-ulnar joint or its triangular ligament. Damage to these will make rotation of the forearm difficult, and the joint will be painful.

(f) Transcarpal amputation (GRADE 3.1)

At this level, supination and pronation of the forearm, as well as flexion and extension of the wrist, are preserved and will improve overall function. Make a long palmar flap and a dorsal flap half as long. Reflect the flaps proximally to the site of bone section, and expose the soft tissues under them. Pull the finger flexor and extensor tendons distally, cut them, and allow them to retract into the forearm. Find the 4 wrist flexors and extensors (*flexor* and *extensor carpi radialis* and *ulnaris)*, free their bony insertions and reflect them proximally to the site of bone section. Find the median and ulnar nerves and the fine filaments of the radial nerve. Pull them distally and cut them well proximal to the site of section. Clamp, tie and cut the radial and ulnar arteries proximal to the site of section. Cut the remaining soft tissues down to bone. Saw across the carpal bones, and rasp all rough edges smooth. Anchor the tendons of the wrist flexors and extensors to the remaining carpal bones in line with their normal insertions to preserve wrist function. Release the tourniquet, control bleeding and close the stump (35.3).

(g) Finger amputations in general

Don't make the mistake of not amputating early enough or often enough. A stiff, painful, useless finger is often worse than no finger. If elaborate procedures are done to save it, not only is it likely to become stiff, but the neighbouring normal fingers are likely to become stiff too. *However, leave as much length in the thumb as possible*, because length here is more important than motion.

Most patients prefer a shorter finger covered with good skin than a longer one covered with poorer skin. Therefore, ask the patient if he uses his fingers for special skills. Ask how long he would prefer you leave the stump. *It is not easy to decide on the best.* Many patients (particularly labourers and even some surgeons) hardly miss an amputated finger, for example.

A flap from the volar surface of the finger is thus usually better than a graft. But, if making a flap means sacrificing too much length, a graft may be necessary. If possible, use full-thickness skin, although a split skin graft does sometimes hypertrophy and stand up to pressure remarkably. The sides and back of a finger are less important, so that a split skin graft is good enough here.

FINGER AMPUTATIONS

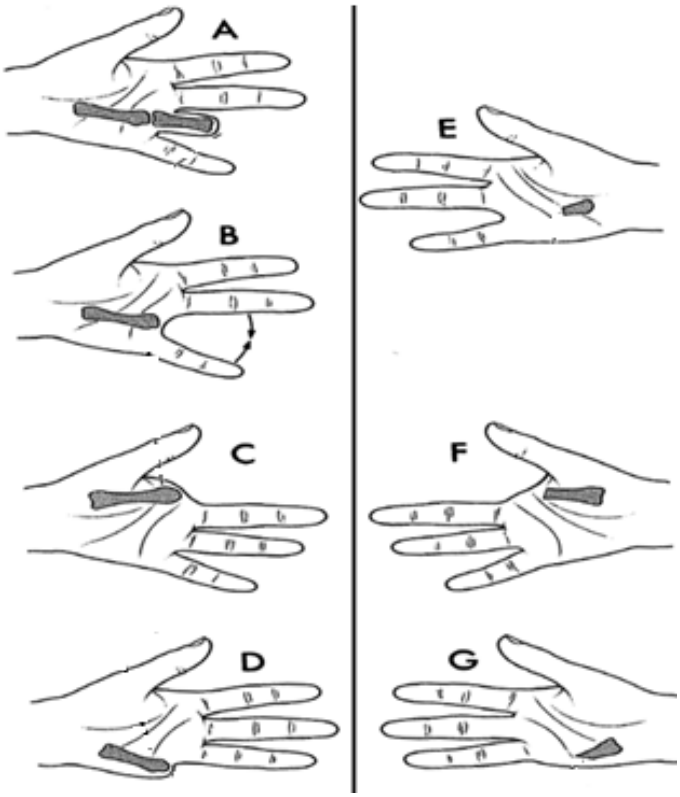


Fig. 35-14 FINGER AMPUTATIONS.
The amputations A-D are easier, uglier, and stronger than E-G. Amputating through a joint is easier than cutting through a metacarpal. A, phalangeal disarticulation, which keeps adjacent fingers apart. B,C,D, mcp disarticulations, where the mc head remains. E,F,G, mc shaft amputation removing the head.
Partly after Farquharson EL. Textbook of Operative Surgery E&S Livingstone 1969 with kind permission.

When amputating through the middle phalanx, try to retain the middle of the shaft, because the *flexor digitorum superficialis* is inserted into it. If you amputate more proximally than this, the patient will have no strength in his finger, although it will help to stop things falling out of his palm. If you are in doubt as to where to amputate, choose the more distal site. You can revise the amputation later.

An amputation through the mcp joint that does not remove the metacarpal head leaves a gap through which beans, rice or money can slip. It is usually said though that this (preferably leaving also a stump of phalanx) makes a stronger hand.

FINGER AMPUTATION INCISIONS

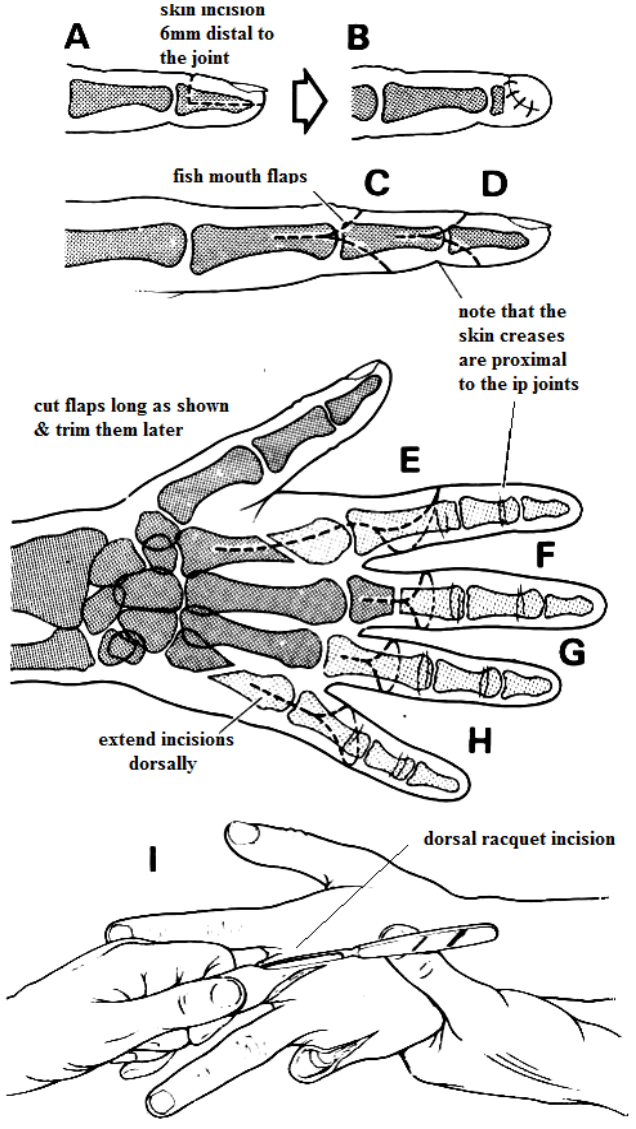


Fig. 35-15 INCISIONS FOR FINGER AMPUTATIONS.
Cut the flaps long; you can always shorten them later if necessary. A, incision preserving the base of the distal phalanx. B, final result. C,D, flaps for amputations through ip joints. E, ray metacarpal amputation of the index finger. F, proximal phalangeal amputation of the middle finger. G, mcp disartculation of the ring finger. H, ray metarcapal amputation of the little finger. I, mcp disarticulations.
Partly after Farquharson EL. Textbook of Operative Surgery E&S Livingstone 1969 with kind permission.

It is certainly an easier operation but a more elegant solution is a ray amputation through the shaft of a metacarpal below its head (35-14). This does narrow the palm, though, and reduces grip and pronation strength.

Retaining the stump of a phalanx (35-14A) further strengthens the hand by keeping the fingers apart and preventing them from deviating towards one another (35-14B). The stump will also help to stop small objects falling through the hand.

Removing an index finger causes less disability than you might expect, and even a surgeon can operate quite satisfactorily without his index finger (35-14F), provided the head of the metacarpal has been removed obliquely from the shaft. The middle finger soon learns to take over unless it is impeded by the index finger stump, which gets in the way. A finger missing from one edge of the hand (35-14F,G) is seldom a great disability, provided the head of the metacarpal is removed, so this is an elegant amputation. If great strength is not important, it is likely to be the best option.

The disadvantage of removing the metacarpal head is that it is a more difficult operation. If you are in doubt, leave the metacarpal head; you can always perform an amputation through the shaft later.

PROVIDE GOOD SKIN COVER

IF IN DOUBT, LEAVE THE METACARPAL HEAD

Use fish mouth flaps (35-15C,D). Plan them carefully in relation to the ends of the bones, and close them without tension, even if the finger has to be shorter. A shorter amputation with loose flaps is better than a longer one with tight shiny ones. Make the palmar flap a little longer than the dorsal one, because this will preserve the maximum amount of pulp tissue, which is very sensitive.

(1) PLANNED FINGER AMPUTATIONS

CAUTION! With all amputations:
(1) If in doubt, make all flaps a bit longer than you think you will need. You can always trim them later.
(2) Ask yourself if the skin of the finger you are amputating could help to close a nearby wound.
(3) *Don't suture the flexor and extensor tendons together over the bone*.
(4) Find the digital nerves and separate them from the vessels. This will be easier if you use a tourniquet. The nerves lie palmar to the vessels. Divide the nerves cleanly 1cm proximal to the volar flap. *Don't include them in the ligature of a vessel*. If possible, bury them in muscle or fat. Neuromas are sure to develop, but if you do this they will be away from the scar and the fingertip.
(5) When you amputate through a joint, trim down the condyles (where necessary), so as to avoid making a bulbous stump.

(2) INDEX FINGER MCP DISARTICULATION (GRADE 2.5)

This operation preserves the head of the metacarpal. Flex the index finger and mark out the incision on its knuckle (35-15E,16A), so that the radial flap is larger and extends nearly halfway down the shaft of the proximal phalanx. It must be long enough to meet the web of the next finger without tension.

Deepen the incision dorsally until you can see the extensor tendon, then cut it and turn it distally. Separate the extensor expansion round the base of the proximal phalanx (35-16B). Cut the collateral ligaments. Cut the flexor tendons as far proximally as you can (35-16C). Cut the rest of the soft tissues, tie the vessels, shorten the digital nerves, and remove the finger. Reduce the bulk of the scar by trimming away the ligaments around the metacarpal head (35-16D), the volar plate, the collateral ligaments, and the flexor sheath.

N.B. When you cut flaps through the webs, use a complete web on one side and no web on the other side. *Don't use 2 half-webs each side.*

INDEX FINGER MCP DISARTICULATION

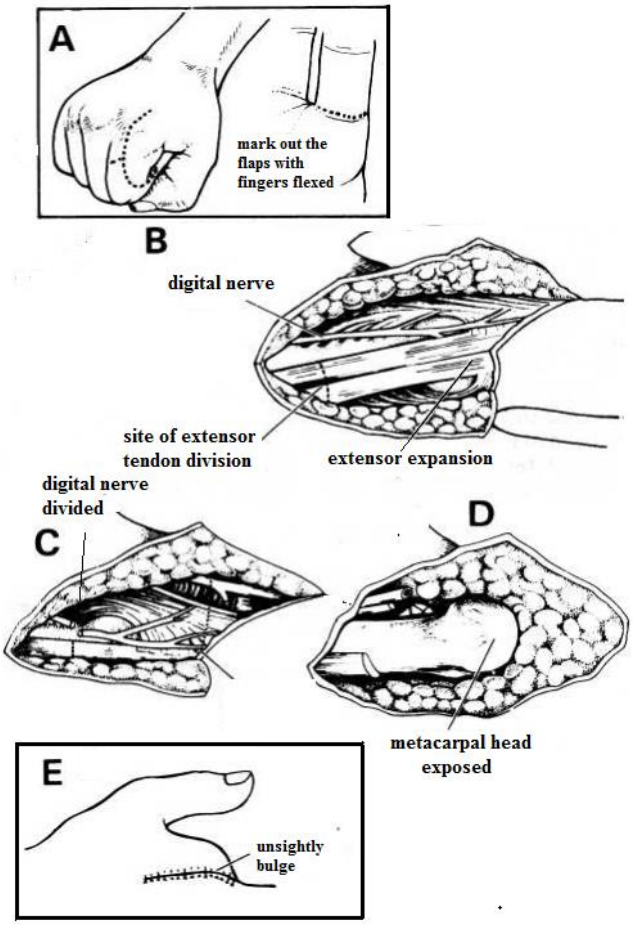


Fig. 35-16 INDEX FINGER MCP DISARTICULATION.
A, mark out the incision with the fingers flexed. B, expose the extensor expansion and split it longitudinally. C, divide the digital nerves. D, expose the metacarpal head and remove the distal part of the finger. E, final result with the metacarpal bulge.
With the kind permission of Peter London.

(3) INDEX FINGER METACARPAL RAY AMPUTATION (GRADE 3.1)

If an index finger stump will be in the way, make a dorsal racquet incision (35-17A). Keep the radial side of the flap long. You may need every millimetre. Preserve the subcutaneous tissue with the flap, and cut the extensor tendons (35-17B).

Reflect the periosteum for 1cm with an elevator, and cut the metacarpal across at the junction of its proximal and middle ⅓ (35-17C), then bevel it dorsally and radially. Separate the *interossei* and *lumbricals* from the shaft of the 2nd metacarpal. Deepen the palmar incision, and remove the flexor tendon sheath. Shorten the flexor tendons as deep in the palm as you can. Cut the vessels and nerves distal to the branches of the palmar skin. Turn the palmar flap medially, and close the skin without tension.

INDEX FINGER METACARPAL RAY AMPUTATION

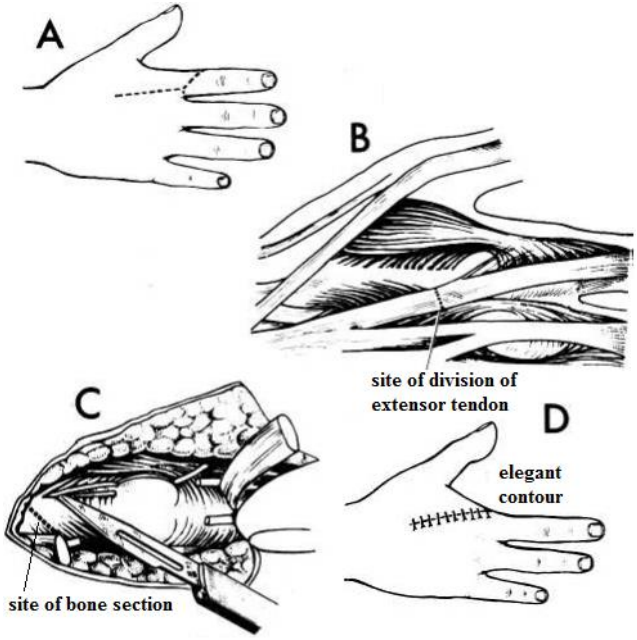


Fig. 35-17 INDEX FINGER METACARPAL RAY AMPUTATION.
A, dorsal longitudinal incision with a circular incision at mid-proximal phalanx level, leaving the skin intentionally long. B, divide the extensor tendons. C, cut through the 2nd metacarpal at an angle. D, final result with a smooth thenar bridge.
With the kind permission of Peter London.

(4) MIDDLE AND RING FINGER METACARPAL RAY AMPUTATIONS (GRADE 3.1)

Leave the base of the metacarpal, and suture the deep transverse carpal ligaments on either side of the missing metacarpal. *Failure to do this will result in a weak grip.*

CAUTION! Don't bandage the other fingers with the amputated one or they may become stiff. Encourage moving them a day or two after the amputation. Use any convenient occupational therapy, such as rolling bandages, to make sure usage of the fingers starts soon post-operatively.

DON'T SUTURE A FINGER STUMP UNDER TENSION

(5) LITTLE FINGER METACARPAL RAY AMPUTATION (GRADE 3.1)

If a little finger is stiff, and gets in the way, hindering hand function by catching on objects, make a dorsal racquet incision (35-15H); preserve the insertion of *extensor carpi ulnaris* on the base of the 5th metacarpal, and the hypothenar muscles. These provide important padding for the hand.

(6) PROXIMAL PHALANX AMPUTATION (GRADE 2.3)

Try to amputate through the neck of a proximal phalanx. *If possible, preserve even a small stump of it.* This is easier than amputating through the mcp joint. Cut appropriate flaps (35-15F).

(7) PIP FINGER DISARTICULATION (GRADE 2.3)

Do this as for a dip disarticulation below, but cut appropriate flaps (35-15C).

(8) MIDDLE PHALANX AMPUTATION (GRADE 2.3)

Proceed as for the distal phalanx below, but amputate through the mid-shaft of the middle phalanx if possible, because this retains the attachment of the *flexor digitorum superficialis* tendon to its sides, and so function at the pip joint.

(9) DIP FINGER DISARTICULATION (GRADE 2.3)

Incise the skin in the mid-lateral lines on either side of the neck of the middle phalanx. Join these 2 incisions to make a dorsal flap at the level of the joint, and a palmar flap 1cm distal to the flexor crease (35-15D). Dissect back the fibro-fatty tissue to find the digital vessels and nerves, the extensor expansion, and the flexor tendon in its sheath. If you cannot preserve tendon insertions, divide them and let them retract; *never suture the extensor to the flexor tendon over the bone stump* because of the 'quadriga effect' where the flexed amputated finger reaches the palm before the other fingers, and so weakens the grip of the hand.

Separate the nerves from the vessels, and divide the nerves proximal to the vessels. Tie the vessels without including the nerves. Complete the amputation by cutting the capsule and the collateral ligaments. Preserve the articular cartilage, which provides a 'shock pad', and close the wound.

(10) DISTAL PHALANX AMPUTATION (GRADE 2.3)

If possible, preserve the base of the distal phalanx, because of the tendons which are inserted there. Also try to preserve as much pulp as possible.

If <¼ of the nail remains, a patient will be troubled later by the irregular hooked remnant, so excise the whole nail bed.

If you have to remove some of the pulp, *don't make a flap; place a non-stick dressing and allow the wound to heal on its own.*

If you can preserve the pulp, flex the terminal joint and make a transverse incision across its dorsal surface 6mm distal to the joint (35-15A). Continue the incision as far as the sides of the phalanx, and deepen it down to the bone. Cut a long rectangular (*not pointed*) palmar flap almost to the tip of the finger. Dissect the flap off the front of the phalanx and reflect it forwards. Cut the phalanx with bone nibblers close to its base and smooth its edges. *Take care to remove bone chips and devitalized bone.*

Trim protruding condyles and the anterior part of the phalanx to make a less bulbous stump; then fold the flap and close the wound (35-15B).

35.5 Hip disarticulation

This procedure is rarely indicated. It may be necessary for advanced sepsis and malignancy; the more aggressive hindquarter amputation may be needed for malignancy.

HIP DISARTICULATION (GRADE 3.2)

Initial preparation is as for an above-knee disarticulation (35.6). Mark out a long posterior flap beforehand: make it as long as you can.

HIP DISARTICULATION

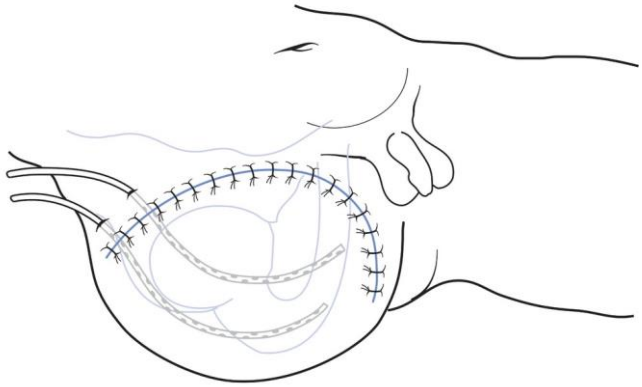


Fig. 35-18 HIP DISARTICULATION FINAL RESULT.
Create a long posterior flap. Place two suction drains. The suture should be anterior, just above the inguinal ligament.
After Greitemann B, Brückner L, Schäfer M, Baumgartner R. Amputation und Prothesenversorgung. Thieme, 4th ed. 2016.

Begin the incision 1-2 cm distal to the inguinal ligament. Expose the femoral vessels (49.3) and tie them. After resection of the anterior muscle group, open the joint capsule and disarticulate the hip by forced external rotation. Then create your dorsal flap as in a below-knee amputation (35.7). You can always trim the flap later if it is too long. Identify the sciatic nerve but *don't pull it*; cut it as far proximal as possible.

Beware of the accompanying artery, which may bleed profusely; if packing for 5mins does not control it, separate the artery off carefully, and tie it. Fill the acetabulum with the gluteal muscles. Leave closed drains and aim to remove these at 24-48h. Try to place the scar anteriorly, outside the weight-bearing zone, if the soft tissue envelope allows it (35-18).

35.6 Above-knee (thigh) and through-knee amputation

Whenever possible, try to save the knee joint. A knee disarticulation is better than an above-knee amputation, especially in children, where this preserves the epiphyses. However, before planning a knee disarticulation, consult if a suitable prosthesis is available.

The loss of the knee needs much more effort and energy in walking because the weight-bearing is in the groin instead of the stump. Make the femoral stump as long as possible, ≥10cm from the greater trochanter.

(a) Gottschalk procedure (GRADE 3.2)

Managing a very short femoral stump is very difficult as the femur stump retracts into abduction and flexion. To avoid this, detach the *adductor magnus* at its insertion, and wrap it round the femoral stump, and reattach it with non-absorbable suture to the lateral surface of the femur, using fine holes drilled through the bone, keeping the muscle under tension.

EFFECTS OF A SHORT FEMUR

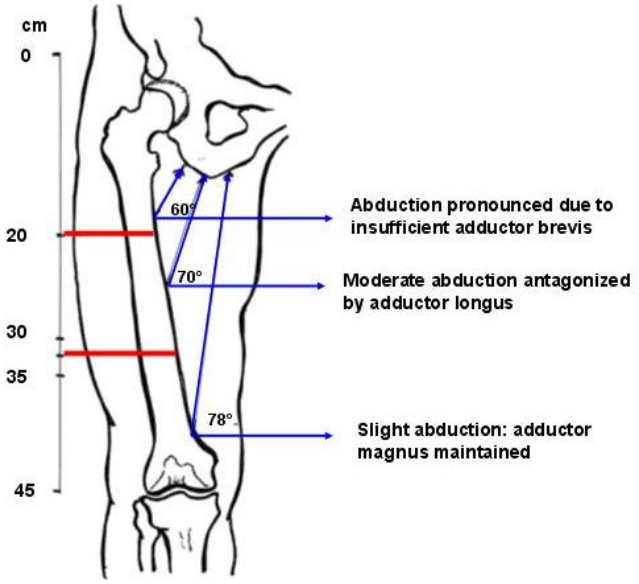


Fig. 35-19 EFFECTS OF A SHORT FEMUR.
The longer the stump, the better the result. A short stump requires much more energy to walk.
After Giannou C, Baldan M, Molde, Á. War Surgery, ICRC Geneva 2013.

Provided an above-knee amputation stump avoids the condyles of the femur, the longer it is the better, although at least 10cm length above the opposite knee is needed for fitting an artificial knee joint.

Be sure to exercise the stump immediately after the amputation, so as to strengthen:
(1) the remaining adductor muscles, and prevent the prosthesis moving outwards on walking,
(2) the extensors, because they will have to extend both the hip and the prosthesis which is to form the knee.

An amputee will also have to learn to balance with the hip instead of the foot muscles.

Study the anatomy of the leg carefully, so that you can find and tie the femoral artery under *sartorius* (35-20).

(b) Above-knee amputation (GRADE 3.1)

Administer an enema before the operation to empty the rectum. Catheterize the patient. Enclose the distal leg as far as the knee in a polythene bag, so as to isolate it from the field of operation. Preferably use spinal anaesthesia. Place a sandbag under the buttock on the side to be operated on.

Prepare the thigh with antiseptic. Raise the leg so that you can prepare the upper thigh and groin. Put a drape behind it and another one in front.

Plan to leave as much (≥25cm) of the femur from the tip of the greater trochanter (35-20B) as you can. Mark equal anterior and posterior flaps.

If there is insufficient viable skin on one side, make the contralateral flap longer rather than amputating more proximally.

Cut and reflect the flaps (35-8). Deepen the medial end of the anterior flap so as to expose the femoral artery in its canal under the *sartorius* muscle. Transfix, tie and divide the femoral artery and vein. Identify the femoral nerve, cut it clean and allow it to retract.

Begin the incision in the *quadriceps* along the line of the anterior flap, and bevel it proximally to the site of section, so as to make a muscle flap not more than 1·5cm thick.

Ask your assistant to raise the leg while you cut across and bevel the posterior muscles distal to the site of section, in the same way as the anterior ones, so they retract. Trim away any excessively bulky muscle masses. Find, clamp, and tie the profunda femoris artery on the posterior aspect of the femur. Find the sciatic nerve under the hamstring muscles, separate it from its bed without tension, cut it cleanly c.5cm proximal to the site of bone section. Tie the artery that accompanies the sciatic nerve, but *not the nerve itself* (35.5).

ABOVE-KNEE AMPUTATION

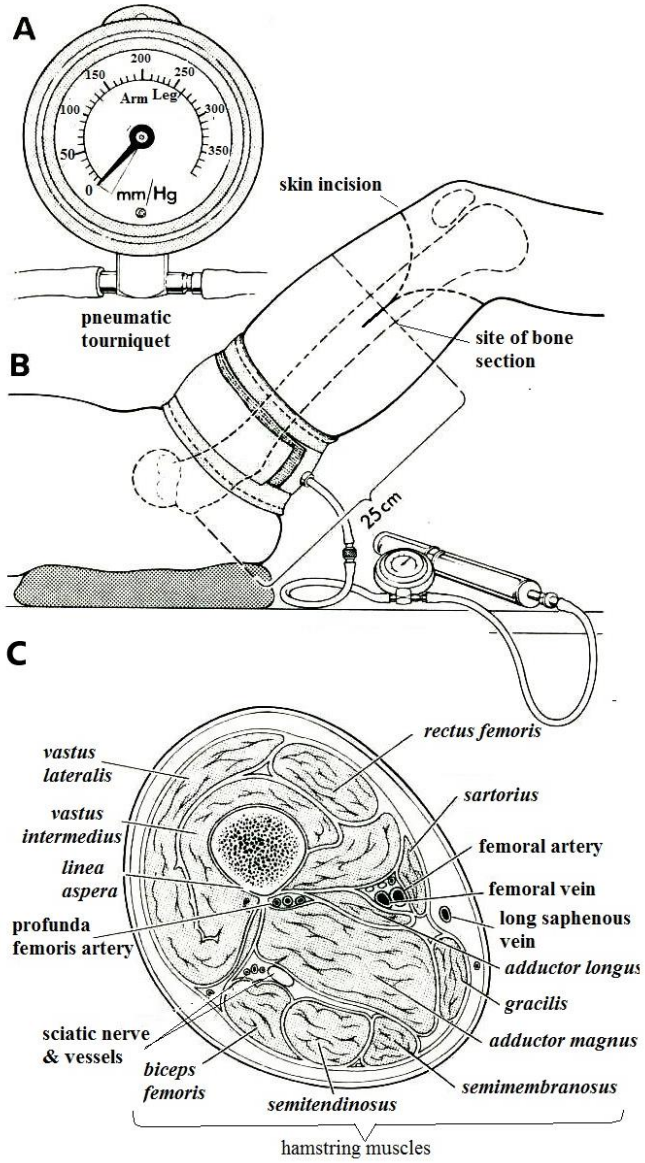


Fig. 35-20 ABOVE-KNEE AMPUTATION.
Provided the stump avoids the condyles of the femur, the longer it is the better. *Don't use a tourniquet in the presence of ischaemia. Take care not to exceed correct inflation pressures and ischaemia time* (3.4)**.**

Elevate the periosteum 0.5cm only all round the femur (35-8D) and saw it across immediately distally. Remove the leg. Rasp away and make the end of the bone smooth. Control the bleeding, suture the anterior muscle flap over the end of the bone, and suture its fascia to the posterior fascia of the thigh. Trim away any excess muscle or fascia. If you insert a drain, put it deep to this flap. Close the wound (35.3). Apply plaster over the bandage.

(c) Difficulties with above-knee amputation

If a haematoma forms within the wound, open it up as much as necessary and evacuate the haematoma, otherwise it is very likely to become infected.

If the wound becomes septic, open it up and debride any dead tissue; you may need to re-fashion a stump higher up. This time, *use delayed primary closure.*

If bone protrudes through the stump, re-fashion it making sure the muscles are long enough to cover the bone end, and insist on exercises to prevent atrophy of the *quadriceps* muscle.

If the patient has to wait a long time for a prosthesis, pad the stump well, make a cast round it and fit it snugly into a sawn-off thinned-down crutch. Keep it in place with more plaster bandages. This will facilitate walking until the permanent prosthesis is ready. *If you don't do this, the quadriceps will atrophy and the patient may never walk again.*

If you have to amputate both legs above the knees, consider the possibility of getting short 'stumpy' prostheses for both legs (60-3). This may be preferable to a wheelchair, and they will be easier to balance with than prostheses of the standard length. The centre of gravity will however be closer to the ground, and two short sticks are needed. These 'stumpy' prostheses are much easier to make, because they don't have jointed knees, and need only be sockets with simple boots on. Keep them in place with cords over the shoulder.

(d) Knee disarticulation (GRADE 2.5)

Knee disarticulation is one of the easier amputations. It preserves the distal femoral epiphysis in a child, and so allows the stump to grow. It cuts little muscle and no bone, so is quick with little bleeding, and infection is unlikely. If performed with lateral flaps, it is a good amputation for ischaemia.

Furthermore, it allows the normal weight-bearing end of the bone to carry the weight of the prosthesis. It is preferable to amputating at thigh level. Prostheses are easier to use than for above-knee amputations.

(e) Through-knee amputation (GRADE 3.1)

Consider a through-knee amputation in any frail and elderly patient unsuitable for a below-knee amputation.

Cut a long, broad anterior flap, and a shorter posterior flap (35-21A). Mark these out with the knee flexed. Start the anterior flap on the medial side 1cm proximal to the knee joint line. Extend it 10cm below this, crossing the leg c.5cm below the tibial tuberosity, and then curve it proximally to end at a point on the lateral side of the knee opposite to where you started. Start the posterior flap at this point, and extend it so it crosses the back of the leg 5cm below the popliteal flexor crease. Then curve it proximally on the medial side to meet the starting point of the anterior flap.

CAUTION! Don't fashion an anterior flap if it might have an inadequate blood supply. If so, cut lateral and medial flaps, the latter 2cm longer than the former, beginning just above the tibial tuberosity.

Get your assistant to hold the knee half-flexed. Make the anterior incision down to bone, and the posterior to deep fascia. Lift the edge of the posterior flap and

divide the medial hamstrings from the tibial tuberosity. This exposes the main trunk of the popliteal artery: doubly ligate this and divide it. Tie off the popliteal vein. Behind the artery, find the tibial nerve, draw it gently into the wound, and cut it clean (35-21D).

N.B. Divide the popliteal artery below its superior genicular branches which supply the soft tissues of the knee. These arise high in the popliteal fossa.

Reflect the anterior flap upwards with its underlying fascia to reveal the patellar tendon. Cut this at its insertion onto the tibial tuberosity. You can then lift up skin, fascia, patellar tendon, lower part of the capsule and the synovial membrane of the knee as a single flap proximally as far as the joint line.

KNEE DISARTICULATION

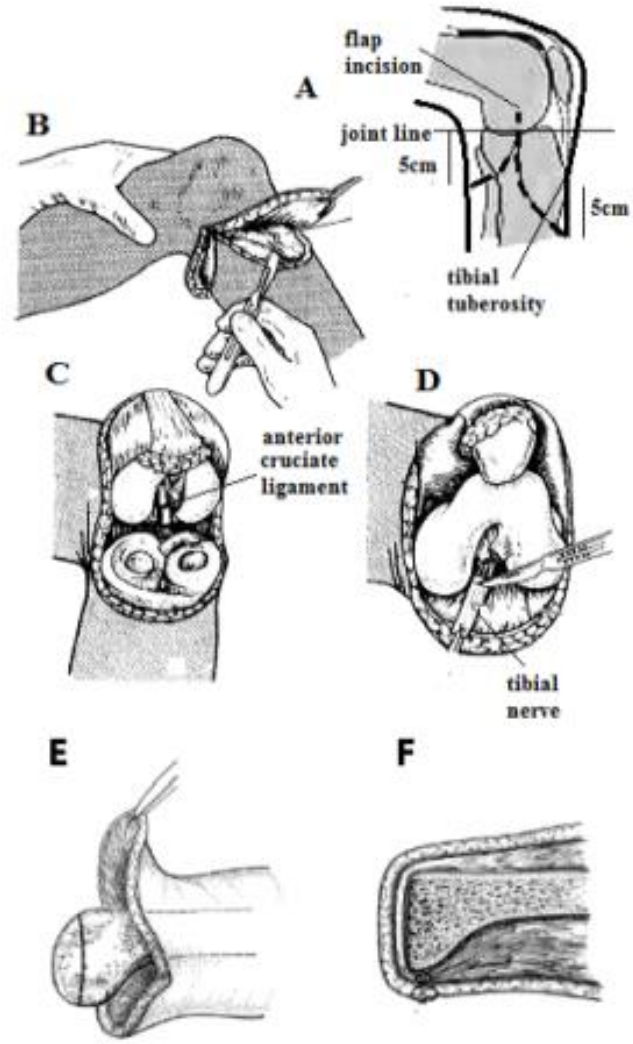


Fig. 35-21 KNEE DISARTICULATION.
A, mark out the flaps, with the knee flexed, starting 1cm above the joint line. *If the blood supply for a long anterior flap is bad*, make medial and lateral flaps. B, raise the flaps. C, cut the cruciate ligaments and the posterior joint capsule and leave the patella tendon anteriorly between the retinaculum. D, cut the tibial nerve. E, cut off the condyles. F, close the stump with the scar posteriorly.
After Campbell WD, Edmonson AS, Crenshaw AH (eds). Operative Orthopaedics. CV Mosby 6th ed 1980 with kind permission.

Now expose and divide the *biceps femoris* tendon and the iliotibial tract on the lateral aspect of the knee. Find the common peroneal nerve deep to the *biceps femoris* tendon, cut it clean proximally so it retracts above the level of the amputation. Then reflect the short posterior flap and complete division of the capsule and ligaments of the knee round the whole circumference of the joint below the menisci. Detach the heads of *gastrocnemius* from the femoral condyles, and remove the lower leg.

CAUTION!
(1) The popliteal vessels lie very close to the posterior surface of the knee joint. If you have already tied them high up, they should not be in danger.
(2) *There is no need to disturb the articular cartilage of the femur, or to remove the patella*.

Leave the patella anteriorly between the reticnaculum, where it is perfectly stabilized. (*Don't remove it.*) It improves the rotational stability of the stump for fitting a prosthesis (35-21E). Nibble or saw off the medial and lateral sides of the condyles. Remove the tourniquet (if present), control bleeding, drain and close the stump with the suture line lying posteriorly (35-21F).

(f) Gritti-Stokes amputation (GRADE 3.1)

A useful variation which creates a bony weight-bearing surface, allowing the patient to crawl, is the Gritti-Stokes amputation. Saw off the end of the femur *above* the condyles at 30º, and saw the posterior surface of the patella off flat, sloping upwards and backwards (*without detaching it from its tendon*). Then bring the patellar tendon round and fix the undersurface of the patella to the joint capsule and bony stump of the femur. *You don't need wires to do this.*

GRITTI-STOKES AMPUTATION

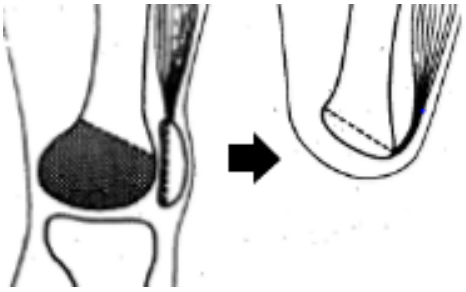


Fig.35-22 MODIFIED GRITTI-STOKES AMPUTATION.
Secure union of the patella to the femoral stump by a slanted bone cut.

35.7 Below-knee (lower leg) amputation

With a good prosthesis, an amputee can walk, run and climb almost normally, even if he is a bilateral amputee. The best length of stump for a prosthesis is 12-18cm below the tibial tuberosity.

TYPES OF PROSTHESES

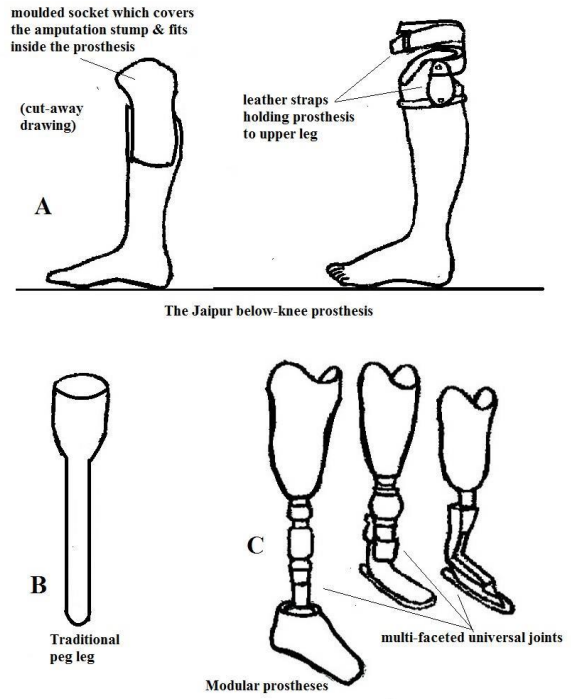


Fig. 35-23 TYPES OF PROSTHESES.
A, the Jaipur foot. B, the peg leg. C, the modular jointed prosthesis. *Kindly contributed by George Poulton.*

The Jaipur type of prosthesis (35-23A) is cheap, versatile and readily available. For the traditional type of peg leg (35-23B), a shorter 10cm stump is needed. An ultra-short lower leg stump is better than no functional knee joint. The shortest possible stump must end just below the tibial tuberosity, where the patellar tendon inserts.

CAUTION! Don't amputate below the muscle area of the calf, because the tissue here has a poor blood supply.

CAUTION! Don't amputate below the knee if there is a fixed flexion deformity of the knee >30º from full extension or *if the popliteal pulse is not palpable* as the flap will depend on the *profunda femoris* artery.

(a) Below-knee amputation (GRADE 3.2)

Initial preparation is as for an above-knee amputation (35.6). Suspend the knee over a screen bar attached to the operating table for ready access; if you cannot do this, place an inverted bowl under the lower leg. Prepare the skin right up to the groin, in case you need to convert to a through- or above-knee amputation (35.6). Make sure you have consent for this.

Mark the skin, ensuring a long posterior flap (35-24A). If you are not certain of the geometry of the flaps, cut them too long rather than too short. Measure the circumference of the leg usually 10-12cm (≥5cm) below the tibial tuberosity, and divide this length into thirds. Start the skin incision anteriorly at this point and continue transversely round each side of the tibia ⅓ of the way round; then continue down the leg the same length (usually 4cm below the anterior incision), and finally join both incisions posteriorly.

If a long posterior flap is not possible because of dubious skin vascularity, the skew flap is an alternative. It also moves the resultant scar away from the anterior crest of the tibia, on which a prosthesis socket rubs, and produces a more cylindrical stump, easier for fitting a prosthesis. In fact, the skew flap is actually a short posterolateral and a longer anteromedial flap based on a longitudinal leg axis tilted 15º laterally. Mark a fish-mouth incision 2cm lateral to the subcutaneous crest of the tibia (35-24C), with the length of the flap ¼ the circumference of the leg.

If you have to make a very short stump in the upper tibia, remove the fibula completely to avoid a tibiofibular disassociation.

BELOW-KNEE AMPUTATION

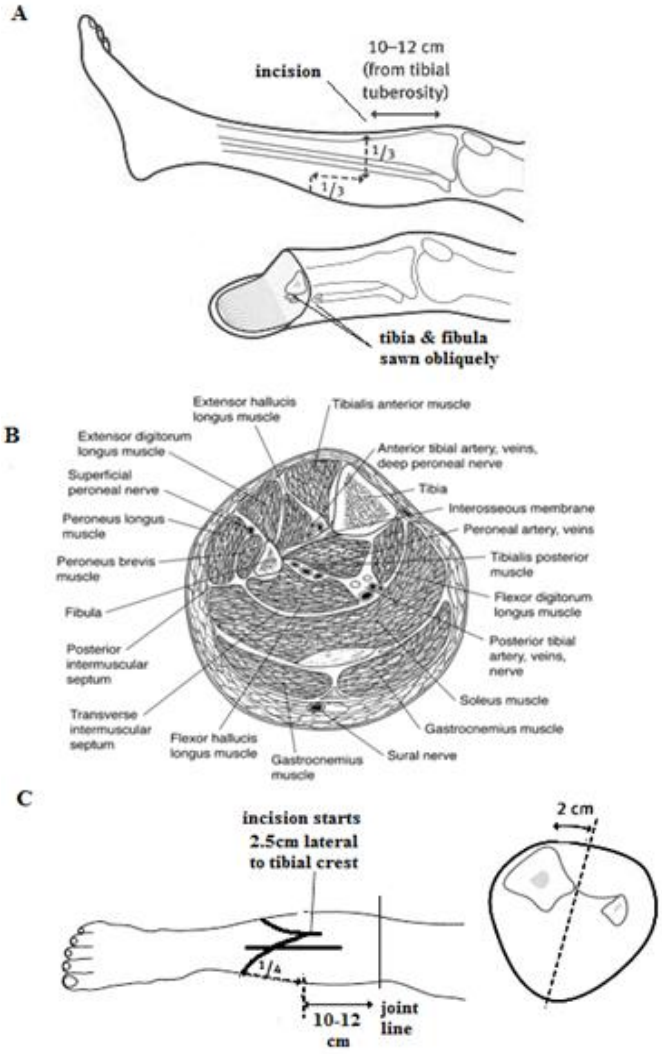


Fig. 35-24 BELOW-KNEE AMPUTATION.
A, incision using a long posterior flap. Mark the skin on either side of the tibia ⅓ of the total circumference at a point 10-12cm below the tibial tuberosity, and then mark down along the leg the same length. B, cross-section through the lower leg. C, skew flap showing apex of unequal fish-mouth incision placed 2cm lateral to the tibial crest 10-12cm below the joint line, with a flap length ¼ the circumference of the leg. The result is a suture line at 15º tilt to the leg axis.
A,C, After Marshall C, Stansby G. Amputation and rehabilitation. Surgery 2010;28(6):284-7. B, After Fraipont MJ, Adamson GJ. Chronic exertional compartment syndrome. J Am Acad Orthop Surg 2003; 11(4):268-76.

Hold the distal tibia forwards with a strong hook inside its medullary canal, and expose the posterior tibial and peroneal vessels lying under *tibialis posterior*; ligate and divide these and cut the posterior tibial nerve clean, allowing it to retract.

Then slice obliquely through the calf muscles to reach the posterior skin incision; a large sharp amputation knife is best for this, giving a clean cut. Remove most of the *soleus* muscle as it plays no part in the vascularity of the myoplastic flap. Bevel the anterior corner of the tibia at 45º and smooth the edges with a rasp.

Then turn the flap anteriorly and suture it over the tibial stump. Secure haemostasis and suture the deep fascia of the posterior flap to the tibial periosteum. It is important that there is absolutely *no tension* in this suture.

Close the stump *again without tension*. *Don't cut off 'dog ears'.*

If there would be tension at this point, divide the tibia and fibula more proximally; you may find you have to divide the vessels and nerves again proximally also.

Avoid a knee flexion contracture at all costs! The normal tendency is to flex the knee, but you must keep it extended and elevated on a pillow or Braun-Böhler frame to decrease oedema.

If you have no frame, apply a plaster back-slab in the immediate post-operative period, and later only at night. Instruct the patient to keep the knee in extension on a pillow, to lie prone in bed as often as possible, and to avoid hanging the leg over the side of the bed or over the hand-piece of the crutches.

(b) Difficulties with a below-knee amputation

If a haematoma forms within the wound, open it up as much as necessary and evacuate the haematoma, otherwise it is very likely to become infected.

If the wound becomes septic, open it up and debride any dead tissue; you may need to re-fashion the stump if there is enough length. However, it usually means making a through- or above-knee amputation. This time, *use delayed primary closure.*

If the wound is repeatedly septic, and you have excluded diabetes, HIV, and removed any foreign bodies, think of TB. *Send samples NOT in formalin.*

If bone protrudes through the stump, re-fashion it making sure the tibia is bevelled and the myoplastic flap is long enough to cover the bone end, and insist on exercises to prevent atrophy of the muscles.

If a fixed knee flexion contracture develops, make a through-knee amputation, or cut the stump even shorter and then fit a peg leg.

35.8 Ankle and foot amputation

Always try to save as much length of the leg as you can. *Don't perform an amputation in the distal ⅓ of the tibia*, as you won´t get a proper stump. If you cannot save the heel, perform a below-knee amputation. However, if you can save the heel, your patient will be able to walk short distances (*e.g.* to the toilet at night) without a prosthesis. In communities where people walk barefoot or farmers work in muddy fields and rice paddies, a heel-preserving amputation, which is an end-bearing stump, is often preferable. Amputation at a higher level will definitely involve the use of special footwear, crutches or a prosthesis.

However, the important elements are preservation of the heel and its special weight-bearing skin. So make sure the plantar flap reaches the anterior incision snugly; it should not be under tension, but also not too loose or the heel will capsize off the end of the leg stump.

TYPES OF HINDFOOT AMPUTATIONS

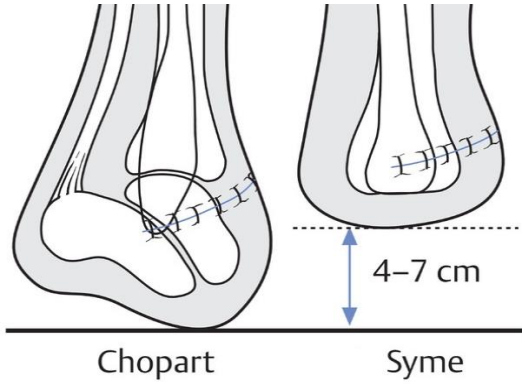


Fig. 35-25 TYPES OF HINDFOOT AMPUTATIONS.
Syme´s amputation is the most technically demanding. Chopart amputation requires close follow-up.
After Greitemann B, Brückner L, Schäfer M, Baumgartner R. Amputation und Prothesenversorgung. Thieme, 4th ed. 2016.

(a) Chopart amputation

In the Chopart amputation, you disarticulate the foot between the hindfoot (talus and calcaneus) and the midfoot (midtarsal disarticulation). You lose less length than in the Syme's operation (35-25). But *beware of developing an equinus deformity* because of the isolated pull of the Achilles tendon. Avoid this by fixing the *tibialis anterior* to the talus and the *peroneus longus* to the calcaneum. So dissect these tendons out carefully. Then put the stump in a cast for 6wks with dorsiflexion.

METHOD
Apply a tourniquet to the thigh (3.4), support the calf with a towel, and let the ankle protrude over the end of the table. Stand beyond the end of the table facing the foot. Mark out the flaps with a permanent marker. Hold the ankle at 90°.

Start the incision 2cm below the distal tip of the lateral malleolus. Bring it over the front of the ankle, level with the distal end of the tibia, to a point opposite to where you started, 2cm inferior to the tip of the medial malleolus in a curved moon shape. Then, complete the incision under the sole of the foot, well anterior to the heel pad.

Carefully dissect out the *tibialis anterior* and *peroneus longus* tendons, divide them as far distally as possible, and hold each out of the way with a stay suture.

Plantarflex the foot and disarticulate between the hindfoot bones (talus and calcaneum) and the midfoot bones (navicular and cuboid). Take care not to damage the plantar flap by retracting this out of the way.

Tie and cut the posterior tibial artery and vein just proximal to the cut distal edge of the heel flap. Divide the posterior tibial nerve adjacent to the artery. Tie the anterior tibial artery in the anterior flap similarly. Then remove the foot.

Drill holes anteriorly into the talus and calcaneum and fix the 2 tendons in place with a screw. Try to get the balance right. You may need to lengthen the Achilles tendon (32.9). Then release the tourniquet, control bleeding, close the wound and put in POP in dorsiflexion for 6wks.

FIXATION OF TENDONS IN THE CHOPART AMPUTATION

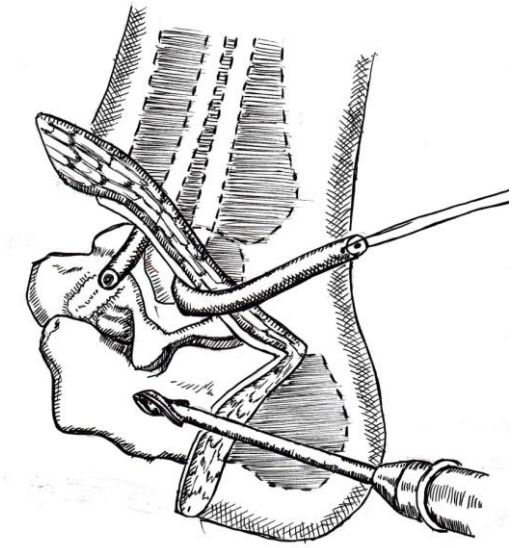


Fig. 35-26 FIXING THE TENDONS IN CHOPART'S AMPUTATION.
With the *tibialis anterior* tendon already fixed in the talus with a screw, drill a hole in the calcaneum to fix the *peroneus longus* tendon.

(b) Syme's ankle disarticulation (GRADE 3.4)

It is possible to disarticulate the ankle and adapt the stump so that it can be weight-bearing (the Syme's amputation). You need to remove all the bones of the foot and saw off the malleoli, so that the end of the tibia is flat. And you have to dissect the full-thickness heel pad very carefully subperiosteally from the calcaneum.

The patient will be able to walk about his house on it, without a prosthesis or crutches. For longer distances he can wear a simple and durable 'elephant boot'.

SYME'S FOREFOOT AMPUTATION

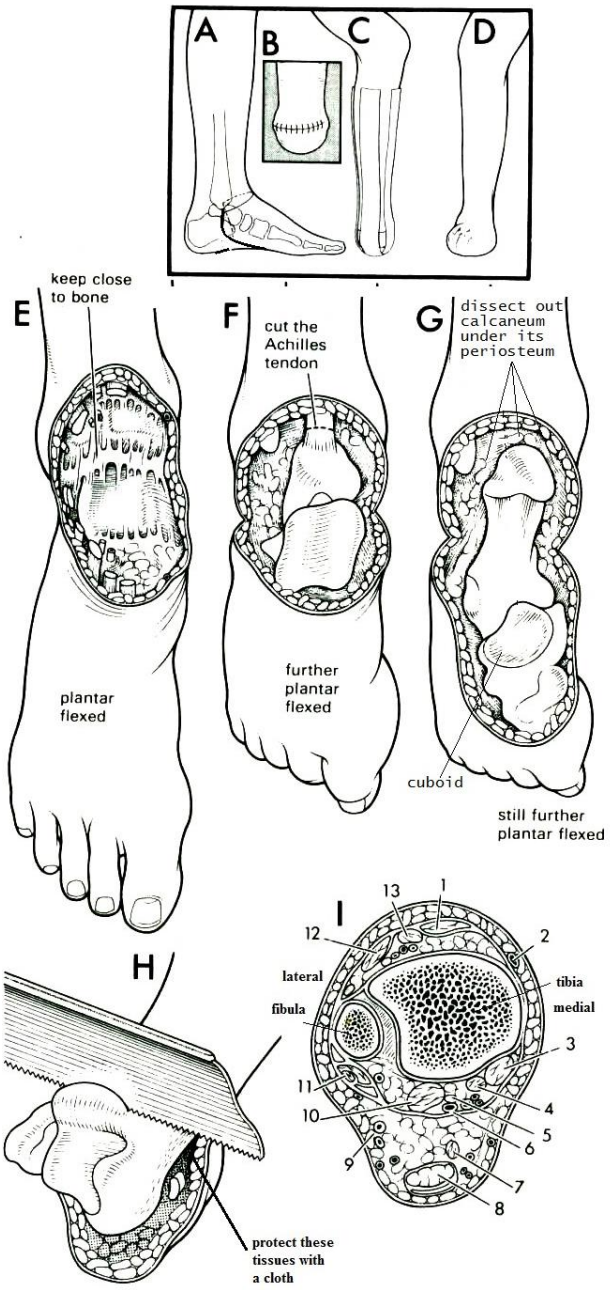


Fig. 35-27 SYME'S AMPUTATION.
A, keep the flap as long as you can (you can always reduce it later). B, completed stump immediately after suture. C, stump held in place with strapping post-operatively. D, ultimate appearance of the stump. E, expose the ankle joint and cut its ligaments. F, further plantar flex the foot and cut its Achilles tendon. G, extreme flexion allows the calcaneum to be dissected out of its surrounding tissues subperiosteally. H, saw through the lower end of the tibia, protecting the soft tissues.
I, cross-section of the ankle joint with (1) *tibialis anterior* tendon, (2) great saphenous vein, (3) *tibialis posterior* tendon, (4) *flexor digitorum longus* tendon, (5) tibial nerve, (6) posterior tibial artery and vein, (7) *plantaris* tendon, (8) Achilles tendon and its overlying bursa, (9) small saphenous vein, (10) *flexor hallucis longus* tendon, (11) *peroneus longus* and *brevis* tendons, (12) *extensor digitorum* tendon, and (13) *extensor hallucis longus* tendon.
After Campbell WD, Edmonson AS, Crenshaw AH (eds). Operative Orthopaedics. CV Mosby 6th ed 1980 with kind permission.

The distal tibial epiphysis is preserved, so it is also a good amputation for a child. It is an excellent amputation if well done, but it is also the most difficult of the amputations we describe. Nonetheless, if a Syme's amputation fails, a below-knee amputation is still possible.

CONTRAINDICATIONS
(1) Arterial disease, unless this is strictly confined to the distal part of the foot. At least one of the posterior tibial or dorsalis pedis pulses should be palpable.
(2) Infection (Syme's amputation has a special posterior flap and is not suitable for delayed primary closure).
(3) If the skin over the Achilles tendon is too thin.

METHOD
Begin as for the Chopart amputation above. Cut all structures down to the bone. Forcibly plantarflex the foot and cut all anterior structures down to the bone.

Put a knife into the ankle joint between the medial malleolus and the talus and cut the deltoid ligament. Do the same on the lateral side and cut the calcaneo-fibular ligaments.

Put a bone hook posteriorly in the talus to plantarflex the foot even more. Using a new, sharp scalpel blade, dissect the tissues away from the medial and lateral sides of the talus and calcaneum, *keeping as close to the bone as you can* (35-27E).

Then cut the calcaneum out of the heel, leaving behind the periosteum and specialized fibrofatty tissue which is essential for weight-bearing. Work at it from all sides keeping very close to bone. *This is the most difficult and the most critical part of the operation. Avoid buttonholing the skin.*

Pull the talus and calcaneum forward with a bone hook. Dissect posteriorly, and cut the posterior capsule of the ankle and the Achilles tendon (35-27F). Using a step incision, cut the Achilles tendon about 10cm proximal to the heel flap. This will prevent the heel stump displacing. *If you don't do this, the Achilles tendon tends to pull up the back of the stump*. Cut it high up, *or else you may injure the posterior tibial vessels*.

Then dissect subperiosteally round the ball of the heel, so as to free the calcaneum and reach the first incision on the sole (35-27G). As you do so, steadily dislocate the foot downwards more and more, until you reach the distal end of the plantar skin flap and finally free it from the ankle.

CAUTION!
(1) *Keep within the periosteum very close to the bone* as you dissect the calcaneum out of the heel flap, or you will cut the posterior tibial and peroneal arteries which are very close to the back of the joint capsule. If necessary, remove the calcaneum piece by piece.
(2) *Don't trim away any muscle or fat in the heel pad*, because it is needed for walking.
(3) Keep close to the bone, and *don't buttonhole the heel flap*.

Divide the vessels and nerves as above. Remove the whole foot except for the heel flap. Dissect the heel flap from the malleoli, and reflect it posteriorly. Saw off the malleoli and the articular cartilage of the tibia in a single cut (35-27H).

Make sure that the ends of the tibia and fibula are accurately horizontal, so weight-bearing is put squarely on the stump.

CAUTION! The cut surfaces of the bones must be parallel to the ground on standing.

Find the nerves and cut them well proximally. Pull the plantarflexor tendons and cut them so that they retract into the stump of the foot. Round off and smooth all the sharp corners of the tibia and fibula. Release the tourniquet, control bleeding, and close the stump. If you are amputating a child's ankle, *don't destroy the distal tibial epiphysis*. Bring the heel flap forward to cover the ends of the bones.

CAUTION!
(1) *Don't remove the dog ears, however big:* they carry an important share of the blood supply of the flap and will disappear later.
(2) Prevent the heel pad from tilting out of alignment with the tibia; *this is a real disaster!*

Apply two long U-shaped strips of strapping (35-27C). Put the 1st piece on starting below the knee posteriorly, bring it round the flap, and then anteriorly, so as to flex the flap over the stump. Apply the 2nd strip from one side to the other. Keep these strips in place for at least 3wks, and replace them as necessary. Check the strapping daily, to make sure that the heel pad is centred over the tibia. Adjust it if necessary. At 2wks, put on a well-moulded cast right round the stump. Avoid weight-bearing. At 6wks, take the mould for the prosthesis, and apply a weight-bearing cast. At 12wks get ready the definitive prosthesis or elephant boot.

(c) Lisfranc amputation (GRADE 2.4)

CONTRAINDICATIONS
Gangrene, particularly in a diabetic. Absent foot pulses.

METHOD
Make a long plantar and a short dorsal flap (35-28). This will bring the suture line dorsally. Start the dorsal incision at the site of bone section on the anteromedial aspect of the foot. Curve it distally a little to reach the midpoint of the lateral side.

Take the plantar incision distally beyond the metatarsal heads 1cm proximal to the crease of the toes. The foot is thicker medially, so make the flap slightly longer on the medial than on the lateral side.

Cut the plantar flap to include the subcutaneous fat and a thin bevelled layer of the plantar muscles. Reflect the plantar flap proximally to the site of bone section and then disarticulate the metatarsals from the midfoot bones (cuneiforms and cuboid).

LISFRANC AMPUTATION

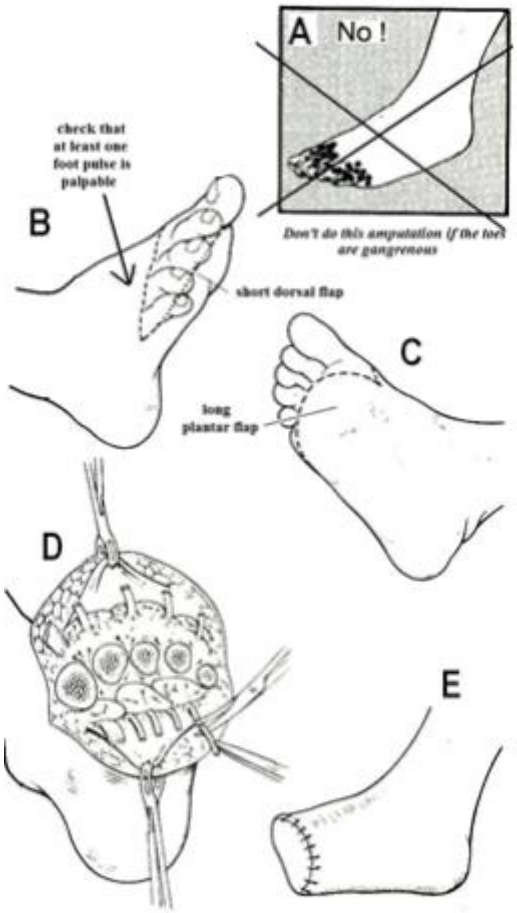


Fig. 35-28 LISFRANC (TARSO-METATARSAL) DISARTICULATION.
A, this is *not for gangrene!* B, make a short dorsal flap. C, long plantar flap. D, expose the metatarsals after division of flexor tendons. E, adapt the length of the posterior flap to give a nice completed stump.
Adapted from Rob & Smith with kind permission.

Find the nerves and cut them well proximally. Pull the plantarflexor tendons and cut them so that they retract into the stump of the foot. Release the tourniquet, control bleeding, and close the stump.

(d) Toe amputations (GRADE 2.2)

N.B. Try to preserve the hallux which gives 'lift-off' when walking.

CONTRAINDICATIONS
Absent foot pulses.

METHOD
Make a V-incision over the mtp joints on the dorsum, and an inverse V across the proximal phalanges on the plantar surface (so the scar finishes up dorsally, 35-29C).

For the hallux, use a modified racquet incision, with the 'handle' over the distal 2cm of the 1st metatarsal, lateral to the extensor tendon, in order to preserve a skin flap long enough to cover the metatarsal head (35-29A).

TOE AMPUTATIONS

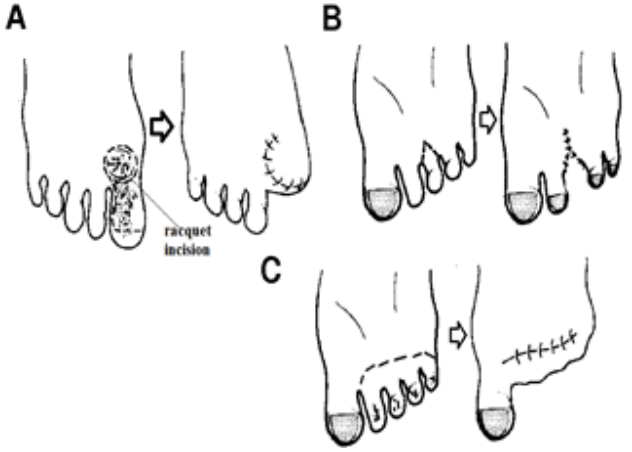


Fig. 35-29 TOE AMPUTATIONS.
A, hallux: make a special curved racquet incision for the hallux. B, 3rd toe: make a V incision proximally on the dorsum, and an inverted V distally on the plantar surface for another toe. C, lateral toes: make a flap to close the wound on the dorsal surface if you are amputating all lateral 4 toes.

Divide both flexor and extensor tendons, and suture them together around the 1st metatarsal head to secure the sesamoid bones. Carefully disarticulate the mtp joint. Release the tourniquet, control bleeding and close the stump.

35.9 Aneurysms

An aneurysm is a dilation of an artery; it can occur anywhere. The 'false' aneurysm of traumatic origin occurs when there is a laceration of the artery and blood leaks out into a confined space, clots and forms a false capsule. 'True' aneurysms can occur in large vessels (aorta or iliacs) but then require advanced vascular surgery; in smaller vessels, their treatment is not so complicated. The blood in an aneurysm does not flow smoothly, and so may clot; pieces of thrombus may detach and be carried further downstream as emboli.

Occasionally the aneurysmal sac may become infected secondarily, or it may originate from a septic embolus (the so-called 'mycotic' aneurysm). Its main danger is increase in size and rupture.

An aortic aneurysm >7cm diameter has a 20% risk of rupture within 1yr. This occasionally occurs into a vein, resulting in an arterio-venous fistula, or stomach or bowel, resulting in initially obscure, intermittent, usually minor rectal bleeding, and then later a sudden massive gastrointestinal haemorrhage.

Remember: *aneurysms pulsate!* So, if you find a swelling which pulsates, *don't incise it thinking it is an abscess!* Check, if you can, by performing an ultrasound of the swelling (38.2e).

Weakening of the arterial wall also occurs with HIV disease, sickle cell disease, syphilis, salmonellosis and *staphylococci*.

There are also rare fungal causes, and elastic tissue disorders such as Ehlers-Danlos and Marfan syndromes.

Aneurysms of the aorta or iliacs require advanced surgery, so refer the patient if possible. For smaller vessels, there are the following options:

In a limb, if the aneurysm is chronic, the collateral circulation will be adequate. Perform a Hunterian ligation: check that pulsation and flow distal to the aneurysm is present (preferably by ultrasound); then expose the artery feeding the aneurysm above and below it, and ligate it doubly on both sides. If the aneurysm sac is large or infected, it is best to open it and remove the contents.

If the aneurysm is acute, the collateral circulation will be inadequate. Excision with vein grafting is necessary. This is not as difficult as it may appear; if you are able to perform bowel anastomoses, you should be able to manage a vascular anastomosis with a fine non-absorbable running suture, with small spaces between bites.

N.B. Don't use haemostats to clamp vessels you wish to suture late: they will be irreparably damaged!

Remember:
(1) use fine instruments (even eye equipment),
(2) use rubber or cotton tape to isolate, retract or interrupt the flow in vessels,
(3) use heparin to prevent blood which is not flowing from clotting: *use a maximum of 3,000 units into the vessel in an adult*.

SAPHENOUS VEIN GRAFT (GRADE 3.3)

Make a 10cm longitudinal groin incision, and carefully dissect out an adequate length of long saphenous vein. Tie its tributaries. Ligate above and below the site you wish to divide the vein. Remove a segment of vein and reverse its direction (because of its valves); then hold its ends with bulldog clamps and fill it with heparinized saline. This will distend the vein to the correct size, and show up leaks from tributaries you have not ligated; put ties round these.

Trim the ends of the vein, and leave it clamped with heparinized saline inside till you are ready to use it, under a warm pack.

Put a gauze or rubber drain behind the artery you wish to repair. Release the clamp on the artery to flush out any clots, and reclamp it. Place the vein graft (*with the valves reversed*, and the proximal clamp removed) adjacent to the proximal arterial segment. Put in two stay sutures bringing together the corners of both artery and vein. With one of these threads, make a continuous suture of the back layer till you reach the other stay suture; knot these together on the outside of the vessel.

SAPHENOUS VEIN GRAFT

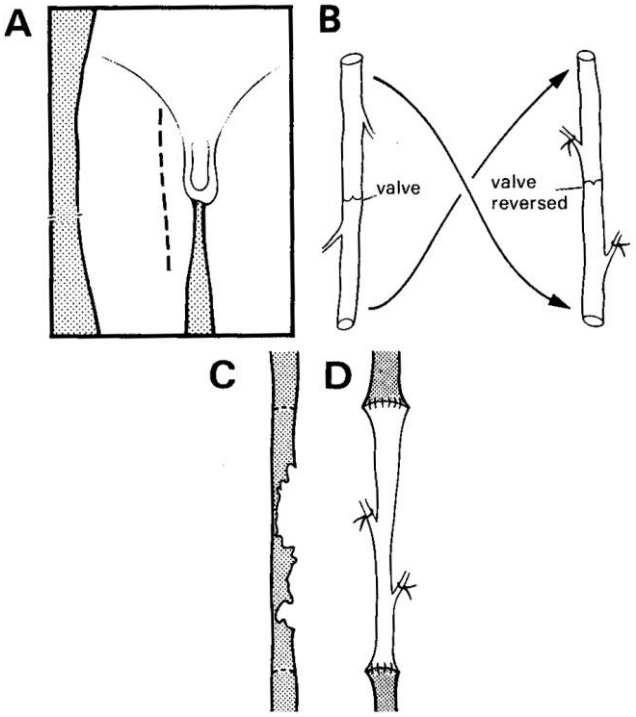


Fig. 35-23 SAPHENOUS VEIN GRAFT.
A, groin incision. B, reverse the orientation of the excised segment of vein. C, damaged arterial segement to be excised. D, vein graft sewn in place.
Kindly contributed by Peter Bewes.

Then continue on the front layer. Now put two more stay sutures bringing together the corners of the vein graft and the distal arterial segment. Complete the anastomosis as before, making sure your knots are outside the vessel. Release the clamp to let the graft fill with blood before finally closing the last suture. Introduce some heparin into the distal arterial segment and then release the distal arterial clamp. Check for leaks; apply pressure and check again. *Don't be too hasty to insert another suture as this may cause a further leak!*

Check if you can palpate a distal pulse; if so, all is well. If not, check that the proximal pulse is palpable; if it is not, release the lower anastomosis slightly with a fine artery forceps to let out clot, and introduce some heparin. Otherwise instil heparin proximally.

36 Thoracic surgery

36.1 Spontaneous (non-traumatic) pneumothorax

Most thoracic surgery is outside the scope of this manual; check elsewhere for drainage of pus in the pleura (9.1), pericardium (9.2) and lung (9.3). *Consult Volume 2 for traumatic pneumo- and haemo-thorax.*

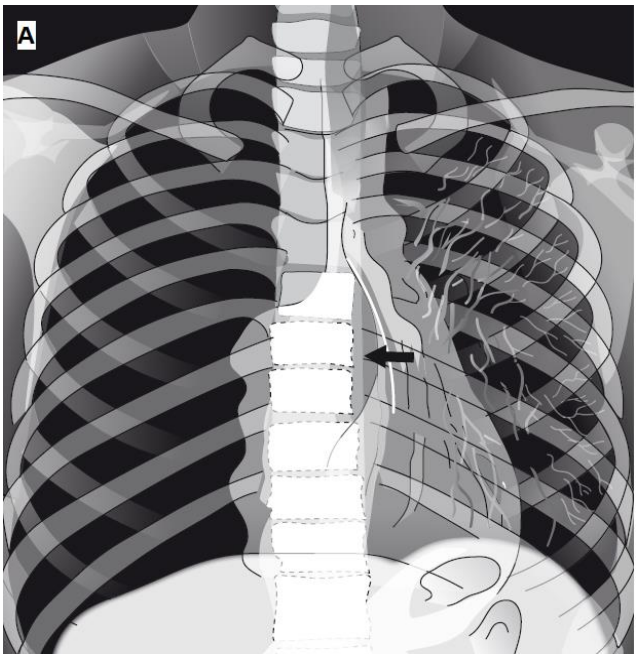
Pneumothorax describes air collecting in the pleural space. This air will compress the lungs, and if there is a communication with the air passages, the pneumothorax may continue to expand alarmingly. The mediastinum may then shift and block venous filling of the heart, producing profound shock (a tension pneumothorax).

Pneumothorax can occur spontaneously, typically in tall young adults, or secondary to lung disease, classically with TB, asthma, pneumocystis pneumonia in HIV+ve patients, anorexics or those with cystic fibrosis. Frequently a ruptured bulla on the lung surface is the cause, and this is more frequent in smokers, especially cannabis smokers.

(a) Clinical features
There may be chest pain and breathlessness: *the severity of symptoms doesn't give an indication of the size of the pneumothorax.* You will note an absence of breath sounds on the affected side, and a thorax resonant or hyper-resonant to percussion. However, signs may be subtle. Agitation may be the sole sign of hypoxia. Check the pulse and blood pressure: hypotension may develop quickly. Check for the position of the trachea manually in the supra-sternal notch: if it is shifted to the opposite side, a tension pneumothorax may be developing, *though it remains central if there are bilateral tension pneumothoraces*.

(b) Radiographs
An *erect* PA chest radiograph, best taken in expiration, shows the typical uniform black appearance of air in the pleural cavity with *the absence of lung markings*. Look for a thin line medially marking the edge of the lung. *Don't confuse this with the medial edge of the scapula!*
In adults, a visible rim >3cm from the chest wall represents a pneumothorax >50%. You occasionally need a lateral chest radiograph to show the pneumothorax. *A supine radiograph may not show a pneumothorax clearly.*

(c) Ultrasound
Place the probe either longitudinally or transversely below the clavicles in the midline, and watch for the sliding movement of the lung against the pleura, seen as a bright line below the dark rib, as a constant 'wiggle'. You can also see, dropping from this line, so-called 'comet tails' which are artefacts. These don't appear in a pneumothorax and there is no 'wiggle'.



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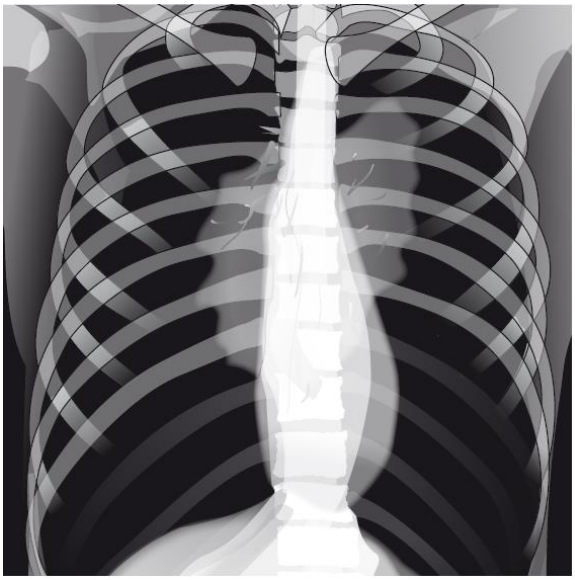

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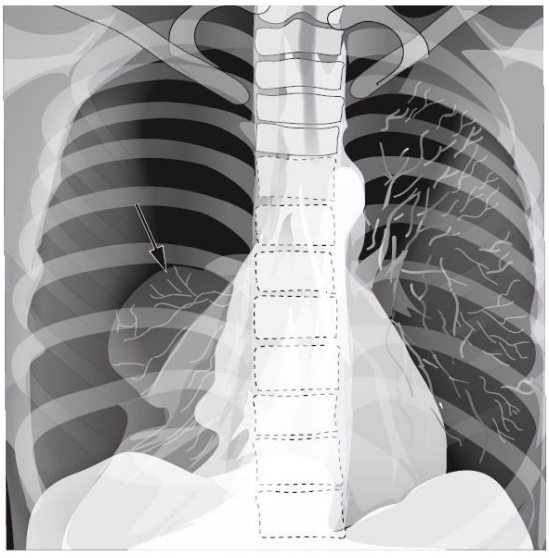

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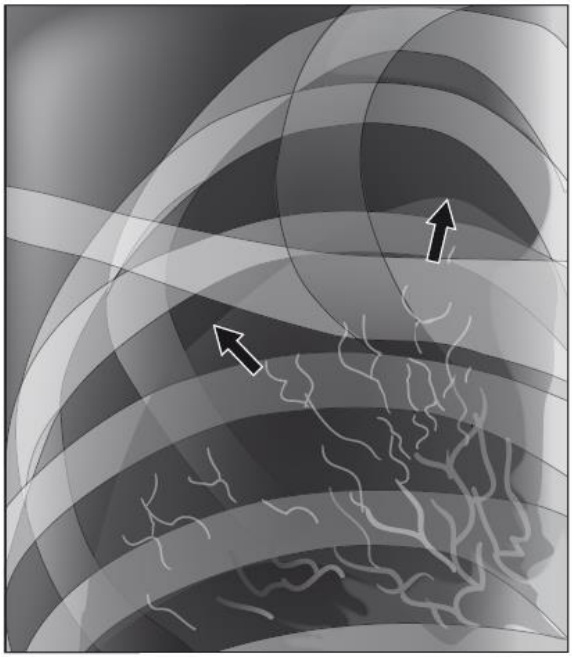


Fig. 36-1. RADIOGRAPHS SHOWING A RIGHT-SIDED PNEUMOTHORAX.
A, tension pneumothorax, with tracheal and mediastinal shift (shown by the arrow). *You should not see this image, because you should already have put in a needle to decompress the pneumothorax!* B, bilateral tension pneumothorax (no lung vessel markings visible, trachea central and a heart that has 'disappeared'). *Likewise, you should not see this image, but have dealt with it by needle decompression beforehand!* C, large pneumothorax, showing the totally collapsed lung border (shown by the arrow). D, medium-sized pneumothorax (supero-lateral section) showing the lung border >3cm from the edge of the chest wall, signifying partial lung collapse with a pneumothorax of >50%.

(d) Treatment

N.B. Don't delay! A tension pneumothorax may develop very quickly! This is a critical emergency! So much air has filled the pleural space that the mediastinum is pushed to the opposite hemithorax, thus reducing venous return to the heart. Immediately insert a large cannula or needle in the 2nd intercostal space, in the mid-clavicular line, and as soon as you can, insert a formal chest drain in the 4th intercostal space in the midaxillary line.

If there are bilateral pneumothoraces, insert a needle as for a tension pneumothorax on both sides, and as quickly as possible, insert a chest drain first on the worse side, and then on the other. Or, insert drains simultaneously if someone can assist you.

If the pneumothorax is small (the rim <2cm from the chest wall) and there is no breathlessness, observe the patient for 24h and repeat the chest radiograph. If the rim has enlarged, aspirate as below.

N.B. You should advise a patient to avoid non-pressurized air travel till the pneumothorax is completely resolved, and to avoid smoking and deep-sea diving permanently.

If it is spontaneous, associated with no fluid, and it is the first occasion it has happened, insert a wide-bore cannula in the 2nd intercostal space in the mid-clavicular line and aspirate through this, or attach it *via* a piece of giving set to an underwater seal drain. Usually 30mins is sufficient, but if you drain <2·5L air, repeat the exercise.
An alternative to using an underwater seal drain with its bottle which might spill, fall over and break, is attaching a sterile glove with 2 fingers cut off to the tube attached to the cannula. This way, air will exit but not enter.

If the pneumothorax is large, insert a cannula or needle first before inserting a formal drain to avoid a sudden rapid evacuation of the pneumothorax, which can cause problems (see below).

If the pneumothorax is associated with air in the mediastinum or pericardium, there may be an oesophageal perforation (30.7).

If it is a repeat pneumothorax, or a hydro- or haemo-thorax, or due to trauma, insert a formal chest drain.

N.B. Don't apply suction (except if you suspect a broncho-pleural fistula: see below)!

N.B. Don't clamp a chest tube unless there is sudden chest pain from rapid re-expansion of a collapsed lung causing pulmonary oedema. *In rare cases this may be fatal!*
Once there is no more air bubbling out of the chest, or >50ml draining per day, remove the drain. Get an assistant to hold the skin opening closed, ask the patient to breathe in, hold his breath, and rapidly pull out the drain.

N.B. Don't remove the drain slowly so that air can return to the pleural cavity by way of one drain hole outside the chest and one inside!
Repeat the radiograph if you have doubts about a persisting or recurrent pneumothorax.

If the pneumothorax is iatrogenic (*e.g.* caused through insertion of a central venous line), treat this initially with puncture and aspiration alone. Keep the patient under observation and repeat a chest radiograph after 24h.

If the pneumothorax persists, probably due to a broncho-pleural fistula, apply suction up to a pressure of 20cm water (=15mm Hg) to see if this makes the lung stick to the chest wall. If this fails, prepare a sterile slurry of 2-5g talc in 50ml saline and add 20ml 1% lidocaine, and inject this via the chest drain into the pleural space using a wide-bore syringe. This causes an adhesive inflammation, and is often very painful, so use copious analgesics. Continue the suction. Repeat the chest radiograph after 24h, and if there is only minimal fluid and residual air, remove the drain. If this fails, wait a week, and if the pneumothorax persists, do the same thing with 1% silver nitrate solution instead of talc.

Avoid positive pressure ventilation in anyone with a pneumothorax: it may enlarge catastrophically!

37 Terminal care & oncology

37.1 A task for every district hospital

One of the first historical tasks of medicine was to comfort the dying and relieve their suffering. Much of this is due unfortunately to preventable disease complicated by delays, lack of treatment or inappropriate treatment (and this includes HIV disease) as well as to malignant disease, which causes severe pain in about 70% of cases. Every day probably >5 million people endure such pain, and only a fraction of these have any alleviation. This usually needs only simple drugs, which are so often successful that they should be available to everyone.

The development of hospices, and their outreach into the community, is helping and respecting the dying, as well as alleviating their pain and their other distressing symptoms. Where hospices have yet to be established, district hospitals have to fill this role. Unfortunately, many of them provide no terminal care whatever: its provision is one of the indicators of 'good care'.

Any patient who receives terminal care is, by definition, going to die. It is therefore only too easy to neglect him. Your own attitude to him and that of your staff is critical. He must feel welcomed by people who are determined to help him. There is always something to be done to make his last days more bearable, even if he is dying. *Never send him back home immediately*; he has come to you for help. A long family discussion may have taken place before he came, and if you send him back home without extensive discussion and explanation, after his family have wasted much effort and expense, he will feel rejected, and so will his family, who may be induced also to reject him as a 'hopeless case'. Actively exclude any differential diagnosis that may be curable. *Don't make the mistake of assuming HIV disease unless it has been proved* (5.5), *nor forget thyrotoxicosis as a cause of weight loss*.

It is important not to leave the patient out of the discussion; otherwise you will create a barrier between him and his family which neither may be brave enough to cross. Remember the stages of grief (according to the Kübler-Ross model): denial, anger, opposition and finally acceptance.

You will have to decide whether to continue to treat him in hospital, or at home. Make this decision on:
(1) The extent of the suffering he will undergo at home from bed sores, from malignant ulcers, and from difficulty with his toilet arrangements, etc. For example, if he needs a catheter which must be changed every month, is there a health unit near him which can do this?
(2) His own wishes, and those of his family.
(3) The length of time he has to live.
(4) Financial considerations.
(5) His desire to die at home (which may be very important in certain communities): it is often much more expensive to transport a dead body than someone who is still living!
(6) People may view your hospital with scepticism if you admit too many patients just to die, as their relatives may really believe you can do something to reverse their condition.

Try to palliate the symptoms of death. You can:
(1) Always alleviate intolerable pain with drugs.
(2) Arrange radiotherapy, chemotherapy (37.4), or surgery, after you have weighed up the benefit to be gained against:
 (a) further suffering,
 (b) affordability of treatment,
 (c) availability of transport etc.

Unfortunately, 'altering the symptoms of death' can sometimes make them worse. An intolerable and burdensome indignity in one culture (a colostomy, for example) may be quite acceptable in another. So make sure that whatever you do, for cure or palliation, does not make his symptoms worse and, particularly, *does not prolong a final illness painfully*. For example, a gastrostomy (13.9) may keep a patient with carcinoma of the oesophagus alive for months but he remains unable even to swallow his own saliva, which he aspirates into the bronchial tree.

Usually, there is no further need for antihypertensive, diuretic or oral hypoglycaemic drugs in the final stages of terminal illness. Assess carefully whether you should prescribe these medications.

Tell the patient, *and* his family, about his illness. This should not be difficult, but you will have to appreciate the local culture. Usually, you will have to tell the full story to the patient *and* a responsible relative. Many patients don't really understand what malignancy is and to many, illness is a spiritual matter.

If you tell him nothing, pretending that he is going to get better, he may eventually lose all faith in you, and alas even in his family, who may be seen as also having conspired to deceive him. In contrast, many patients have thanked their doctors for telling them the truth. It gives them time to prepare for their own end. This is important in many faiths, where a priest may need to be specially called in.

Unfortunately, some patients cannot accept the whole truth immediately. So judge this carefully. How much of the truth is he really able to take at a time? Whatever you tell him, it must be true. You don't have to tell everything at once, but you must not minimize the problems by obscuring the realities.

You should always start with the truth because:
(1) The patient is going to get worse anyway, and will eventually know.
(2) The relatives will know about any falsehoods, and when their turn comes to be ill, they will not know whether to trust their doctors and especially you.
(3) The patient may have personal matters to set in order.
(4) You will save him the expense of going from doctor to doctor, vainly seeking a cure.
(5) You will relieve his family of the responsibility of knowing what to say to him.

Don't be drawn into telling a patient 'how long he has got to live' because you may be hopelessly wrong, and many patients and their relatives may take your answer literally if you give a figure or a date!

If one of the differential diagnoses is a curable condition, be sure to investigate sufficiently to exclude it. Unless you do this, you will miss diseases that could have been treated. So, *don't accept a diagnosis of malignant disease until it is confirmed*, preferably by biopsy. *You should never tell a patient he has a malignancy without proof*. If you later try to tell them the diagnosis was wrong, they will be confused and lose confidence in you.

PAIN PERCEPTION

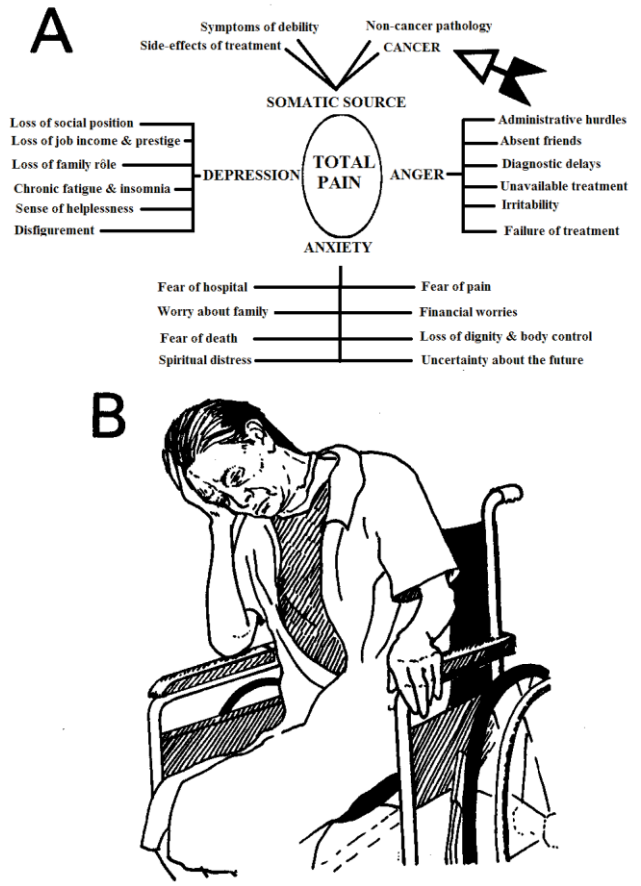


Fig. 37-1 PAIN PERCEPTION.
A, shows the factors that influence a terminal cancer patient's total perception of pain. Only one of these is the cancer itself. Try to influence as many of the other factors as you can. B, the patient herself.
After Cancer pain relief and palliative care. WHO Technical Report Series 804, 1990 Fig 5, p.21. (WHO 90746).

Many patients have indeed been palliated for supposedly malignant disease, only to be shown at post mortem to have had some treatable condition. What you may think is a hepatoma (15.11) may turn out to be a liver abscess (15.10); a rectal lesion may be an amoeboma (14.5), and not a carcinoma (12.11); 'malignant ascites' may in fact be tuberculous (16.2). Also be sure to have proof of HIV disease: *don't assume it!*

MARY (24yrs), who had led a rather tumultuous life as a teenager, and had run away from home, had lost a huge amount of weight, had diarrhoea, had a persistent tachycardia and low-grade fever, and had developed sores on her body. Because of her past, everyone assumed she had HIV disease, especially when one of her former partners died of HIV complications. However, a conscientious doctor took a good history, examined her and did one relevant blood test: she was thyrotoxic!
LESSON *Don't assume HIV disease until it is proven.*

Remember palliative surgery may be very effective in pain relief, especially in situations of obstruction: colostomy for obstructing colorectal cancer (11.6), gastrojejunostomy for stomach cancer (13.10), cholecystojejunostomy for cancer of the head of the pancreas (15.9), tracheostomy for laryngeal cancer (29.15), amputation for a very painful limb (35.3), orchidectomy (27.26) for prostatic cancer or perhaps excision of a fungating breast tumour (24.5).

37.2 Controlling cancer pain

When a patient has cancer, its physical effect is only one determinant of his 'total perceived pain'. The perception of pain is profoundly modified by his psychological state, and by spiritual, social, and financial factors. Depression, anxiety, anger, hopelessness, and a fear of impending death can all add to suffering, and worsen the pain. Taking the edge off anxiety with chlorpromazine, for example, may greatly reduce the total perceived pain, and may help with other factors, where this is possible. Although chlorpromazine is a tranquillizer, and is not an analgesic, it may be so effective in altering an anxious patient's 'total perceived pain' that no analgesic is required.

In the control of cancer pain, chlorpromazine and drugs like it are called 'adjuvants'. If an adjuvant alone fails, start the patient on WHO's three-step ladder (37-2). This consists of:
(1) A non-opioid (aspirin or paracetamol). If these fail, you can try other non-steroidal anti-inflammatory drugs (NSAIDs) than aspirin but they tend to be more expensive and *all* are gastric irritants, so add antacids or anti-H_2 blockers.
(2) A mild opioid (codeine or tramadol).
(3) A strong opioid (morphine).
All of them can be used with or without an adjuvant. Strictly speaking, the use of an 'adjuvant alone' is not one of the steps on the WHO ladder, which starts with Step 1 (a non-opioid, perhaps with an adjuvant).

Chlorpromazine alone ('step zero') may be a valuable initial step, and may be easier to continue at home, especially in a community with a poor understanding of the potentially harmful effects of strong analgesics.

The effects of opioids and non-opioids are additive, and make a useful combination, in that non-opioids act peripherally, whereas opioids act centrally.

Make sure that patients know that pain can be treated. Use drugs *regularly* every 4h by the clock or continuously IV. Make sure you use them before the pain starts again, and *don't prescribe them 'as required'*.

Use the oral route where the gastrointestinal tract is working well, and allow the patient himself or his family to administer the drugs appropriately. When the patient needs morphine, *don't be afraid to supply it:* almost all pain yields to it. So *don't underprescribe or underdose*. It might be easier to use opioids as an outpatient because of strict drug control policies within the hospital. If legislation controlling the availability of opioids makes this difficult, strive to have it changed. The right dose, of the right drug, at the right time, will completely control cancer pain in 90% of cases. Drugs are much less successful in many forms of non-malignant pain.

PAIN CAN BE RELIEVED:
PRESCRIBE DRUGS 'BY THE CLOCK'

WHO 3-STEP LADDER

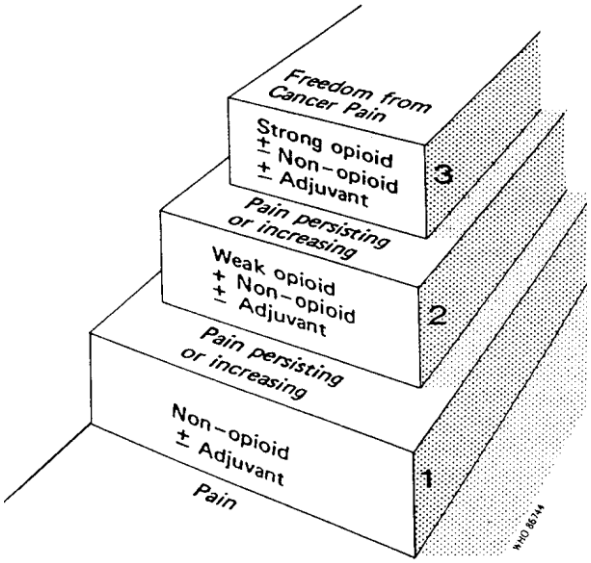


Fig. 37-2 THE WHO 3-STEP LADDER OF PAIN RELIEF will deal with the pain of about 90% of cases of terminal cancer. Prescribe a patient the right dose of the right drug at the right time. Prescribe it at regular intervals, and *not merely on demand*.
After Cancer pain relief and palliative care. WHO Technical Report Series 804 #891021, 1990 Fig 1, p.9.

Be careful to distinguish:
(1) Tolerance, which is a state in which increasing doses are needed to maintain the initial analgesic effect.
(2) Physical dependence, which is the onset of acute symptoms and signs when the drug is discontinued.
(3) Psychological dependence, which is the craving that is shown by drug abusers.
Tolerance to opioids is common in cancer patients, but is rarely a problem, and physical dependence does occur. Psychological dependence is rare, and is unimportant, because the patient is going to die anyway.

Don't withhold opioids from dying patients 'because they might get addicted'!

Finally, *don't remove the dignity of dying*. We have all got to die one day. Respect the wishes the dying person has. Many may wish to die surrounded by their family, and not by oxygen, tubes, cardiac monitors and alarm buzzers, and with tubes in their every orifice.

(a) Pain assessment

Ask about the location, distribution, quality, and severity of the pain; whether it is continuous or intermittent; and what factors make it worse or better. Pain precipitated by movement is likely to be less easily relieved than persistent pain. Enquire about the extent to which it limits sleep and other activities, and ask the patient to compare it with other pains (toothache, labour pain etc). Assess it on a scale of 0-10, with 10 being the worst pain the patient can imagine. Let him give you a value of his own pain score every time you see him. Evaluate his psychological state (anxiety, depression, suicidal thoughts etc), so that you can assess his 'total perceived pain' in terms of its physical, psychological, spiritual, social, and stress components. Consider special types of pain, and the relief of other symptoms.

(b) The analgesic ladder. Use drugs in the following order, with the aim of progressively:
(1) Increasing hours of pain-free sleep.
(2) Relieving pain when at rest.
(3) Relieving pain during standing or activity.

Titrate the dose of the drug you give against the amount of pain, gradually increasing it until you get the effect you want. Prescribe each dose by the clock, before the effect of the previous one has fallen off, so as to remove the memory and fear of pain.

Use the WHO pain ladder (37-2):
(0) Use enough chlorpromazine to cause drowsiness, especially at night, and use it regularly.
(1) If chlorpromazine is not enough, add a non-opioid (aspirin or paracetamol). Replace aspirin with another NSAID if the patient finds it more effective (and can tolerate it).
(2) If chlorpromazine and non-opioids are not enough, add a weak opioid (codeine or tramadol).
(3) If chlorpromazine, non-opioids, and a weak opioid are not enough, replace the weak opioid with a strong one (morphine). The indication for morphine is the intensity of pain, not the brevity of the prognosis. Pethidine, fentanyl and tilidine have shorter durations of action and are not very suitable for terminal care.

Alternatively, omit any of these stages if necessary.
If a drug ceases to be effective, *don't change to an alternative of similar strength*. Instead, go to the next step, and prescribe one that is definitely stronger.
When a patient goes home, he ideally needs the same drugs as in hospital. Prescribe plenty of chlorpromazine, and if necessary aspirin or paracetamol.

If opioids are needed, explain their dangers and that they are to be given to nobody else. If you think the relatives are unreliable, try to arrange hospice care.

(c) Monitor the pain. Do this within 4h, within a few days, and always after 1wk. Make sure that treatment continues to match the pain with the minimum of side and toxic effects. Try to anticipate and prevent them, and treat them systematically, especially constipation and nausea.

CAUTION!
(1) Determine the analgesic dose for patients individually.
(2) Always use oral drugs where you can.
(3) Pain is often worse at night, so treat insomnia vigorously. You may be able to double the opioid dose at bedtime in order not to wake the patient to administer a night-time dose.
(4) Exclude acute conditions that require urgent treatment.
(5) Learn to use a few drugs well, and *don't search desperately for one which will suit better*.
(6) If you decide to allow him home with a strong opioid, his relatives must understand its dangers, and return any drugs which are not used. You may need to collect these.

(d) Adjuvants

(1) **Chlorpromazine** is a *Group 1* phenothiazine which has pronounced sedative effects, but moderate anti-muscarinic (dry mouth, hypotension, blurred vision, urinary retention) and extra-pyramidal (tremor, facial tics, restlessness) effects. Start with 75mg nocte.
If anxiety persists during the day, add 25mg tds up to a total daily dose of 300 mg. (In severe psychoses, increase this to 1g daily.)

(2) **Thioridazine** is a *Group 2* phenothiazine with moderate sedative, marked anti-muscarinic, but mild extra-pyramidal effects. It is useful in the elderly disturbed patient. Start with 50mg od up to 300mg od.
N.B. Rarely, this drug can cause ventricular dysrhythmias.

(3) **Fluphenazine, perphenazine, prochlorperazine** and **trifluoroperazine** are *Group 3* phenothiazines with less sedative, less anti-muscarinic, but more extra-pyramidal effects. They are useful for their antiemetic effects (especially prochlorperazine).

(4) **Haloperidol** is a butyrophenone with properties like the *Group 3* phenothiazines and is useful for the severely agitated from 0·5mg od to 3mg tds according to response. Unlike the other drugs, it can be given to children at 12·5-25μg/kg bd.

CAUTION! Don't use more than one antipsychotic drug at the same time.
Remember you can get much relief with trans-cutaneous electric nerve stimulation (TENS); portable battery devices are available at US$40.

(e) Non-opioid analgesics

(1) **Paracetamol** 0·5-1g every 4-6h is universally available and cheap. As a result, it is maligned by both doctors and the public, but it remains an extremely effective analgesic. Overdosage is dangerous because liver damage results, which may not be apparent for a week, especially in alcoholics and those taking anticonvulsants.

(2) **Aspirin** 300-900mg every 4-6h is also universally available and cheap. It has anti-inflammatory properties and so can be used in combination with paracetamol. Its limitation is its gastric irritation effect. Other NSAIDs may be better tolerated (mefenamic acid, naproxen, piroxicam, indomethacin, ibuprofen, diclofenac) but they all cause gastric irritation. Piroxicam is useful as it has a prolonged duration of action.

N.B. All non-opioids act outside the brain.

(f) Weak opioid analgesics

(1) **Codeine phosphate** 30-60mg every 4-6h is very useful and not used enough. It is seriously constipating, and so, unless there is diarrhoea, combine it with a laxative every other day.

(2) **Tramadol** 50-100mg qds is a newer opioid and is very useful, but remains expensive. It can give hallucinations.
N.B. Tolerance and dependence are unusual.

(3) **Buprenorphine** 200-400μg qds sublingually or by skin patches is very useful because of its prolonged (8h) action, but is more expensive. Dependence can be a problem, and respiratory depression is only partially reversed by naloxone.

(g) Strong opioid analgesics

Morphine 5-10mg 4hrly orally, as a starting dose, and increasing when necessary, to 200mg or more 4hrly. Most patients are best controlled on 5-30mg every 4h by the clock. This is most easily given as solutions of morphine sulphate 1-20mg/ml in 5% alcohol, or chloroform water, as a preservative, stored in a dark bottle and not exposed to sunlight. Morphine is bitter, and you may prefer to mask the taste by taking it with some other drink. Try to get this mixture made up locally. If necessary, you can mix in 50-100mg of chlorpromazine in each dose. Capsules of morphine exist but are expensive. Most palliative care patients need 75mg/day for the last 3 months of their life for adequate pain relief.

Constipation may be more difficult to control than pain. Almost all patients receiving regular morphine need a laxative every other day.

Most patients receiving regular morphine need an antiemetic such as chlorpromazine (which the patient may be using already), or prochlorperazine 5-10mg tds increasing to 4hrly, or metoclopramide 10mg tds increasing to 4hrly, or haloperidol 1-2mg od.

Efficacy decreases with repeated use (tolerance), so that increasing doses are needed (37-3). Withdrawal symptoms may occur if treatment is stopped abruptly (physical dependence).

Occasionally opioids cause neurotoxic symptoms: delirium, hallucinations, myoclonic attacks and also hyperalgesia or allodynia (excessive sensitivity to pain). In this case, ensure good hydration and stop the current opioid and use an alternative, calculated for equal effect, reduced by 25% to account for cross-over tolerance:

Morphine 10mg = Codeine 100mg = Tramadol 40mg = Buprenorphine 150µg

Opioids cause respiratory depression: treat an acute overdose with naloxone 0·4-2mg IV every 2-3mins until respiratory function improves. Consider if this is really appropriate with a terminally ill patient.

(h) Muscle relaxants

Diazepam 5-10mg od is as effective as any other relaxant in treating muscle spasms. It helps also as a night sedative, although other benzodiazepines may give less hangover effect. It is important, though, to remember that the quality of sleep is poorer with sedatives because the regeneration of the body is less.

PLASMA DRUG LEVELS IN PAIN RELIEF

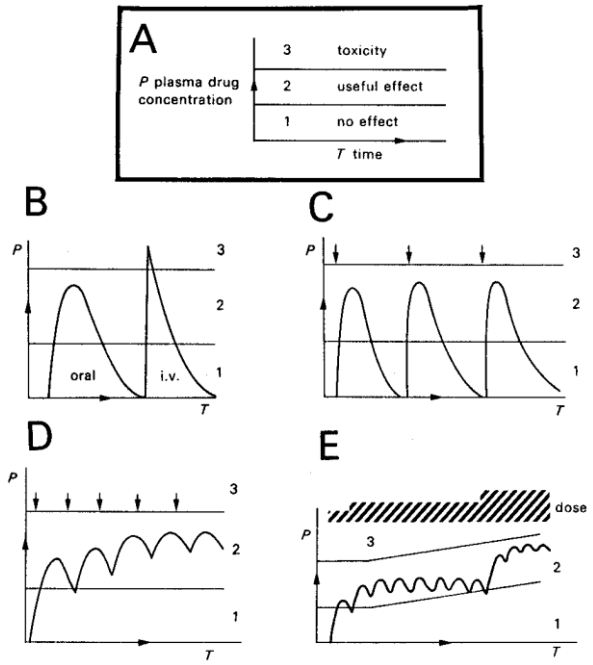


Fig. 37-3 PLASMA DRUG LEVELS FOR PAIN RELIEF.
A, plasma concentration zones in relation to drug effects. These same zones are used in the diagrams which follow. B, plasma concentration-time curves for an oral and an intravenous dose. Notice that after an oral dose the concentration rises to a lower peak, and persists longer. C, doses are too widely spaced to maintain analgesia. Larger doses at the same time intervals would risk toxicity. D, doses spaced satisfactorily to maintain analgesia (4hrly for morphine). E, effects of tolerance. The thresholds between no effect and a useful effect and between a useful effect and toxicity are rising.
After Vere DW, Oxford Textbook of Medicine, OUP 1983.

(i) Gastrointestinal analgesics

(1) **Loperamide** 2-4mg qds and/or **hyoscine hydrobromide** 300µg qds may help colicky intestinal pain. Antacids and **metoclopramide** will help with gastric distension and hiccough.

(2) **Cyclizine** 50mg tds or **domperidone** 10-20mg tds are useful adjuncts for nausea.
Remember to use laxatives if there is constipation, which is common and very distressing for the patient. It is an almost invariable side-effect of opioids.

(j) Neuropathic analgesics

Amitryptyline 10-25mg, **carbamazepine** 100-200mg bd, or **phenytoin** 150-300mg nocte are useful for shooting or stabbing pains. They will also control convulsions caused by renal failure or cerebral oedema.

If there is a specific point giving rise to pain or a specific nerve involved, inject the site with **methylprednisolone** 40mg (or **hydrocortisone** 200mg) in 2ml **lidocaine**.

(k) Steroids

Dexamethasone 4-8mg od (or **prednisolone** 30-60mg od) is especially useful to decrease chronically raised intracranial pressure, pain from nerve compression as well as chronic bronchospasm. It may improve appetite and promote a sense of well-being.

(l) Difficulties with terminal care

If you cannot rely on regular doses of opioids, you may be able to use a continuous IV infusion: 100mg morphine in 1L 5% dextrose at 25-50 drops/min provides 0.1-0.2mg/kg/h for a 70kg patient.

In a child, adjust doses appropriately. Anxiety and nausea are usually more of a problem.

If there is bone pain from metastases, arrange radiotherapy if possible.

If there are bed sores, turn the patient 2hrly, clean the wounds and debride them if necessary (34.16).

If there is a smelly ulcer or fungating tumour, use metronidazole 400mg tds and apply yoghurt or honey 4-6hrly.

If there is intractable vomiting, put up an IV line and pass a nasogastric tube (4.9) and use an antiemetic IV or PR. This will relieve nausea: intractable vomiting is a horrible way to die. Try metoclopramide, chlorpromazine, prochlorperazine, domperidone, or cyclizine. *Avoid metoclopramide and domperidone if vomiting is due to malignant bowel obstruction*, because they increase bowel motility, and may make things worse. Use dexamethasone if you suspect cerebral metastases.

If there are excessive respiratory secretions, try hyoscine 0·4mg qds sc.

If the mouth is dry, check the hydration, make sure there is good mouth care, and suggest sucking of ice or pineapple. Treat any candidiasis present. Stop hyoscine and reduce opioids.

If there is dyspnoea from a pleural effusion, drain it (9.1). When the effusion is fully drained, insert some talc to cause an inflammatory reaction to cause the pleural surfaces to stick together (36.1).

YOUR ACTIVE INTEREST IS ESSENTIAL!

37.3 Treating malignancy in a district hospital

Malignant disease is a worldwide problem but many cancers are avoidable (*viz.* Kaposi sarcoma and HIV, hepatoma and hepatitis B, bladder carcinoma and schistosomiasis, bronchial carcinoma and smoking). Incidence rates vary markedly in different parts of the world; in the developing world the five most prevalent are: cancer of the cervix, stomach, mouth, oesophagus, breast, with cancer of the liver and lung, lymphomas and leukaemias not far behind.

In some areas, certain cancers are very common (*viz.* Kaposi sarcoma in sub-Saharan Africa, bladder carcinoma along the Nile, Burkitt's lymphoma in Central Africa, nasopharyngeal carcinoma in southern China).

Although many malignant tumours can now be cured if they are diagnosed sufficiently early and treated appropriately, most of the patients who consult you will probably have such advanced disease that the help you can provide will be limited. Although a few tumours can be managed optimally with very limited facilities, many cannot. Nevertheless, if such a cancer is treatable, particularly for children, try to find the effective treatment.

Although malignant disease kills huge numbers of people worldwide, its treatment and cure is usually very expensive; however, the relief of suffering is often *not* very costly. Moreover, there is always something you can do, even if only good terminal care (37.1).

Your hospital may be a long way from any referral centre, so that if you don't diagnose and treat a patient with cancer, it is likely that nobody else will. You may be unable to get prompt and reliable histological reports, or even any reports at all, and there will almost certainly be no one to examine frozen sections for you. You are unlikely to be able to refer anyone for radiotherapy, or even perhaps for expert surgery, so you will have to rely on simple surgery, and some of the easier and cheaper chemotherapeutic regimes. Keep costs in mind, especially if these have to be borne by the relatives, and the possible benefit of expensive treatment.

Remember the possibility of complications and side-effects. *Don't end up by making things worse!*

(a) Management of malignant disease

(1) **Make the diagnosis** of malignant disease clinically, and examine the patient carefully to assess its extent. Assess the complications.

(2) **Confirm the diagnosis histologically** before you start treatment unless you have strong evidence of Burkitt's lymphoma (17.6), *e.g.* on cytology. If you are excising a lymph node (17.3), take the whole node if this is easy, but if it is not, a part of the node will do. If you are sending away a node for histology, always cut it across: with experience you will be able to recognize the caseation of tuberculosis, and to distinguish hyperplasia from a tumour. *Don't wait for the result if you think TB is a real possibility: start treatment.* However, after taking a biopsy for malignancy, wait for the histological report before starting toxic chemotherapy.

N.B. When you take tissue from an ulcer or a large mass, take some from the edge of the lesion, so that you include normal and abnormal tissue.

(3) **Stage the tumour** according to standard criteria. Most malignant tumours have 4 stages, or sometimes 5, some of which may be subdivided. The first 2 stages are usually curable, whereas the last 2 can usually only be palliated. Be thorough, and assess the tumour carefully: you may need a GA. Both the stage of a tumour, and often its histological grade, influence the prognosis, both with and without treatment. Occasionally, you will see some large resectable tumours, malignant or benign (24-7). *Don't assume that a tumour is malignant, until you have proved it so*.

(4) **Decide what is best for the patient.** Can you treat him? Can someone else treat him? Can nobody treat him? Should you aim for cure or palliation? For example, chemotherapy can usually achieve a radical cure with Burkitt's lymphoma (17.6), and quite simple surgery can cure skin (34.5), early breast (24.4) and penile cancers (27.33). Palliation may greatly help a patient with prostatic cancer (27.22), pancreatic cancer (cholecystojejunostomy), or oesophageal cancer (insertion of a prosthetic tube). A chest drain may relieve a massive pleural effusion, whose recurrence you may forestall by installation of talc (9.1). Often though, you can do little, because both surgery and chemotherapy may only prolong his suffering and that of his relatives.

(5) **Decide if you are going to try to refer the patient, or treat or palliate yourself.** If a referral centre can do nothing for him, *don't refer him*. If private doctors cannot help him, *persuade him not to waste his money on them*.

When you make the difficult decision as to whom to treat and whom only to palliate, base your decision on the response of the tumour, and not on the patient's political influence, his social status, or his ability to pay for the drugs. It is hard to be realistic when it comes to recommending palliative chemotherapy! Unfortunately, in places where medicine has to be bought by individual patients, the poor are likely to get nothing.

Even so, make every effort to have some drugs available to treat such conditions as Burkitt's lymphoma (17.6) and choriocarcinoma (23.10).

You will also have to decide where the treatment of tumours comes in your own priorities, when more cost-effective calls on your resources are so great.

ADVANCED UNTREATED VISIBLE NEOPLASIA

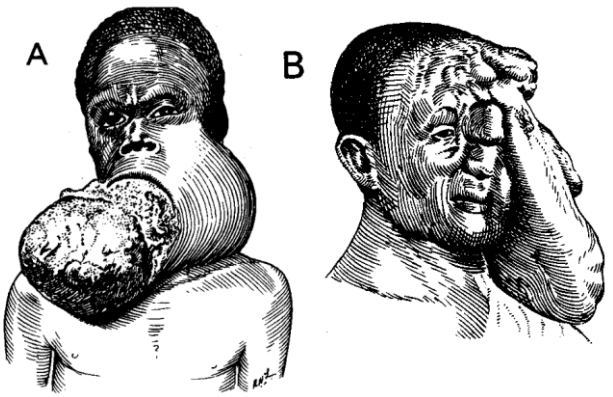


Fig. 37-4 ADVANCED UNTREATED FACIAL NEOPLASIA.
A, very large giant cell tumour of the mandible (31.6) **in a boy of 12yrs. B, large angiosarcoma** (34.15) **of the blood vessels of the scalp. In the absence of adequate treatment, tumours may become massive.** *After Bowesman, C, Surgery and Clinical Pathology in the Tropics. Livingstone, 1960 with kind permission.*

(b) Sending specimens. When you fix a tissue, place it in at least twice its volume of formol saline, which is 100ml concentrated (40%) formaldehyde solution with 9g salt in 900ml water. Send small specimens in a screw-capped universal container inside a plastic bag in a cardboard box, jiffy bag or padded envelope. Alternatively you can use the tied-off finger end of used gloves for very small specimens. Or, fix the specimen first in formol saline for a few days, then wrap it in formol-saline-soaked cotton wool; pack this in a plastic bag. *Make sure it does not dry out in transit.*

N.B. If you want to send a very large specimen for histology, such as an entire kidney, cut it so that the fixative can reach its interior, but leave the slices together at one edge, so that they can be put together again, and the shape of the specimen is preserved. Fix the whole specimen in a bucket of formol saline. When it is fixed, seal it in a polythene bag and pack it in a cardboard box; you may then have to send it by messenger!

CAUTION! The danger in sending pathological specimens is that they will leak in the post, contaminate the mail, and make you very unpopular with the post office!

N.B. Remember to label and orient your specimen carefully, with a hospital number in case the name is unreadable, and give accurate clinical information to the pathologist on your request form.

Unfortunately, there are no other tumours which are quite so readily treated by chemotherapy as Burkitt's lymphoma. You need to know where chemotherapy alone or adjuvant chemotherapy (*i.e.* in association with surgery) is useful.

37.4 Primary cancer chemotherapy

For chemotherapy you will need an accurate scale to measure weight, and a height scale on the wall. From these you can work out a patient's surface area. You must also be able to monitor the blood urea, the haemoglobin, the total and differential white count (from which you will be able to work out the absolute granulocyte count), and if possible the platelets.

ADVANCED TREATABLE MALIGNANCY

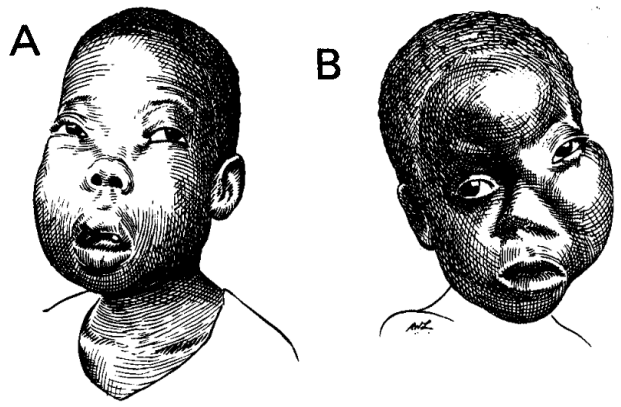


Fig. 37-5 BURKITT'S LYMPHOMA (17.6) **should be your 1st priority for chemotherapy. A, note the swelling of both maxillae. B, note the protrusion of the eye: *without immediate treatment, vision will be lost.***
After Bowesman,C. Surgery and Clinical Pathology in the Tropics, Livingstone, with kind permission.

NARUNDRNATHAN (2½ yrs) complained of a loose tooth and swelling of the upper jaw, which had started 2wks previously. He was taken to a dentist, who noticed proptosis in the left eye, and referred him to the children's ward. Here he was found to have several abdominal masses, was diagnosed as having Burkitt's lymphoma, and was referred to the teaching hospital. There was no money left in the transport vote to buy petrol for the hospital ambulance, so his parents took him there by bus. The consultant oncologist was away at a conference in Oxford, so he was sent back again, and asked to re-attend 1wk later. While he was waiting to be sent to the oncologist again, he died, after a total history of less than a month. LESSONS (1) The diagnosis of Burkitt's lymphoma is usually sufficiently distinctive for treatment to be started without waiting for a biopsy report. (2) Burkitt's lymphoma needs urgent treatment, which should begin within 48h, is not expensive, and is highly effective. (3) Treatment is easily possible in a district hospital.

Before you use any cytotoxic agent, you must decide if the misery, which its side-effects may certainly cause, will outweigh the benefit you expect it to have. Chemotherapy is one of the treatment methods for which compliance is absolutely necessary. The indications for chemotherapy are limited but:
(1) if you don't treat cancer patients, it is probable that nobody else will either;
(2) some drugs are comparatively affordable, and some regimes are practical and highly effective, particularly those for Burkitt's lymphoma (17.6), for which treatment is urgent;
(3) patients and their families much prefer to be treated near their own homes;
(4) it is often cheaper for patients to obtain their cytotoxics elsewhere (if you cannot supply them) and bring them to the hospital for you to administer. We comment on those that may still be affordable in a low-income set-up.

Use the WHO tumour categories:
Category 1: Chemotherapy will cure or significantly prolong life:

Acute leukaemia, lymphoma, gestational trophoblastic disease (GTD), seminoma, teratoma, nephroblastoma, neuroblastoma, rhabdomyosarcoma, Ewing's sarcoma, small cell lung carcinoma, retinoblastoma, and Kaposi sarcoma.

Category 1/2: Chemotherapy usually prolongs survival:

Breast carcinoma, osteosarcoma.

Category 2: Chemotherapy will reduce tumour size, improve the quality of life and may prolong life:

Chronic leukaemia, multiple myeloma, ovarian carcinoma, cervical and endometrial carcinoma.

Category 2/3: Chemotherapy may reduce tumour size, but overall benefit is equivocal:

Most remaining malignancies.

Category 3: Chemotherapy is of little certain benefit:

Melanoma, hepatoma.

Chemotherapy in a district hospital is never easy. Your laboratory facilities may be minimal, your drugs limited, your nurses inexperienced with chronic cancer patients, and your rehabilitation facilities rudimentary. You do however have two advantages: you can follow up a patient more easily than a referral centre, and his relatives are likely to live much nearer.

Use the KARNOFSKY PERFORMANCE SCALE to assess the quality of life of the patients you treat:

100%	No evidence of disease.
90%	Normal activity but signs or symptoms of disease present.
80%	Needing effort for normal activity.
70%	Totally independent, but unable to work or do normal activity.
60%	Requiring occasional help, but meeting most personal needs.
50%	Requiring considerable help, and needing frequent nursing care.
40%	Chair or bed-ridden, needing special care.
30%	Severely disabled, needing hospitalization.
20%	Disability complicated by severe sickness.
10%	Moribund.
0%	Deceased.

Before you start treatment, decide on your goal. Although a particular tumour may be curable, not all patients with it may be cured. Base your decision to treat a patient on: the stage of the tumour, the sites of its metastases, its particular histology, the state of his vital organs, his nutrition, the presence of other diseases, especially HIV, his willingness to accept toxic symptoms, and the staff and facilities you have to treat any complications he might get.

Use drugs only from a recognized source, and gain experience in their use. *Don't use them unless*:
(1) You know or are prepared to look up their general mode of action.
(2) You know their toxic effects and dangers, and have the necessary facilities to monitor these.
(3) You have decided what you are aiming for: cure or palliation?
(4) *You remember that an overdose can be fatal*. Even a normal dose can sometimes be fatal. Doses are usually given in relation to surface area, so have a table ready to relate this to height and weight. Carefully follow the rules about reducing the dose and stopping treatment when necessary. Remember that experienced doctors with compliant patients sometimes have disasters; for example, a patient can die from septicaemia in 48h.

If you do attempt cancer chemotherapy, you will have to know your limitations, and care for patients meticulously. Finally, do your utmost to see that a patient completes his course and is not abandoned. Either strive to provide a full course, or don't attempt chemotherapy. There is no justification for the idea, 'let's just try a little cyclophosphamide'.

Most cytotoxic drugs have their main action on rapidly dividing cells, which unfortunately include the cells of the bone marrow, the mucosa and hair. Malignant cells divide continuously, whereas marrow cells are quiescent for part of the time. Intermittent doses allow the marrow to recover, whilst maintaining an anti-tumour effect; but *don't wait so long that the tumour re-grows between courses*. A common regime is to give high intermittent doses over <24h, and to repeat them every 2-4wks.

You can often use cytotoxic drugs in combination because:
(1) They often act synergistically at different stages in cell division, or in different ways.
(2) Smaller doses can be used.
(3) If they have different toxic effects, their combined toxicity is minimized, because doses can be smaller.
(4) The tumour is less likely to become resistant. However, combined therapy is more expensive, and there may be more toxic effects, although each is less severe.

Tumour cells tend to multiply at a constant rate (37-6), depending on the proportion of cells dividing. Some grow very fast: Burkitt's lymphoma may double in size in 24h. Because of the constant rate of cell multiplication, a tumour grows exponentially, and its bulk increases more rapidly as it grows.

Similarly, chemotherapy kills a constant proportion of dividing cells, so that if it is sensitive, its size is also reduced exponentially. A large tumour may thus shrink rapidly to begin with, and then more slowly as it gets smaller.

Many cytotoxic drugs are very irritant indeed. If they extravasate into the tissues, they cause large necrotic ulcers.

N.B. If you inadvertently extravasate cytotoxic drugs, inject LA with hyaluronidase around the subcutaneous area affected, to dissipate the toxic tissue effects.

Infuse some cytotoxics, such as vincristine, dactinomycin, and doxorubicin, through a freely running IV line. Administer others by bolus IV injection with care! The IV route is usually better than oral, because high levels of the drug reach the tissues. Injecting irritant drugs into veins can cause thrombophlebitis. Over a long period, this can easily make all the accessible veins blocked. Try to insert a long cannula into a central or large peripheral vein, and bury it under a tunnel in the skin to prevent it becoming infected.

N.B. You can administer very few cytotoxic drugs IM.

Fatal complications of cytotoxic drugs are mainly septicaemia (80%), or intracranial haemorrhage (20%), from thrombocytopenia. Careful monitoring will minimize these dangers. You should only administer chemotherapy if the white cell count is >3/ml, and the platelet count is >150/ml. It is risky, but may be justified, to use chemotherapy if the white cell count is between 2-3/ml and the platelet count between 100-150/ml; however, if you don't have powerful antibiotics, platelet transfusions or fresh blood, and especially if the disease is one with a small or no chance of cure, you may do a patient more good by withholding chemotherapy completely, until his white count and platelet count rise. Treating septicaemia is difficult, and may require all the antibiotics you have; bleeding may be catastrophic and relentless.

Obviously, check if a female patient is pregnant before starting chemotherapy. In rare instances, you may need to sacrifice the foetus to save the mother. You may need to add non-hormonal contraceptive measures (to avoid venous thrombosis), and restrict breastfeeding.

DON'T MAKE A PATIENT'S QUALITY OF LIFE SO POOR IF REMISSION IS BRIEF

(a) Which cytotoxic drugs?

Most basic regimes use either vincristine or cyclophosphamide and usually both. The next most useful drug is methotrexate, which, in low-dose ranges, rarely causes myelosuppression or damage to mucosal linings, and so does not require folinic acid antidote rescue. Other drugs are dactinomycin, chlorambucil, doxorubicin, melphalan and cisplatin.

(b) Management

You will probably find it convenient to admit a patient beforehand for initial assessment and investigation, which must include a full blood count, pregnancy and HIV test, urea estimation and screening for liver disease. After the 1st course of chemotherapy, he can be discharged, and readmitted for each further course.

Be sure to deal with all treatable infections before starting chemotherapy. An infection which cannot be controlled is a contraindication to the use of cytotoxic drugs. A low-grade fever may quickly develop into septicaemia.

Establish a baseline from which you can monitor the response to treatment weekly, and before each course of treatment.

(1) Monitor the clinical condition on the Karnofsky performance scale.

(2) Follow the size of the tumour, measured with a tape measure or ultrasound in two planes at 90º. If there is a lymphoma, or metastatic glandular deposits, count all the nodes and measure them with a tape. Assess the degree of involvement of any organs that are infiltrated.

(3) Watch for toxic effects, and infection, clinically at least 3 times wkly.

(4) Monitor the full blood count.

(5) Culture the urine.

(6) Do other tests as necessary, for example the blood urea. If there is a Hb <70g/L before you start, transfuse with packed red cells, if necessary, repeatedly.

(7) Follow the weight.

(8) Assess the psychological state on a numerical scale.

INTRAVENOUS INJECTION

Oral chlorpromazine 25-75mg 1-2h before the injection will help considerably, by reducing nausea and vomiting. Further doses of this or other drugs, such as metoclopromide, cyclizine, haloperidol or domperidone, may be necessary.

If you are administering chemotherapy by bolus IV injection, make sure you wear gloves to protect yourself, use sterile precautions and have all your material ready: adhesive tape in the proper lengths, an infusion set or syringe ready for use, and the drug already drawn up in a syringe and well diluted! Start with the distal veins and use them in rotation. Use the longest possible cannula and inject some saline to be sure that it is not blocked or dislodged. When you are sure, inject slowly over 10mins. If the cannula becomes dislodged, start again with a fresh one. At the end of the injection, flush the vein with 10ml of saline.

CAUTION!

(1) Use an absolutely clean technique: *don't touch the tip of the needle or the infusion set*. If you happen to contaminate them, discard them and use fresh ones.

(2) Infuse dactinomycin, doxorubicin, and vincristine into a free-flowing infusion.

EFFECT OF CHEMOTHERAPY

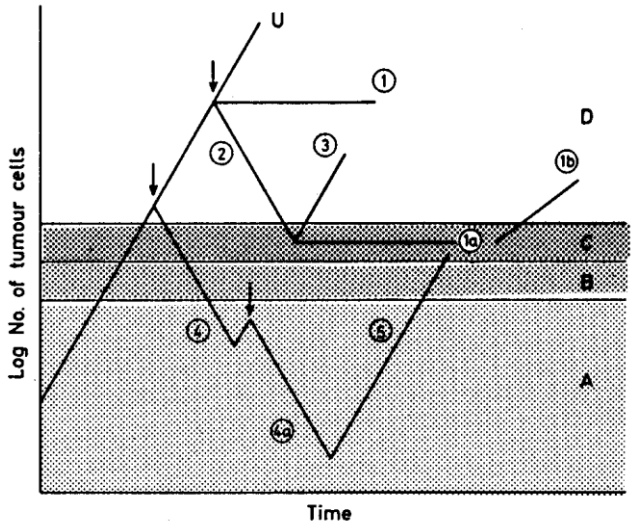


Fig. 37-6 THE EFFECT OF CHEMOTHERAPY on the size of a tumour cell population. A, B, C, and D represent differences in a patient's clinical condition. In A, there is no evidence of a tumour. In B, it can only be shown by special laboratory tests. In C, there is radiological or clinical evidence of disease. In D, there are symptoms. The growth of the tumour untreated is assumed to be exponential (U). Three courses of chemotherapy (shown by arrows) at the same dose destroy the same proportion of tumour cells, whatever the total number of cells when treatment begins. In (2) the symptoms are relieved, but there are still clinical and radiological signs of disease. In (4) the symptoms are relieved, and all clinical and laboratory evidence of the tumour disappears. In (4a) treatment starts when the patient is apparently healthy.
After the effect of treatment has worn off, the tumour grows at the same rate as it did before. In (3) the symptoms return rapidly. After (4) further growth is arrested by course (4a), which delays the return of symptoms (5). Maintenance therapy, at a low dosage that sustains a steady state, does not relieve symptoms in (1) and does not cause any regression in (1a). Maintenance treatment is continued in (1b) but the tumour has become resistant to treatment.
After Galton DAG. Medical Aspects of Neoplasia, in Oxford Textbook of Medicine. OUP, Oxford 1983 Fig 1 p. 493.

ALKYLATING AGENTS
These damage DNA and so interfere with cell replication:

Chlorambucil 6mg/m² (max 10mg) orally od for 4-8wks.
Use for lymphoma.

Cyclophosphamide 0·75-1·5m² IV every 7-10 days.
Don't use if blood [urea] >17mM.
Use for non-Hodgkin's and Burkitt's lymphomas, breast cancer, teratoma.

Melphalan 10mg/m² orally od for 4 days repeated 4-8wks.
Don't use if blood [urea] >17mM.
Use for myeloma, ovarian and breast cancer.

CYTOTOXIC ANTIBIOTICS
These inhibit cell division, and form irreversible complexes with DNA. They also block DNA-dependent RNA synthesis:

Dactinomycin 2mg/m² IV bolus every 3wks (or 1mg/m² on days 1 and 3) infused in a running IV infusion.
Use for paediatric cancers, teratoma.

Doxorubicin 50mg/m² IV bolus every 3wks infused in a running infusion. Reduce the dose in liver disease. *Beware cardiotoxicity. N.B.* It colours urine red.
Use for lymphoma, breast cancer and Kaposi sarcoma.

ANTIMETABOLITES
These are incorporated into nuclei or combine irreversibly with cellular enzymes, preventing normal cell division:

Methotrexate 15mg/m² orally for 4 days, repeated every 14th day. (30mg/m² wkly IV if the blood [urea] <7mM.)
CAUTION!
(1) *Don't use methotrexate if the blood [urea] >17mM,* because it accumulates when renal function is impaired.
(2) *Don't use it in the presence of an effusion (pleural or ascitic),* because it accumulates in the fluid, and so stays in the body long enough to cause an overdose.
(3) *Don't use aspirin with methotrexate.*
(4) At high dosage, you must use folinic acid 15mg qds for up to 8 doses to counter the folate antagonist effect of methotrexate.
Use for Burkitt's lymphoma (the low-dose range), oral cancer (an intermediate dose: 40mg/m²), nasopharyngeal cancer (high-dose range: 150mg/m²), GTD (100 mg/m²), breast cancer and teratoma.
N.B. This drug is also used for some inflammatory conditions, such as rheumatoid arthritis, or psoriasis.

5-fluorouracil 500mg/m² IV od day 1&8.
Reduce dosage in liver disease.
Use for breast cancer.

VINCA ALKALOIDS
These act at the metaphase of mitosis and inhibit RNA synthesis: they are neurotoxic, resulting in peripheral paraesthesiae, loss of deep tendon reflexes (*e.g.* foot drop), and may cause ileus, abdominal pain and constipation.

Vincristine 1·4mg/m² (max 2mg) IV bolus wkly, infused in a running infusion. Reduce the dose in liver disease.
CAUTION!
Use for all lymphomas, nasopharyngeal cancer, breast cancer, Kaposi sarcoma, teratoma, nephroblastoma.

PLATINUM COMPOUNDS
These destabilize DNA. They are nephrotoxic, ototoxic, and neurotoxic.

Cisplatin 70 mg/m² wkly max, or lower doses with other agents such as fluorouracil and folinic acid.
Use for testicular, lung, cervical, bladder, ovarian and head + neck cancer.

VARYING THE DOSE INTERVAL

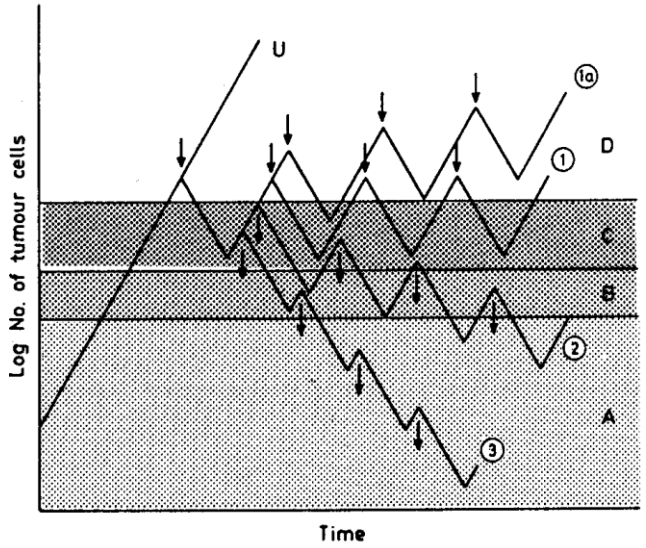


Fig. 37-7 THE EFFECT OF VARYING THE INTERVAL between courses of treatment, at the same dosage, on the size of a tumour cell population. The stages in a patient's clinical condition are the same as in 37-6. In (1), the interval between courses allows symptoms to return, but is short enough to maintain an essentially steady state. In (1a), the intervals are rather longer, and after the second course treatment fails to relieve the symptoms. In (2), the 2nd course begins before the return of symptoms, and the 5th begins when there are no clinical or radiological signs of disease, although the tumour can be shown by special tests. In (3), the intervals between courses are short enough to reduce the tumour cell population considerably.
After Galton DAG, Medical Aspects of Neoplasia in Oxford Textbook of Medicine, OUP, Oxford 1983, Fig 2, p. 494.

37.5 Looking after the AIDS patient

The terminal care of the AIDS patient should be no different in terms of help and sympathy than for the patient with cancer or other infectious disease. However, the HIV patient may suffer more with guilt, and social exclusion in certain societies. There may also be smelly wounds and fistulae. This situation becomes more complex when the relatives know the HIV status and the patient doesn't (or they don't think he does), or *vice versa*. Also the HIV patient is often much younger than the cancer patient, and may well not be ready to die. This is not necessarily more frequent with children. Such patients (and their relatives) ideally need the psychosocial support of a pastoral care team: if you are over-committed in a district hospital, you will face burnout very quickly if you take on this workload as well; *but you can organize so that others will.*

You should take precautions with dealing with the HIV+ve patient's secretions, blood and waste products. This also applies to wrapping up the dead body, and performing a postmortem examination.

If, rarely, the deceased has not told his relatives of his diagnosis, you have an obligation to inform them of the true cause, because of possible HIV transmission to the spouse and the various ramifications and consequences of this.

37.6 Postmortem (autopsy) examination

A postmortem examination, also known as an autopsy, is a medical procedure that consists of a thorough examination of a body to determine the cause and manner of death and to evaluate any disease or injury that may be present. Even though you may not be a pathologist, you may find yourself needing to perform an autopsy. This may be for legal or medical purposes, especially when the cause of death is unknown or unclear. Sometimes an external examination suffices, but occasionally the body needs dissection and internal examination. You may require permission for internal autopsy in some cases; alternatively it may be demanded by the police or the courts. After completing an internal autopsy, you must reconstitute the body by replacing the organs which don't require histological examination and closing the skin.

Postmortems are important in clinical medicine as they can identify medical errors and assist in the improvement of care. A systemic review of studies of autopsies calculated that in c.25% of cases, a major diagnostic error is revealed. A large meta-analysis suggested that c.30% of death certificates are incorrect and that 50% of autopsies performed produced findings that were not suspected before death.

There are 2 main types of autopsies, forensic and clinical. There may be strong cultural objection to an autopsy, and you need to be guided by local laws and practice. However, many relatives are comforted to know exactly from what their loved one has died, especially if there is suspicion of foul play.

(a) Forensic postmortem
In 44BC, Julius Caesar was the subject of an official autopsy after his murder by rival Roman senators, and the physician's report noted that the second stab wound Caesar received was the fatal one.

The principal aim of an autopsy is to determine the cause of death, the state of health prior to death, and whether any medical diagnosis and treatment before death was appropriate. A forensic autopsy is frequently performed in cases of sudden death, where completion of a death certificate is open to question, or when death is believed to be due to an unnatural cause. These examinations are performed under a legal authority and usually don't require the consent of relatives of the deceased. The most extreme example is the examination of murder victims, especially when medical examiners are looking for signs of death or the murder method, such as stab or bullet entry wounds and exit points, signs of strangulation or traces of poisoning.

Deaths are classified as:
(1) Natural,
(2) Accidental,
(3) Homicide,
(4) Suicide, or
(5) Undetermined.

(b) Clinical postmortem

Clinical autopsies serve two major purposes: to gain more insight into pathological processes and determine what factors contributed to a patient's death, and to ensure an adequate standard of care at hospitals. Autopsies can yield insight into how patient deaths can be prevented in the future.

There are 2 parts to the physical examination of the body: external and internal examination. Microscopy supplements these and frequently assists in assigning the cause(s) of death. Occasionally you need to request toxicology tests.

Make sure you wear gowns, gloves and a mask.

N.B. There is still a hazard of infection from HIV, hepatitis, Ebola and TB from postmortems!

Identify the body by a hospital label secured to it.

(c) External examination

After receiving the body, note the kind of clothes and their position on the body before they are removed. Next, collect any evidence such as residue, flakes of paint or other material from the external surfaces of the body. Then undress the body carefully, noting any tears in the clothing. Examine any wounds or lesions present and preferably take photographs, recording the sites. Generally, take 3 views: *overview*, *frame* and *close-up*, with a scale indicating sizes. Include the nametag if possible. Then clean, weigh and measure the body in preparation for the internal examination on a table in the autopsy room.

Make a general description of the body as regards ethnicity, sex, age, hair colour and length, eye colour and other distinguishing features (birthmarks, old scar tissue, moles etc). Use a standard examination form to record this information. In some countries, an autopsy may comprise an external examination only. This concept is sometimes termed 'view and grant'. The principle behind this is that the medical records, history of the deceased and circumstances of death have all indicated the cause and manner of death without the need for an internal examination. This, however, is rarely likely to be the case in your situation.

If there is any injury to the body like gunshot or stab wounds, describe these carefully. Describe each individual wound, and locate its position on the body by distance (in cm) from the midline or a local landmark like the nipple, umbilicus or symphysis pubis. Describe the features of the entrance and exit wounds. Note whether any cavity is penetrated. If you find a foreign body *in situ*, state where and describe the nature of the object (*e.g.* calibre of bullet, knife). In instances where there are dozens of knife wounds, it might be necessary to handle them in groups: photographs are very helpful in this case.

Remember to look at the back! Beware that bruising may indicate how a body has been lying after death.

Look for generalized skin diseases, especially rashes or *petechiae.* Cherry-red skin or mucosa is a sign of carbon monoxide poisoning. Examine for localized deep burns, especially of hands and feet, suggestive of electrocution. If you see a black eschar, think of anthrax. Peeled-off skin suggests burns, an epidermolysis (often HIV-related), or necrotizing fasciitis. Red-white-blue patches on the skin suggest hypo- or hyper-thermia. Animal bite marks are fairly obvious, but consider also human bites.

A body found late may show signs of serious decomposition. In this case, be very careful to look for any signs of penetrating injury because many other signs may be lost. In a case of drowning, try to establish if the deceased was alive before being immersed in water, by the finding of diatoms in intact tissues.

At this point, you may find that external examination is inadequate. You then need to proceed to internal examination, for which you may need special permission.

(d) Internal examination

Place a plastic or rubber brick called a 'body block' under the back of the body, causing the arms and neck to fall backward whilst stretching and pushing the thorax upward to make it easier to cut open. This gives you maximum exposure to the trunk. The internal examination consists of inspecting the internal organs of the body for evidence of trauma or other indications of the cause of death.

Make a large and deep Y-shaped incision starting at the top of each shoulder and running down the front of the chest just lateral to the nipples, meeting at the lower point of the sternum. This allows maximum exposure of the neck structures for later detailed examination. The cut then extends all the way down to the pubic bone (making a deviation to the side of the navel). Use shears to open the chest cavity in order to allow the sternum and attached ribs to be lifted as one chest plate; in this way, you can see the heart and lungs *in situ* and avoid damage to the pericardial sac. Use a scalpel to remove any soft tissue still attached to the posterior side of the chest plate. Now the lungs and the heart are exposed. Set the chest plate aside, eventually to replace it at the end of the autopsy.

If there is a penetrating injury, examine the trajectory and assess the damage made. *You will not be able to prove the existence of a pneumothorax unless you open the chest under water!* Look in the pleural cavity for evidence of fluid: is it blood, pus or a simple effusion?

Remove the organs in a systematic fashion. Unless there is evidence of pathology or damage in the neck or lower thorax, divide the major mediastinal structures as high as possible, and likewise divide the aorta, inferior vena cava and oesophagus just above the diaphragm, and thus pull out the thoracic contents *en bloc.* Examine this heart-lungs-oesophagus specimen on the laboratory table.

Open the pericardial sac to look for a haemopericardium and view the heart. Open the pulmonary arteries to search for a blood clot (thrombo-embolus). Examine the lungs, particularly for signs of pneumonia (pus in the parenchyma), oedema (fluid oozing out on squeezing) and bullae on the surface. Open the trachea to look for thick secretions, or stomach contents (broncho-aspiration). Open the heart cavities in a coronal plane, and look at the heart valves, the thickness of the heart wall, and signs of infarction.

Examine the abdominal cavity to look for free fluid: is it blood, bile, ascites or pus? (You can test the fluid for protein or amylase if you are uncertain.) Check for signs of organ perforation by gently squeezing the stomach, duodenum, intestines and gallbladder.

Look for penetrating injuries or haematomas from blunt trauma. Palpate the organs to determine if there is an obvious tumour, inflammation or adhesions. Look for signs of distended stomach, small or large bowel. Is the liver enlarged, cirrhotic, or mushy yellow (fulminant necrosis, *e.g.* due to mushroom poisoning); is the spleen enlarged?

Examine the abdominal organs systematically one by one after first examining their relationships and vessels. Carefully remove the liver (with the gallbladder), kidneys and spleen. Examine and weigh these organs and slice them to see if they are diseased. *Don't forget to cut the adrenals through*. Inspect major blood vessels and cut them open if you suspect any pathology. Next examine the stomach and intestinal contents, which may be useful to indicate the time of death, from an understanding of the natural passage of food through the gastrointestinal tract after ingestion.

To examine the brain, make an incision from behind one ear, over the crown of the head, to a point behind the other ear. When the autopsy is completed, suture the incision neatly so it is not noticed when the head is resting on a pillow in an open coffin. Pull the scalp away from the skull in two flaps with the front flap going over the face and the rear flap over the back of the neck. Then cut the skull with a saw to create a 'cap' that can be pulled off, exposing the brain. Look at the brain *in situ*. Then sever the brain's connection to the cranial nerves and spinal cord, and lift it out of the skull for further examination. Look at the meninges for a thickened cloudy appearance, suggestive of infection. Slice the brain, looking for oedema, infarcts, hydrocephalus, haemorrhage or tumours. You may only see damage at the level of the brain stem. Weigh the brain.

TAKE PHOTOGRAPHS OF RELEVANT FINDINGS

(e) Microscopy

By making imprints or smears of different organs, you can confirm inflammation, tumour, or necrosis by cytology with use of Giemsa-stain even in remote clinics. If facilities for histology are available, the result is usually only available much later, and may not be useful for immediate purposes. Nonetheless you can cut open a tumour and get a pretty good idea if it is malignant, benign, or inflammatory (*e.g.* from TB) most of the time.

(f) Reconstitution of the body

An important component of the autopsy is the reconstitution of the body so that it can be viewed, if desired, by relatives of the deceased following the procedure. After the examination, the body has an open and empty chest cavity with chest flaps open on both sides. It is unusual to examine the face, arms, hands or legs internally. All organs and tissue can be returned to the body unless any tissue is needed for further investigation. Place the organs in a cellulose or plastic bag to prevent leakage and return them to the appropriate body cavity. Place the body block that was used earlier to elevate the chest cavity to elevate the head, close the chest flaps and suture the skull cap back in place.

Clean the body surface thoroughly with water and a sponge to remove blood or excreta.

Remember you need to get consent in writing if you wish to preserve body parts for teaching purposes. Always record organs sent for forensic or pathological examination elsewhere.

(g) Weights of organs of a normal adult

(75kg, norm ±20%)

Lung: right	450 g
Lung: left	400 g
Heart	300 g
Liver	1500 g
Brain	1300 g
Kidney (rt or lt)	150 g
Spleen	150 g

(h) Weights of normal organs of the newborn at term

Lungs	70 g
Heart	15 g
Liver	150 g
Brain	450 g
Kidneys	30 g
Spleen	10 g
Pancreas	5 g
Adrenals	5 g
Thymus	10 g

(i) Checklist

Try to fit the diagnosis to the symptoms before death. The following are some causes of non-traumatic sudden death:

Respiratory	Tracheal occlusion	Secretions, Foreign body, Laryngeal oedema, Strangulation/asphyxia, Goitre/neck tumour, Ludwig's angina.
	Pulmonary failure	Pneumonia/TB, Pulmonary embolism, Tension pneumothorax, Haemothorax, Smoke inhalation, Pulmonary contusion.
Vascular	Haemorrhage	Trauma, Ruptured ectopic gestation, Ruptured aneurysm, Bleeding peptic ulcer, Bleeding oesophageal varices, Intestinal haemorrhage, Uterine haemorrhage.
Cardiac	Cardiac failure	Myocardial infarction, Cardiomyopathy, Cardiac rupture, Cardiac tamponade, Aortic dissection.
Cerebral	Cerebral failure	Cerebral haemorrhage, Cerebral infarction, Cerebral oedema, Meningitis, Pre-eclampsia.
Renal	Renal failure	Pyelonephritis, Glomerulonephritis.
Adrenal	Adrenal failure	Haemorrhage, Infarction, Anaphylaxis.
Peritoneal	Peritonitis	Sepsis, Intestinal volvulus, Pancreatitis.
Gynaecological	Uterine failure	Sepsis, Rupture.
Sepsis	Septicaemia	Necrotizing fasciitis, Gas gangrene, Tetanus, Other sources.
Toxins	Toxaemia	Poisons, Venoms.

38 Imaging

38.1 Radiology methods for the generalist

X-rays are electromagnetic waves of 0·1-0·5Å (0·01-0·05nm); they are usually generated in vacuum tubes by passing a high voltage current to heat tungsten filaments. These can occasionally break and need replacing, or else if they overheat (>3380˚C) give off tungsten vapour which is deposited on the inside of the vacuum tube, so reducing the output of X-rays. The heat generated by the X-rays must be dissipated, and most machines have a thermostat cut-off device.

The basic WHO radiological system (1.12) is the most appropriate for the district hospital. The machine consists of an X-ray tube, high tension cable, collimator and grid (to reduce the X-ray beam to the area of interest and improve the definition) and a cassette plate with its holder.

Wear lead body protection, and try to obtain a radio-activity dosimeter if you take many radiographs.

Take care to position your patient correctly, remove clothing (especially with buttons and objects in the pockets), ear-rings and piercings, make sure that braided hair is out of the way *(it can look like TB on a chest radiograph!)*, and select the correct X-ray penetration (usually indicated on the machine). Check if a female patient might be pregnant (*a history alone may not be accurate:* do a β-HSG test): *avoid X-ray exposure in this event*.

Make sure the radiograph is correctly labelled (best with a marker on the cassette) as to left or right.

Carcinoma of the oesophagus may be so common that you will find a barium swallow, which is quite easy to do, particularly useful. A barium meal and enema are more difficult, but with reasonable practice you can learn quite a lot from them. Upper gastrointestinal endoscopy (13.2) and procto-sigmoidoscopy (26.1) will usually give you more information, if available. Cystoscopy (27.3) may give you the information you need, and is cheaper than contrast radiology. Information from an ultrasound may, however, make this superfluous. However, a urethrogram, by outlining the passage to the point of a stricture or fistula, is simple and helpful.

N.B. You can often demonstrate a bowel fistula by injecting dye rather more easily, avoiding the need for X-rays.

(a) Intravenous urogram (IVU)

INDICATIONS

N.B. Do an ultrasound first, if you can!

(1) An obstructive uropathy, particularly a hydronephrosis or a ureteric stricture, especially in areas where schistosomiasis is endemic, provided renal function remains fairly good.
(2) To see if a mass is renal.
(3) To assess the function of the contralateral kidney, when you consider referring for nephrectomy.
(4) Renal trauma.
(5) Renal or ureteric stone (27.13).

CONTRAINDICATIONS

(1) If the blood [urea] >10mM, an IVU will probably fail because the contrast will not be excreted in adequate concentration to be visible.
It is certainly not worth doing if the blood [urea] is >17mM.
(2) Renal failure.
(3) Hepatic failure, which may be aggravated.
(4) Cardiac failure; there is a risk of arrhythmia.
(5) Dehydration.
(6) Babies <2yrs old.
(7) The first trimester of pregnancy.
(8) Multiple myeloma.
(9) Sickle cell disease.
(10) Any previous reaction to contrast, or to iodine.
You can prepare an allergic patient by administering prednisolone 50mg orally 12 and 2h before the injection of contrast.
Be prepared for an anaphylactic reaction: have ready 0·5ml of adrenaline 1/1000 and promethazine 25mg IM.

PREPARATION. Restrict the patient to oral fluids and use a laxative to empty the bowel. Air will not spoil the film, but a mixture of air, fluid and faeces will. *Do not use an enema*. If the IVU is urgent, do it without preparation.

CONTRAST MEDIUM. Use 'Urografin' (Meglumine and sodium amidotriyoate) 60% or 'Conray (Iothalamate meglumine) 420'. These are hyperosmolar solutions.
FILMS. Use 18x24cm for the bladder; 24x30cm for the renal area; 30x40, 35x35, or 35x43cm for the whole abdomen.

UROLOGICAL RADIOGRAPHS

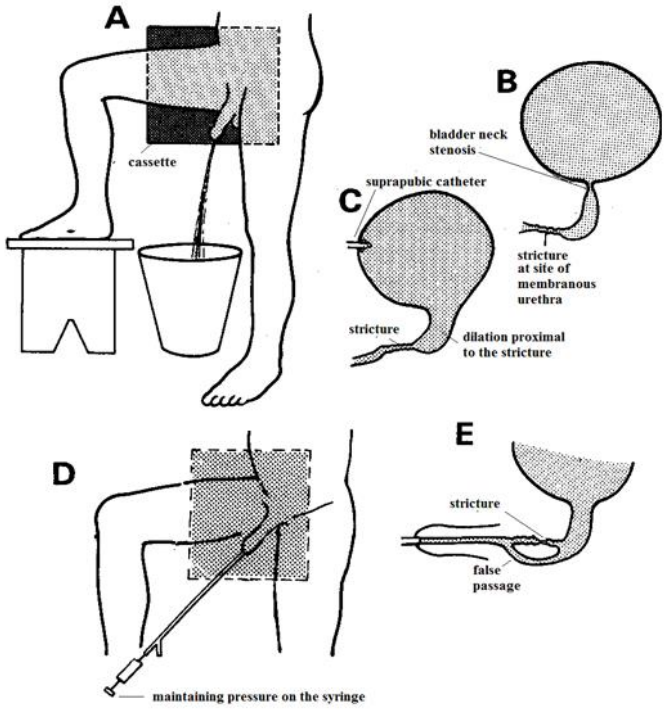


Fig. 38-1 UROLOGICAL FILMS.
A-C Micturating cystourethrogram. D,E Retrograde urethrogram. A, fill the bladder with contrast medium, and take a film whilst the patient is passing urine. B,C, 2 types of obstruction. D, inject contrast up the urethra from below (this shows an oblique view at 25°). E, demonstrating a false passage.

METHOD. The following method minimizes the number of plates needed. The views are all AP; 60mA at 68kV should be enough for a 60kg adult.
Lay the patient supine on the X-ray table. Take a preliminary view of the abdomen and pelvis on a 30x25cm plate, before infusing the contrast medium. Inject 40ml IV rapidly.
(1) At 3mins take a 25x30cm plate of the kidneys.
If the calyces are of obviously normal shape and size at 3mins (be quick: it will need 5mins in the developer, fixing and washing), you can omit the 20min film.
(2) At 20mins quickly take a 35x43cm plate to show the kidney, ureters and bladder.
(3) At 30mins ask the patient to empty the bladder, and then take a small plate to show the residual urine.
If the function of the kidneys is impaired, so that there is little excretion in the standard films, repeat them at 2h, and if necessary at 6h.

(b) Retrograde urethrogram (38-1D)
INDICATIONS
(1) Urethral stricture, or leak.
(2) Congenital anomalies.
(3) Prostatic abscess.
(4) Urinary inguino-scrotal fistulae.
(5) False urethral passages.
(6) Urethral rupture.

CONTRAINDICATION. Acute urinary infection.

CONTRAST MEDIUM. Use contrast medium such as 'Umbradil Viscous U', 45% diatrizoate (Hypaque), or Urografin 60%.
N.B. You can dilute these with an equal volume of water and still get good pictures.
CAUTION! Do not use barium, or an inorganic iodide.

METHOD. This is most often done in males. Using aseptic precautions, insert a Ch12 Foley catheter for only 2cm into the urethral orifice. Gently inflate the balloon with *only* 1-2ml of sterile water. The expanded balloon will now fix the catheter in the *fossa navicularis* (27-18). Fix a length of tubing to the catheter. You can now position the patient more easily, and be further from the radiation yourself when you inject the contrast medium. Check that there is no extravasation by injecting sterile water from a bladder syringe connected to the Foley catheter.
Then inject c.10ml of the medium and take an oblique film at 25° (with the patient lying on his side with the pelvis tilted to 45° and the upper hip out of line of the X-ray beam). Take the film maintaining mild pressure on the bladder syringe. If the bladder is reasonably filled, you can follow a retrograde urethrogram with a micturating cystourethrogram.
The membranous urethra is separated from the penile urethra by a line, which runs from the junction of the upper ⅓ and the lower ⅔ of the pubic ramus on one side, to a corresponding point on the other side.
A stricture shows as a significant narrowing in the urethra outlined by the contrast, almost invariably in the membranous urethra.

(c) Micturating cystourethrogram (MCU, 38-1A)
N.B. This shows up the proximal end of the stricture, and will also show bladder neck stenosis. The bladder must be filled with contrast medium to begin with. If you can pass a catheter through the urethra, you can fill it that way, but contrast may leak out when you remove the catheter. If a suprapubic catheter is *in situ*, use that to fill the bladder.
INDICATIONS
(1) Urethral stricture.
(2) Suspected abnormalities of the bladder neck.
(3) The assessment of vesical diverticula.
(4) Cysto-ureteric reflux.

CONTRAINDICATION. Acute urinary infection.

CONTRAST MEDIUM. Use either:
(1) 40ml 60% 'Urografin' diluted in 400ml of saline,
(2) 250ml 30% 'Urografin', or
(3) 400ml 12·5% sodium iodide with 0·1% sodium metabisulphite. This is the cheapest.
The sodium iodide must be sufficiently pure; *small contaminants of fluoride in it can be nephrotoxic.*

METHOD. Empty the bladder through the catheter. Using an intravenous infusion set or a large syringe, fill the bladder again through the catheter with 300-400ml of contrast medium until there is a strong desire to pass urine. Clamp a suprapubic catheter or remove a urethral catheter. Take an erect oblique AP film at 80mA and 80kV with the patient standing to pass urine. Or take an oblique film at 25° in the lying position; there will be less blur due to movement. Use a cassette to hold the X-ray plate still.

(d) Contrast swallow (38-2)
INDICATIONS
N.B. Do an endoscopy first if you can.
Dysphagia due to:
(1) Carcinoma of the oesophagus (30.5).
(2) Post-corrosive or reflux strictures (30.3).
(3) Achalasia (30.6).

CONTRAINDICATIONS
(1) Impaired conscious level.
(2) Dysphagia of neurological cause.
(3) Tracheo-oesophageal fistula.
(4) Suspicion of an oesophageal tear.

CONTRAST MEDIUM
(1) Thin barium, gastrografin or Iodipamide (Endografin).
(2) Barium sulphate; thicker than for a barium meal, yet not so thick as to aggravate an obstruction.
CAUTION! Check to make sure the patient does not have a complete dysphagia, which may cause aspiration. Do not use barium carbonate, which is highly poisonous.

METHOD. Stand the patient in front of the X-ray screen facing you. Ask him to fill the mouth with contrast medium, but not to swallow it until you ask him to. Use a closed feeding cup (which is less likely to spill in the dark), or a stiff 5mm plastic straw. The mixture of air and barium swallowed will produce an informative ‘double contrast’ film.

SOME CONTRAST SWALLOW RADIOGRAPHS

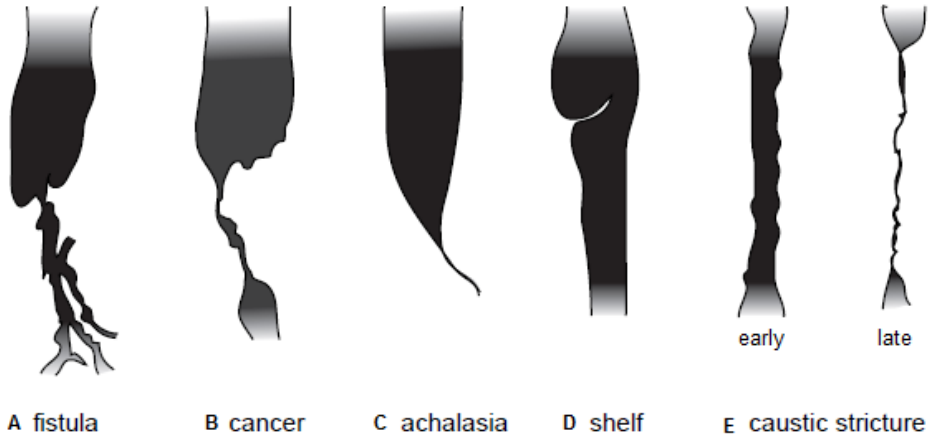


Fig. 38-2 SOME OESOPHAGEAL SWALLOW RADIOGRAPHS. A, malignant fistula into pleural cavity or bronchus. B, late oesophageal cancer. C, achalasia of the cardia. D, a shelf as in a short corrosive stricture or Plummer-Vinson syndrome. E, grades of long caustic stricture (early & late).

Adjust the X-ray machine to provide a narrow vertical aperture. Then ask the patient to swallow and take a film while he is swallowing the contrast. Then take a lateral film with the patient standing with his hands above his head. You should be able to see all but the upper end of the oesophagus quite easily. If necessary, take 2 oblique views, in addition.

(1) **Malignant strictures** (oesophageal carcinoma) cause a narrowing with an abrupt start and an irregular rounded shoulder; above it the oesophagus is usually dilated. The lumen through the tumour is irregular and is typically rat-tailed, and you can usually see the end of the stricture. You should be able to demonstrate 90% of carcinomas with simple screening. An ordinary PA film may show widening of the mediastinum. Be sure to use a long plate to get the whole oesophagus on to it, and *do not centre the X-ray tube on only one part of it*.
A mouthful of contrast medium and one large film will usually show an advanced tumour.

(2) **Late inflammatory benign strictures** due to reflux produce a stricture which is usually long and smooth.

(3) **Achalasia** (uncommon, 30.6) shows as a 'bird's beak' at the bottom end of a greatly dilated oesophagus. Take oblique films.

(e) Barium meal
INDICATIONS
N.B. Do an endoscopy first if you can.
(1) Carcinoma of the stomach.
(2) Gastric outlet obstruction.
(3) An upper abdominal mass suspected of being gastric.

METHOD
Perform a barium swallow first with thin barium.
Adjust the aperture of the X-ray beam to let you see the entire stomach. Ask the patient to stand facing you.
Then ask him to swallow thicker barium, enough to fill half the stomach. If you can, screen him in this position and in the left oblique position (he should look beyond your left shoulder), or use the right oblique position.

(1) STOMACH
Watch for peristalsis carefully. Does barium pass through normally all the way to the pylorus?
Suggesting carcinoma: an immobile area of stomach lining, persistently irregular surface, or consistent filling defect make carcinoma very likely.
Suggesting a gastric ulcer: an ulcer, usually on the lesser curve, in the distal half of the stomach. Expose 1-3 plates, asking the patient to hold his breath as you do so.
N.B. Looking for a hiatus hernia is not very reliable with radiographs, and anyway does not always correlate with gastro-oesophageal reflux.
Suggesting a diaphragmatic hernia: a round volume of contrast in the thorax.

(2) DUODENUM
Note if there is a delay in the passage of barium through the pylorus: it should start passing at 1-5mins. Use a special attachment which will exclude all X-rays except those in a 10cm circle. Turn the patient to face obliquely to your right as far as is necessary for you to see the pylorus and duodenal loop. Expose plates as the barium passes. You may be able to recognize a deformed duodenal ampulla while you screen, but you will see scarring or a duodenal ulcer more easily on static films. With experience, you will recognize enlargement of the duodenal loop (as by carcinoma of the head of the pancreas).
Suggesting gastric outlet obstruction: pyloric delay keeping contrast in the stomach.

(f) Barium enema (38-3)
INDICATIONS
(1) Suspected amoebic strictures.
(2) Carcinoma of the large bowel.
(3) A mass which might be colonic.
(4) Inflammatory colitis.
CAUTION! Do not do a barium enema until you have performed both proctoscopy and sigmoidoscopy, which will often establish the diagnosis more easily and cheaply.

CONTRAINDICATIONS
(1) Patulous anus.
(2) Rectal biopsy immediately preceding the enema.

EQUIPMENT
An X-ray machine with a screen. A 2L douche can. A flatus tube, Ch30 rubber or plastic, 2m long. 2 large artery forceps. A Higginson's syringe (38-3). A grid on top of the X-ray plate.

METHOD
Prepare the patient with oral bowel preparation and a rectal washout (not merely an enema) within the previous 12h; maintain good oral hydration during this time, but withhold solids. Oral preparation is not necessary in children and a rectal washout alone is satisfactory.

CONTRAST MEDIUM. Use barium/air contrast with the equipment described (38-3).
N.B. Do not use oral bowel preparation on a patient with obstruction.

CAUTION! Manipulate the flatus tube with gloves. Have toilet paper and a bedpan ready: it may be required urgently!

CONTRAST COLONIC ENEMA

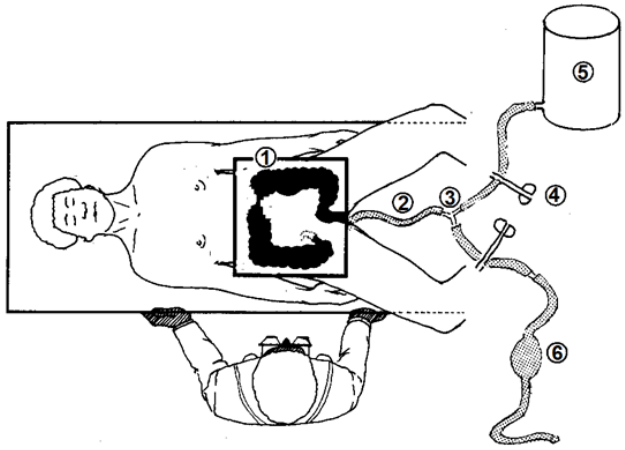


Fig. 38-3 ARRANGEMENTS FOR A RADIOLOGICAL ENEMA. You will probably be able to demonstrate the large bowel as far as the hepatic flexure without much difficulty; the ascending colon is more difficult. ***Note that you should wear goggles and an apron.***
1, a radiographic grid and X-ray plate; these are usually in a cassette under the X-ray table. 2, Ch30 flatus tube. 3, a standard Y-connector. 4, artery clamps. 5, a 2L stainless steel douche can. 6, a Higginson's pump syringe. (The illustration shows a typical 'filling defect' suggesting a colonic cancer, 12.11**).**
Adapted from James Cairns & Rogers Mungalu.

Lay the patient supine on the X-ray table, and ask the patient to flex and abduct the hips. Cover the male gonads with a lead shroud. Lubricate the flatus tube well with lubricant, and push it through the anus, as far as it will go easily. Extend the legs on the table. Inject barium and air, as required, to show the large bowel up to the caecum. Inject a fair amount of barium first, and follow this by pumping air with the Higginson's syringe to move the barium proximally, clamping and releasing the tubes as necessary. Instil some barium, and then some air. You will probably be able to demonstrate the large bowel as far as the hepatic flexure without much difficulty; the ascending colon is more difficult. The limiting factor is the distension of the large bowel with barium and air, and the urge to defecate that this produces. If there is much discomfort, wait 2-3mins and try again with more barium and more air.

If possible, watch the movement of barium and air on screening, and expose plates of critical areas. Before defecation, which the patient is usually keen to do without delay, take a standard abdominal film. Expose another plate after defecation. The film of barium left on the mucosa will often give the clearest picture. Finally, remove the flatus tube and allow bowel emptying into a bedpan.

(g) Carotid arteriogram (38-4)
INDICATIONS
(1) Suspected extra- or sub-dural haematoma.
(2) Intracerebral mass.
(3) Suspected cerebral aneurysm in a patient with a subarachnoid haemorrhage.
(4) Arteriovenous malformations.

CONTRAINDICATIONS
(1) Bleeding disorder.
(2) History of transient ischaemic attacks.
(3) Previous allergy to contrast, or to iodine.

EQUIPMENT. An X-ray head table, preferably marked to line up the midline. 2-way connecting tubing. Quickly changeable cassette box.

METHOD. Place the patient supine with the head carefully placed AP in the midline position; it is useful to be able to strap the forehead in place. If the patient is restless, sedate him appropriately. Sterilize the skin of the neck on both sides and infiltrate 2ml 1% lidocaine under the skin over the pulsations of the carotid artery. *Do not put in too much*, otherwise palpation of the artery will be difficult. If you have an ultrasound, this is very useful to localize the artery.

CONTRAST MEDIUM. Prepare 20ml iopamidol (Isovue 200 or 370) or iotalamic acid 45% (Conray 280) in a sterile syringe. These contain 200, 370 or 280g iodine/L respectively. *Do not use stronger solutions* as they may cause vasospasm. Make sure the radiographer is ready.

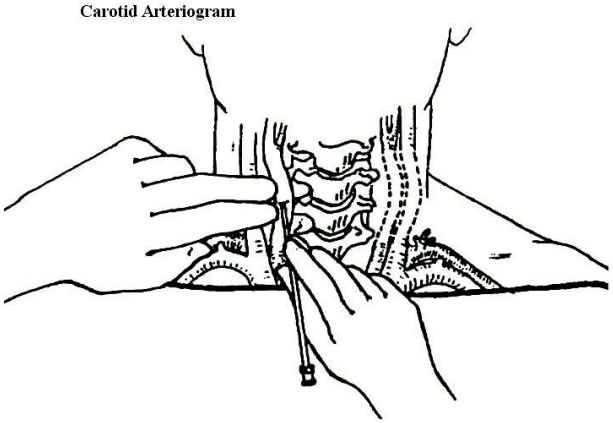


Fig. 38-4 TECHNIQUE OF CAROTID ARTERIOGRAPHY. Direct puncture of the carotid artery.
After Rosenfeld JV, Watters DAK. Neurosurgery in the Tropics. Macmillan 2000 p.41 Fig 2.16 with kind permission.

Puncture the carotid artery directly from the front with a 19G 5-8cm long lumbar puncture needle (38-4), and secure it. This should be flushed with saline and have a plastic connection, already attached, to which you can fit the syringe with the contrast medium. Inject a 10ml bolus of contrast and take films whilst you are injecting, and then every 2secs after you have put in 5ml. If you cannot do this, inject all the contrast quickly and take one film as the last drops of the contrast injection are going in. (*You must shout to the radiographer when to shoot!*)

Then inject another 10ml contrast and get 2 more films for the capillary and venous phases after 3 & 6secs.

Being careful not to displace the needle, turn the patient's head on its opposite side, and repeat the injection of 10ml contrast to get films in the lateral position.

Finally, when you are satisfied with the quality of the films, remove the needle and press on the puncture site for a full 1min, *without occluding the flow in the carotid artery*.

Look for displacement of the anterior cerebral artery from the midline: this is why accurate alignment of the head is so important. Look for an area of the brain on its edge that seems to receive no blood supply. Look for a 'tumour blush' or vessels displaced around a lesion. Look for a 'blow-out' of an aneurysm.

(h) Myelogram
INDICATIONS
(1) Progressive neurological limb deficit suggestive of an intraspinal lesion, *viz.* para- or tetra-paresis.
(2) Acute spinal cord compression.
(3) Sciatica not improving with conservative treatment.

CONTRAINDICATIONS
(1) Raised intracranial pressure.
(2) Septic lesions on the back.
(3) Allergy to iodine.

METHOD. Place the patient in the lateral position with the lumbar spine well flexed but not crooked, with a pillow between the knees. Find the CSF as for a lumbar puncture at the L4-5 level, and inject 20ml iopamidol. For lumbar views, take AP and lateral films soon after the injection. For thoracic views, tilt the table, so that contrast runs up the spinal canal, and then take more AP and lateral films.

(i) Sialogram (17.5)
INDICATIONS. Stricture or stone in the salivary duct.

CONTRAINDICATION. Active sialadenitis.

METHOD. Anaesthetize the buccal mucosa with lidocaine spray. Find the duct orifice: the parotid (Stensen's) duct opens opposite the 2nd upper molar tooth (31-1) on the buccal side, and the submandibular (Wharton's) duct opens at the side of the frenulum of the tongue. Pass a probe to dilate the orifice, and inject a little lidocaine; then inject 0·5ml contrast and immediately take the films of the mouth.

(j) Sinogram and fistulogram
INDICATIONS. To demonstrate a sinus or fistula.

METHOD
If you suspect a fistula to extend from skin to the large bowel, use bowel preparation beforehand.
Make sure you plug the sinus or fistula, so that when you inject contrast it does not spill back out: you can use stoma paste for this, or pass a small Foley catheter and inflate the balloon to secure it in place. Take films in AP, oblique and lateral positions.

(k) Operative cholangiogram (15.8)
INDICATIONS
If during cholecystectomy you feel a stone or if you have lost a stone in the common bile duct, or there has been a history of jaundice, or the common bile duct is dilated.

METHOD
When you have clearly identified and exposed the cystic duct, attach a 20ml syringe with saline to a fine plastic cannula and flush out air bubbles, and pass the cannula into the cystic duct by making a small opening into it. Secure the cannula in place with a fine suture, after making sure the saline passes freely into the duodenum. Remove instruments and swabs from the operative field, and cover the wound with a sterile towel. Tilt the table 10-15° to the left, and get the X-ray machine properly positioned. Withdraw bile into the cannula to make sure it has not slipped out of the duct, and then inject 4ml of 25% warm sodium diatrizoate (Hypaque) contrast medium and take a picture. Repeat this with 8ml for a second exposure. Gallstones appear as filling defects, so it is vital that air bubbles are removed as these may be confused with gallstones!
Whilst waiting for the films, *do not proceed with removing the gallbladder*, because you may want to do a cholecystojejunostomy (15.9) if you cannot remove a stone impacted in the distal common bile duct.
If the films show no flow into the duodenum and there is no sign of a filling defect, try flushing with warm saline and adding 20mg hyoscine IV (an antispasmodic), and repeating the test with 12·5% sodium diatrizoate. (You can use air, however, if all you are testing for is free flow into the duodenum.)

(l) Hysterosalpingogram (HSG: 19.3)
CONTRAINDICATION
Pregnancy.
CAUTION!
(1) Before you start, do a pelvic examination (or ultrasound scan) to exclude pregnancy and active pelvic infection, and
(2) Do a β-HSG test within 10 days of the 1st day of a woman's menstrual period, and not in the premenstrual or active menstrual phases of the cycle.

PREPARATION. Administer prophylactic tetracycline and metronidazole beforehand. Use a special cannula such as the Leech-Wilkinson screw-in type (the Miller cannula causes less trauma to the cervix, but does not make such a good seal with it) or a very small size Foley catheter but this needs a special syringe to provide a proper seal.

CONTRAST MEDIUM. Prepare a suitable 20ml syringe filled with a water-soluble, such as 'Urografin', or other hyperosmolar contrast medium, which may help open the tubes.

METHOD. If possible, screen the patient during injection of the contrast medium. Lie her supine on the X-ray table with her hips and knees flexed, and the plate under her pelvis. A tube-plate distance of 1m is satisfactory.
Insert Cusco's speculum (19-1) and clean the cervix with cetrimide or povidone-iodine. Hold the cervix gently with a single-toothed tenaculum, lightly closed to the first ratchet; this should cause little discomfort.

Expel all air from the syringe and cannula, inject 20ml contrast medium firmly through the cervix, and take a film. If there is a cornual obstruction, 20ml will not pass through the Fallopian tubes out of the uterine cavity. If possible, take a second film 3h later. The contrast should have spread into the peritoneal cavity. If it remains loculated, this suggests adhesions and impaired fertility.

38.2 Ultrasound methods for the generalist

Unlike radiographic images, sonographic pictures are not generated by radiation but by sound waves of frequency 1-13MHz. The lower the frequency, the shorter the wavelength and so the greater the penetration.

This technique is therefore *non-invasive.* Sound waves are sent into the human body and are reflected in it. These reflections take place at the interface of different tissues/structures due to various sound transmission abilities. Therefore the brightness of an organ on the display is not related to its physical density but to its echogenicity. This depends on the difference of acoustic impedance, *i.e.* (density) x (speed of ultrasound waves passing through), between the tissues.

Consequently, fluids like blood, urine and pleural fluid appear black (anechoic) as there are no interfaces where sound could be reflected. Unfortunately, pulmonary/intestinal air and bone reflect almost all the sound waves, obscuring any image from what lies behind the air or bone. However, you can usually get round this problem (except in the adult head).

Ultrasound is cheap and quick and can help you in a whole set of potentially difficult or dangerous situations. But the quality and reliability depend, more than in other techniques of examination, on the experience and knowledge of the examiner, and the quality of the machine. If there is a doctor experienced in ultrasound around, ask him to teach you; it's worthwhile to learn this technique.

You will find a large variety of ultrasound scanners available on the market. Therefore, if you want to buy one, make sure you order an instrument of a specification that will allow you to get useful data from it. It may be better to have no scanner than to have one that gives poor-quality images which can lead to wrong diagnoses.

Use sector (convex) probes for the abdomen, and a round probe for cardiological examinations. Use linear (flat) probes for scanning superficial structures, including the breast and thyroid. A combined linear and sector probe can cover all areas; convex probes are also useful in the majority of body areas. *Remember to orientate the probe in your hand correctly so the image on the screen corresponds with the patient's position and is not back to front, or upside down! (Touch the side of the probe where the marker is situated to see on the screen to which side this corresponds.)*

N.B. Increase in gain brightens the image; increase in depth magnifies the image.

A good compromise frequency is 3·5MHz, while 5MHz is useful for scanning children and superficial organs. The WHO manual of diagnostic ultrasound (Vols. 1 & 2) produces good guidance on this:

The specifications for a general purpose ultrasound scanner are as follows (*N.B. check technical terms in guidebooks!*). It should have:

1. A transducer which is curvilinear (convex), or a combination of linear and sector.
2. A transducer with a central frequency of 3·5MHz, with accurate focusing. If you can afford it, an optional transducer of 5MHz is useful, but should not replace the 3·5MHz one.
3. A sector angle of ≥40°and a linear array 5-8cm long.
4. Simple control buttons easy to use. Overall sensitivity (gain or transmitter power) and time-gain-compensation should be an integral part of the circuit. These vary the clarity of the image from a pre-set level. However, this is not essential because if the time-gain-compensation is at the correct level for obstetrics, with a pre-set alternative for the upper abdomen, you can still examine >80% patients satisfactorily.
5. A frame rate 15-30Hz for the linear probe and at least 5-10Hz for the sector probe.
6. A frame freeze density 512x512x4 bits (to provide 16 grey levels).
7. At least one pair of electronic omni-directional calipers with quantitative readout, to measure lengths on the screen.
8. Software to add patient identification data to the screen and the final record.
9. Software to obtain a permanent record (memory) of the scan.
10. A video monitor screen measuring at least 10x10cm², preferably larger.
11. A reasonable weight, so that an average adult can move it over at least 100m; if on wheels, these must be suitable for rough irregular surfaces, but one that you can carry and even put in a vehicle is better. However, the more your machine is mobile, the more easily it may be stolen!
12. Protection for the local climate, and against dust, damp, and extremes of temperature. It should be possible to use the scanner continuously within a temperature range of 10-40°C and 90% relative humidity.
13. Connection to the local power supply and be compatible with the voltage, frequency and stability of the local current. It should be able to stabilize a voltage variation of ±10%. If there is greater fluctuation in the local current supply (and you must test this before you buy), you need an additional voltage stabilizer.
14. A local service provider for the equipment.
15. Instructions easy to understand, in a language staff can read!

N.B. Many ultrasound scanners incorporate biometric tables (*e.g.* foetal maturity measurements) in the microprocessor memory. These are useful, but check that the measurements you make are done in exactly the same way as was used to provide the tables. Biometric tables may not be universally applicable and should be adjusted for local conditions.

N.B. You do not need a printer! You can use petroleum jelly for the transducer if special jelly is too expensive.

(a) Artefacts

Before you start to have a closer look at ultrasound applications, note the simple phenomena which appear on an image due to the physical characteristics of sound. Find out what is really artefact and what is not. Some artefacts are useful, *e.g.* in diagnosing a pneumothorax (36.1).

(1) **Distal acoustic enhancement**
This is the relative enhancement of the signal of a wave beyond a fluid-filled cavity compared with the signal had it crossed through surrounding tissue. While passing the fluid, the sound is not much weakened. This causes the tissue beyond the fluid to appear brighter. Use this effect to distinguish between cystic structures and tissue!

(2) **Acoustic shadowing**
Structures which reflect sound very strongly like bones, stones and pulmonary/intestinal air prevent sound waves passing through. This results in blackish bands behind such structures which make evaluation there impossible.

Use this effect in atherosclerotic plaques and to reveal stones in the gallbladder, kidney and bladder! If the transducer has poor contact with the skin of the patient, there will be black bands through your image, too. But these ones start right at the skin level and they will disappear upon using more air-displacing gel.

ABDOMINAL ULTRASOUND TECHNIQUE

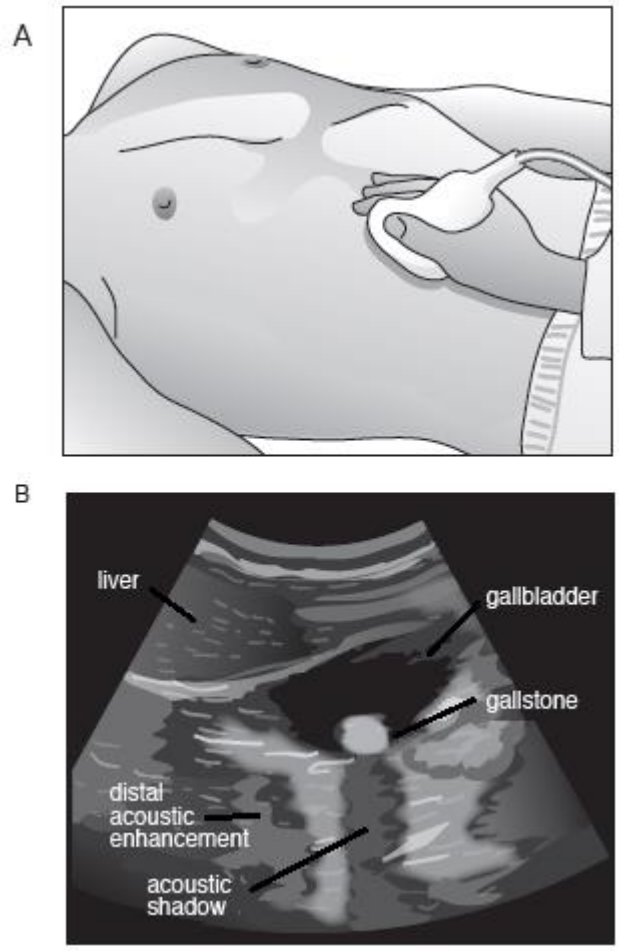


Fig. 38-5 TECHNIQUE OF ABDOMINAL ULTRASOUND.
A, position of the transducer. B, ultrasound image of a stone in the gallbladder.
After Sonographiebilder.de/II, Medizin/Albertinen-KH Hamburg.

(3) **Section thickness**
As the sound bundle that is emitted by the transducer has a (very small) thickness, sometimes the wall of a cystic structure appears thickened and fuzzy. Carefully distinguish this from layered material like blood clots or small concretions which change their localization after turning the patient around!

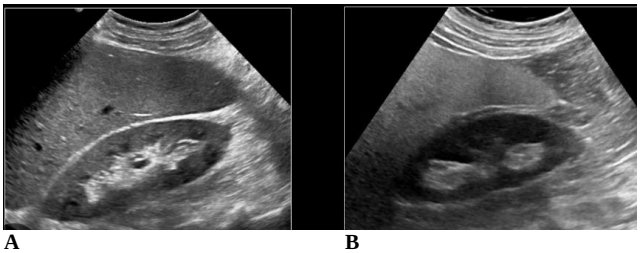
(b) Liver

APPROACH. Although the liver is easy to find, it is a challenge to examine it completely, not only for you but also for your patient who might get exhausted in holding his breath. Try to give your patient continuous breathing instructions and don't forget to allow him to breathe out after holding his breath. Ask for a deep inspiration and apply the transducer a little bit to the right side in the epigastric angle.

You should now comfortably see the dark IVC which is used to separate the left from the right liver lobe in ultrasound; the hepatic veins lead into the IVC, which is normally 13-15mm in diameter.

METHOD. Expect a healthy liver and kidney to have a similar echogenicity. A diffusely increased brightness of the liver is typical of a fatty liver, whereas an apparently reduced brightness is in most cases due to an increased brightness of the kidney (38-6). Sometimes you find a focally increased deposit of fat within the liver. These fatty infiltrations can look quite solid but are always sharply demarcated and never displace adjacent veins or bulge hepatic borders.

ULTRASOUND SCAN OF LIVER & KIDNEY



A **B**

Fig. 38-6 Comparative echogenicity of liver and kidney. A, the two have similar density. B, the fatty or cirrhotic liver is hyperechoic.
After Owen JW, Radiology Education.

In general, you don´t measure the size of the liver but instead evaluate the inferior marginal angle of the right hepatic lobe which should be <45°. The margin will appear rounded if the liver is enlarged. Throughout the parenchyma of the liver you will find porto-venous branches and hepatic veins. The former are always surrounded by a bright rim representing accompanying biliary ducts and peri-portal connective tissue, whereas the latter lack such a sign.

You can divide focal lesions into hyperechoic (brighter than the parenchyma) and hypoechoic lesions. Common findings in the liver are cysts (38-7A). Usually you don't need to be worried if the cyst appears anechoic and fulfils the cyst criteria (see below). These cysts are benign and require no further diagnosis. If a cyst is not anechoic anymore but displays internal echoes, you need to think of intracystic haemorrhage or of a parasitic hepatic cyst with septation (38-7B).

A common infection is due to *Echinococcus granulosus* (15.12), in which you will find one or several cystic structures with intraluminal echoes or solid parts (38-7C). The less frequent but more dangerous echinococcal disease (*Echinococcus multilocularis*) will appear as a mixed solid/cystic lightly calcified structure. Although it is difficult to differentiate such lesions from abscesses, hepatocellular carcinoma or metastases, *beware of aspiration* as there is a high risk of rupturing the cyst and seeding the parasite into the abdominal cavity.

ULTRASOUND SCAN OF LIVER CYSTS

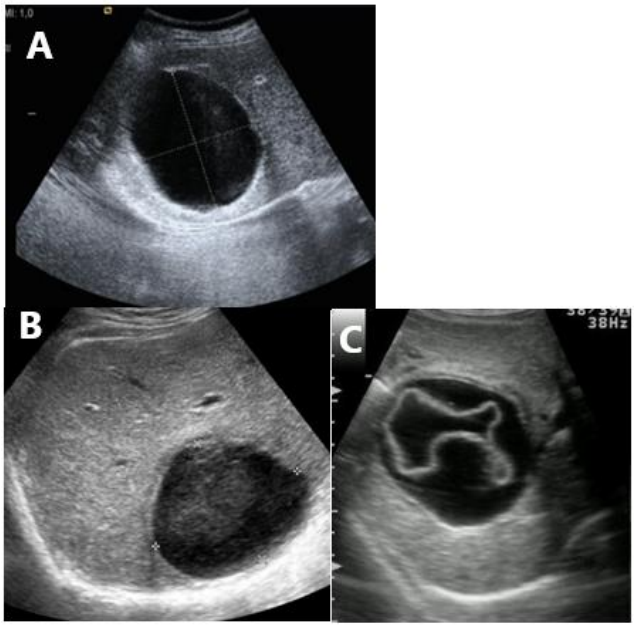


Fig. 38-7 Different liver cysts. A, a simple cyst (which may still be a hydatid). B, haemorrhage into a cyst. C, floating membrane inside a hydatid cyst.
After Davarci I, Tuzcu K, et al. W Indian Med J 2014;63(5):545-7 (A), Peer S. Radiopedia.org (B), Sonographiebilder.de,Medizin/Albertinen-KH Hamburg (C).

While you scan the parenchyma of the liver, pay attention to any kind of focal lesions which appear and disappear suddenly. Look for micro-abscesses suggestive of TB.

Although metastases in the liver present with a wide variety of echogenicity, a very typical sonographic sign is a dark narrow rim around the lesion which is called a 'halo'. Especially in fast-growing metastases, you can sometimes find a cystic hypoechoic centre caused by central necrosis. If a focal lesion has the same brightness as the liver parenchyma, you may only detect it due to space-occupying effects.

Look out for vessels which bend suddenly, bulging of the liver borders or jaundice in your patient because of compression of the biliary ducts. Focal lesions which present brighter compared with liver tissue can be harmless hepatic haemangiomas. They vary in size but are homogeneously hyperechoic, have a sharp but possibly irregular demarcation and typically display a bright narrow rim. Larger haemangiomas in which you also might find a dorsal acoustic enhancement sometimes become heterogeneous and are then difficult to differentiate from tumours. If your patient has a fatty liver, keep in mind that haemangiomas exceptionally are silhouetted hypoechoic against the very bright liver tissue.

Look for lymph nodes in the periportal area: these are very suggestive of TB.

(c) Gallbladder

To avoid acoustic shadowing while looking at the gallbladder:

(1) Use appropriate pressure for your abdominal scan. This will push intestinal air out of the field of view. Your view of posteriorly located organs will not be disturbed by acoustic shadowing.
(2) Use adequate breathing instructions. In inspiration the stomach and gas-containing bowel loops are displaced inferiorly and your view will not be disturbed by acoustic shadowing.

APPROACH. Apply the transducer in a sagittal orientation along the right mid-clavicular line just below the ribs (35-5A). Do your examination while the patient takes a deep breath and holds it. You will find the gallbladder as an anechoic structure near to the abdominal wall (38-5B). If you fail to see it, check that it hasn't already been removed, and place the patient in the lateral decubitus position. Try to get a true longitudinal view by rotating the probe on its axis: you will then see the gallbladder as an 'exclamation mark' on its side, with the right portal vein as the dot. If this fails, try with the patient in the knee-elbow position (12-5). Alternatively, especially in obese patients, view the gall bladder through the gap between the 9th and 10th ribs on the right anterolateral thoracic cage.

METHOD. Perform the examination before food. After food, the contracted gallbladder will not give you a good idea of oedematous wall thickening, polyps or stones and tumours (which are actually extremely rare).

Be sure to scan the gallbladder completely in 2 planes. Pay attention to luminal structures within the gallbladder. Stones usually generate acoustic shadowing, but tumours do not. If the stones are <4mm diameter, they may not give a shadow. Most often they lie in the most dependent part of the gallbladder and move about when the patient's position changes, unless they are impacted together and fill the gallbladder completely (38-8).

Peri-vesicular fluid will appear as a black fringe around the gallbladder as a sign of inflammation, perforation or ascites. The gallbladder wall is thickened (>2mm) in both acute and chronic cholecystitis: if >5mm it is definitely pathological.

ULTRASOUND SCAN OF CHOLECYSTITIS

Fig. 38-8 Cholecystitis: many stones giving an echo, and a thickened gallbladder wall.
After Shah C, Johnson PT. 123sonography,.com

If you press directly with the probe on the fundus of the gallbladder and this causes acute pain, it is a true 'Murphy's sign' and a very reliable sign of acute cholecystitis.

Make sure you are not causing pain by pressing on the ribs! It is crucial to recognize any air in the gallbladder due to a colonic fistula or to infection with gas-producing bacteria, as this is associated with a high risk of perforation. Cholecystitis usually occurs with gallstones present, but they may be absent especially in HIV disease.

If you suspect cholestatic jaundice, ultrasound can help you determine the level of obstruction. Measure the maximum diameter of the gallbladder, scan the common bile duct (best in the transverse view) which you will see as 2 bright lines anterior to the portal vein, and evaluate the small intra-hepatic bile ducts. Usually the intra-hepatic bile ducts are not visible but you will find them next to portal veins if they are dilated. If you find no dilation in either intra-hepatic or extra-hepatic bile ducts, jaundice is unlikely to be due to cholestasis, but from liver disease.

ULTRASOUND SCAN OF DILATED BILE DUCTS

Fig. 38-9 Dilated bile ducts have a 'tramway' appearance. Here many ducts are dilated from distal obstruction.
After Atlas of Ultrasound 2023 Erlangen Cookie-Richtlinien-EU.

You may spot worms or flukes moving in the bile ducts.

(d) Pancreas

APPROACH. Apply the transducer in a sagittal orientation to the right of the midline, tilt the head infero-laterally, and press hard. The pancreas is a relatively ill-defined structure superior to the aorta, IVC, superior mesenteric artery and vein.

METHOD. Adjust the position of the probe till you get an optimal view of the pancreatic tissue. This should be homogeneous, but if not, and if it is brighter and has fluid around it, or a visibly dilated pancreatic duct within it, these are signs of pancreatitis. If you see ≥5 dark structures in the area of the pancreas, one of these lesions is likely to be a pathological lymph node or a cyst: check for distal acoustic enhancement to confirm the presence of fluid in a cyst.

(e) Aorta and inferior vena cava (IVC): Retroperitoneum

APPROACH. To scan the superior retroperitoneum, apply the transducer in the epigastric angle, along the right of the *linea alba*.

It is helpful to ask your patient to take and hold a deep breath. With this manoeuvre the liver will move caudally and replace disturbing air-filled intestines.
After you have examined the superior part of the retroperitoneum, move the transducer caudally and do the same for the inferior retroperitoneum.

METHOD. As there are many things to pay attention to, try to stick to a concept. *Don't rush!* Your aim is not only to evaluate the aorta and the IVC for an aneurysm, a thrombosis or dilation but also to scan for lymph nodes. Firstly tilt the transducer probe to the patient's right side to scan the aorta which you will find dorsal to the liver. Does the wall of the aorta look smooth or do you find any evidence of artherosclerosis? Are there any localized dilations of the vascular lumen?

If there is a suspicious aneurysmal dilation, measure its diameter as the risk for rupture increases with size. Look out for intraluminal clots or a 'double' arterial wall, suggesting a dissection. These both appear less anechoic than the vessel itself because they might not only cut off the arterial supply to the spinal cord or to the kidneys but also increase the risk for rupture, too (38-10).
Then tilt the transducer probe to the left side to visualize the IVC and its continuation into the right atrium. In right heart failure you will find a dilation of the IVC as well as a loss of its typical 'double' pulsation. The normal IVC is smaller than the aorta. If you suspect right heart failure, scan also for pleural effusions and dilated hepatic veins. When you scan a vein, compress it from time to time. As long as a vein is compressible, there is almost certainly no thrombosis. Although this is a simple method, it is important as only a fresh thrombosis appears bright on your image: therefore you might overlook an older one if you don't compress the vein repeatedly.

ULTRASOUND SCAN OF AN AORTIC ANEURYSM

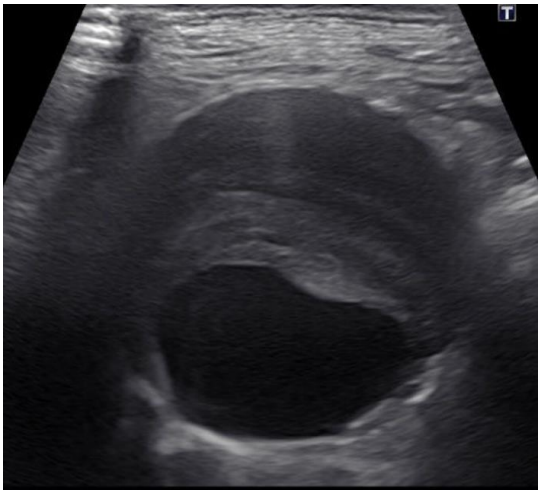


Fig. 38-10 Distended aorta with intramural atheroma.
After Atlas of Ultrasound 2023 Erlangen Cookie-Richtlinien-EU.

After your examination of the upper retroperitoneum, move the transducer caudally and do the same for the lower retroperitoneum. At some point you will not be able anymore to follow the aorta/IVC as it splits up into the external and internal iliac artery/vein.

If you suspect a deep vein thrombosis and you would like to scan further, change to a linear array transducer to examine the limbs.

(f) Kidney

APPROACH. The right kidney is much easier to examine than the left because you can use the liver as a window to scan through. Place the transducer in the mid-axillary line and ask the patient to breathe in deeply. Move the transducer slowly from the costal margin towards the iliac crest till you find the kidney, which is behind and inferior to the liver. Then rotate the transducer on its vertical axis so you see the whole length of the kidney, which lies obliquely. The left kidney can be difficult to visualize due to interfering ribs and intestinal air, so place the transducer in the posterior axillary line. If you have difficulty seeing the kidney, try placing the transducer head perpendicular to the intercostal spaces superior to the costal margin. *Try to avoid turning the patient to the side*: the kidney will move away from the abdominal wall and the image will probably worsen.

N.B. A kidney-shaped structure not in the correct position is likely to be an intussusception! This makes a doughnut-like image in the transverse view.

METHOD. A typical longitudinal section will be most useful for your evaluation of the parenchyma, but it is good to do a scan in both longitudinal and transverse views. The medullary pyramids are displayed like a row of black (hypoechoic) holes between the parenchyma and the central collecting system (brighter due to its hyperechogenicity).
It might be difficult to differentiate vessels, cysts and medullary pyramids especially if scanning conditions are poor.
Cysts are a common finding especially in the elderly and you usually should not be worried about them. Be suspicious if a cystic structure is combined with calcifications (it might be renal tuberculosis) or if the cyst has non-homogeneous solid parts which might represent a malignancy. Normally the parenchyma of the kidney looks slightly darker than the liver; it becomes brighter (diffusely increased echogenicity) in all types of renal damage: it is a sensitive but non-specific sign.
However, in normal children, the kidney is usually slightly brighter than the liver. If you are thinking of nephrotic syndrome, search for pleural effusions and ascites. Measure the size of both kidneys and compare them. With age the parenchymal rim decreases physiologically but it might also be a consequence of several inflammatory episodes or chronic urinary obstruction. Renal disease usually causes the kidney to shrink <9cm in length.
If you are thinking of urinary obstruction, scan carefully for renal pelvic stones (you might only detect the shadows), for tumours in the uterus or the urinary bladder and an enlarged prostate. You cannot easily see strictures of the ureter.
In urinary obstruction, distinguish 4 distinct degrees:
(1) I° : distension of the renal pelvis
(2) II°: distension of the infundibula and calyces (38-11A)
(3) III°: additional pressure atrophy of the parenchyma
(4) IV°: virtual disappearance of the parenchyma (38-11B).

You might have a patient who has cysts and distended calyces. To differentiate between these conditions, scan in 2 planes and look for communication between the cystic structures. You will not find any if it is a simple cyst.
Cysts are typically in the cortex or periphery, and are smooth-walled. Hydronephrosis will communicate with a dilated renal pelvis; if it is due to an enlarged prostate or pregnancy, it will resolve after emptying the bladder. Classically in advanced hydronephrosis the appearance resembles dark branches like the fingers of a hand.
You may find a stone casting a shadow like a gallstone (38-11C) but they are more difficult to spot.

ULTRASOUND SCANS OF KIDNEYS

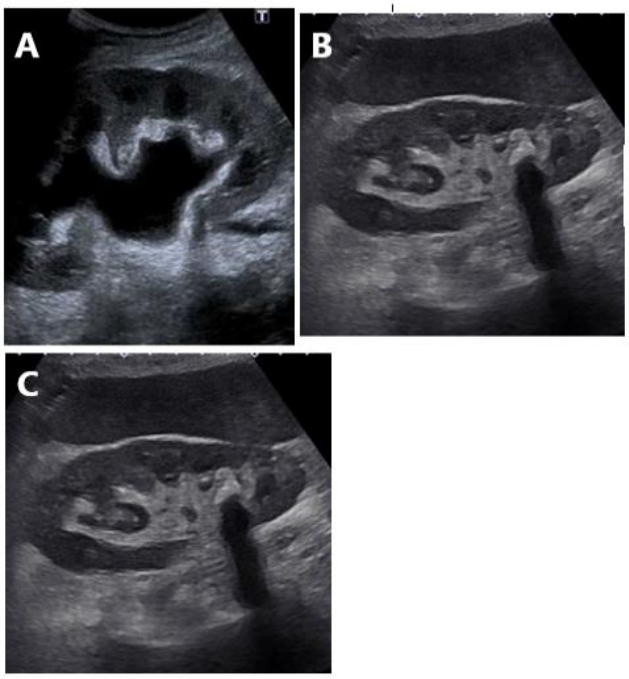


Fig. 38-11 Pathological kidneys showing: A, grade II, and B, grade IV obstructive nephropathy. C, a renal stone giving a shadow.
After Atlas of Ultrasound 2023 Erlangen Cookie-Richtlinien-EU.

(g) Spleen

APPROACH. Place the probe in the sagittal plane in the left posterior axillary line, and locate the spleen in front of the left kidney. It should be smaller (c.8cm) and brighter than the kidney and if it is larger, measure its size. Then look for a dilated portal vein. Look for micro-abscesses suggestive of TB.

(h) Retroperitoneal lymph nodes

APPROACH. Have your patient lying supine. It is best to perform this step right after you have done the scan of aorta and IVC.

METHOD. After you have evaluated the aorta and the IVC, direct your attention to potentially enlarged lymph nodes. Rock the transducer probe further to the left side until the IVC has vanished completely and perform a slow but steady movement with the probe back to the right side across and beyond the aorta.

Evaluate the space anterior and posterior to both vessels as well as the aortocaval space for ovoid space-occupying lesions.

Usually pathological lymph nodes are darker (hypoechoic) but there is no general rule and particularly lymph nodes which are enlarged due to metastasis of solid tumours have a very similar brightness as their surrounding tissue.

If the diameter of a lymph node is >1cm, if it is round and not oval, and if there is a group of suspicious lymph nodes, you should consider malignancy. Keep in mind that there are some structures which you can easily mistake for enlarged lymph nodes. Vessels can be differentiated easily if you perform a scan in two planes and tilt the transducer probe continuously: lymph nodes will appear and disappear while vessels can be tracked and join other vessels.

Sometimes bowel loops with content might look like a group of lymph nodes. It may help to apply rapid pressure impulses with the transducer to trigger peristalsis. It's especially important to keep anatomy in mind when scanning the superior retroperitoneum, as this is an area where you likely do find enlarged lymph nodes. Therefore be careful not to mistake either muscular extensions of the diaphragm, the oesophagus or the left renal vein for enlarged lymph nodes. Some lymph nodes are only detectable because they compress adjacent vessels, change their course or increase the distance between two structures, *e.g.* the retro-aortic distance should be <5mm.

If you have found any suspicious lymph nodes, try to describe in as much detail as possible what you have seen: *e.g.* how many nodes, what size, where exactly, any sign for abscess formation (anechoic centre). Using your knowledge of the lymphatic pathways, try to deduce where the site of the primary tumour might be. For example, in young men para-aortic nodes might suggest a testicular tumour. Additionally scan for hepatomegaly or splenomegaly.

Sonography is an appropriate technique to find suspicious lymph nodes but keep in mind that in a vast majority of your patients, these changes are due to inflammation and not to malignancy!

Make sure that you have screened the following typical locations in 2 planes:
(1) just below the diaphragm where the oesophagus passes through,
(2) at the coeliac axis,
(3) before, behind, beside and between aorta and IVC,
(4) where the common iliac artery splits into internal and external iliac arteries.

N.B. **To confirm the diagnosis of malignant lymph nodes**, look for:
(1) a round shape,
(2) loss of a brighter area at the hilum of the node,
(3) a longitudinal diameter >2cm.

(i) Urinary bladder

APPROACH. Apply the transducer in a sagittal orientation below the umbilicus right at the midline. While moving it down, rock the transducer head caudally. The urinary bladder will appear as an anechoic black triangle.

METHOD. If possible, the bladder should be filled to the maximum. If the patient is catheterized, clamp the catheter some time before you plan the examination. With an empty bladder, you will neither be able to evaluate the wall nor detect suspicious intra-luminal lesions.

Scan the urinary bladder completely in longitudinal, sagittal and transverse planes and observe the wall carefully for irregularities.

In chronic cystitis and schistosomiasis, you will find a diffuse wall thickening >5mm, whereas a tumour will present as a more localized swelling. Keep in mind that you might find a thickening of the entire wall due to chronic urinary bladder outlet obstruction too. To differentiate between these, it is useful to calculate the residual post-micturition volume remaining in the bladder. The simplified formula, $\frac{2}{3}(d^1d^2d^3)$, *i.e.* two-thirds the product of the three greatest diameters, is sufficient for your evaluation. Suspect a bladder outlet obstruction if the calculated volume is >100ml.

(j) Prostate

APPROACH. Apply the transducer in a sagittal orientation below the umbilicus right in the midline and rock the probe caudally while moving it down. In the sagittal as well as in the transversal section you will find the prostate just dorsal to the urinary bladder.

METHOD. Make sure that the urinary bladder is filled to the maximum before performing this examination. This makes it very easy to identify. While scanning the prostate in 2 planes, pay attention to its shape which is not exactly round but should be smooth on the surface. If the prostate is enlarged, it might elevate the floor of the urinary bladder.

To evaluate the enlargement of the prostate, measure the greatest transverse diameter from the transverse section, the greatest supero-inferior diameter from the sagittal section and the greatest antero-posterior diameter from either image plane. Use the simplified volume formula to calculate the volume. It should be ≤25ml.

Keep in mind that the grade of prostatic hypertrophy does not necessarily correlate with the symptoms the patient has. Expect prostate cancer if the prostate has invaded the urinary bladder or destroyed the continuity of the bladder wall. Usually, prostatic cancer appears darker than normal prostatic tissue.

(k) Uterus and ovaries

APPROACH. Ask your patient to have a full bladder for this examination. Apply the transducer in a longitudinal orientation caudal to the umbilicus right along the midline and rock it caudally. Her uterus will appear dorsal to the triangularly shaped urinary bladder. Also scan her uterus in a transversal orientation.

METHOD. The myometrium of the uterus should show a homogeneous pattern with a smooth surface. The brightness and width of the endometrium vary with her menstruation cycle. Right after her menstruation, you will only detect a bright reflex, whereas after ovulation the central reflex has disappeared and the endometrium appears hyperechoic throughout.

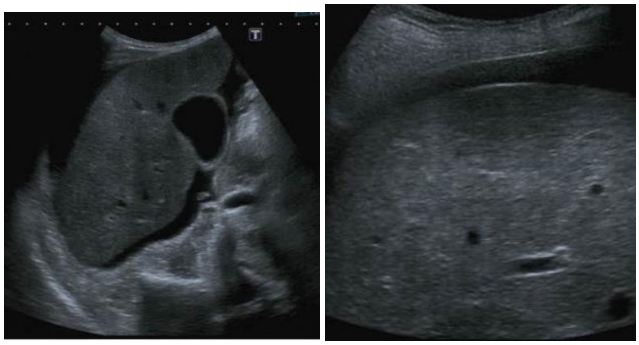
Common but benign findings are fibroids which usually display the same or less brightness as the myometrium. Sometimes fibroids contain calcifications or central necrosis. In these cases, a follow-up is necessary as they sometimes become sarcomas. Measure the size of a fibroid accurately to exclude rapid progression on her next visit and be sure that the fibroid is indeed part of the uterus and not a solid ovarian tumour which is in most cases not benign.

Using transabdominal ultrasound, ovaries will not be easy to find unless they are pathological. The most common findings are ovarian cysts which can grow to respectable size but are harmless. However, rapid growth, solid parts or septations within the cyst might be a sign of teratoma or malignancy and need further examination. An adnexal mass with solid and cystic components is likely to be an ectopic gestation; if it is more solid, it is probably a tumour, and if more cystic, an abscess. Bilateral cystic adnexae often imply endometriosis.

(l) Acute abdominal pain

APPROACH & METHOD. In much abdominal pathology you will find fluid in the abdominal cavity (38-12). Owing to the anatomy of the diaphragm, pelvis and spine, fluid will collect at typical sites: between liver and kidney, between kidney and spleen, dorsal to the urinary bladder.

ULTRASOUND SCAN FOR FREE FLUID



A **B**

Fig. 38-12 Free fluid around the liver, separating it from the diaphragm (A, top left. B, top). ***N.B. This amount of fluid is usually from a traumatic haemoperitoneum or ascites, but may be from generalized peritonitis.***

After Atlas of Ultrasound 2023 Erlangen Cookie-Richtlinien-EU.

Place the transducer on the right anterior axillary line as you would do to scan the right kidney. If possible, ask the patient to hold his breath in inspiration. If he is already intubated, ask the anaesthetist to stop ventilation for a moment.

Look for a blackish fringe between liver and kidney. The diaphragm appears superior to the liver as a bright thick curved line. Look for darkness beyond (superior to) it, indicating the presence of a pleural effusion.

Now place the transducer in a sagittal orientation right on the midline caudal of the umbilicus and rock the transducer caudally. You should now find the urinary bladder in its typical triangular configuration. Look for any collection of fluid behind the bladder or, in women, behind the uterus.

You may also find a circumscribed fluid collection anywhere in the abdomen, and even within the liver or spleen; this is likely to be pus.

You should also be able to spot abnormal masses (including an ectopic gestation) and para-aortic and mesenteric lymphadenopathy.

Look specifically for thickened structures, such as the gallbladder and its wall, or the appendix: if this is swollen >6mm, appendicitis is likely *but you should not rely on ultrasound appearances to diagnose appendicitis* (14.1).

(m) Neonatal skull

APPROACH. Have the setting for your young patient well prepared. There should be an infrared light over the examination table and the room should be as quiet as possible. Ask the parent to have the child fed about 1h before and to bring some toys which the baby is interested in. Although you will start the examination with the sector transducer, you will probably wish to switch to the linear transducer in between, in order to use its better near-field resolution.

The neonatal skull offers the unique opportunity to scan the brain, the ventricles and the subarachnoid CSF spaces. The anterior fontanelle serves as your acoustic window until its closure at the age of 18 months. Place the sector transducer in a sagittal orientation on the anterior fontanelle and tilt it slightly laterally.

METHOD. On the laterally tilted sagittal section you will get lots of information at once. At first sight, you will recognize the lateral ventricle as an anechoic curved fringe. Scan consecutively left and right. Attached to the caudal parts of the lateral ventricle you will find brighter areas representing the choroid plexus. The plexus should have a regular bright echogenicity, a smooth outline without any bulging.

Acute intracerebral haemorrhage presents hyperechoic, usually located near the ventricles, which makes it sometimes difficult to distinguish from normal choroid tissue.

Pay careful attention to the contour of the ventricles as CSF filled parenchymal defects near the ventricles might be a clue for an already resorbed intrauterine haemorrhage. Between the 24th and 32nd gestational weeks, the ependymal matrix, which covers the inside of the ventricles, proliferates. During these weeks, the premature neonate is extremely susceptible to intracerebral haemorrhage. But keep in mind that the contour of the ventricle might also change due to an obstructive (internal) hydrocephalus caused by other reasons than intracerebral haemorrhage.

Additionally you just might find little ventricular cysts which are benign as long as they do not impair the CSF circulation. Therefore, if you have found any evidence for alterations at or near the ventricles, you should evaluate the width of the ventricles and the subarachnoid CSF spaces in the coronal plane.

In the mid-coronal plane, you will easily find the connection between the 3rd ventricle and the lateral ventricles across the foramen of Munro: this structure looks like the letter 'Y'. Also in this plane the choroid plexus is displayed as a hyperechoic structure with a smooth outline best seen in the lateral ventricles. In this plane you can measure the width of the 3rd ventricle which should be <10mm in the neonate. If you suspect a non-communicating hydrocephalus, you do not only expect dilated ventricles but also narrowed subarachnoid CSF spaces due to ventricular extension.

If you find widened subarachnoid CSF spaces, you should think of cerebral atrophy instead. To measure the subarachnoid CSF space, it is an advantage to switch to the linear transducer for the best near-field resolution. Apply the transducer on the anterior fontanelle in the mid-coronal plane and determine the cranio-cortical distance which should be <4mm and the inter-hemispheric width which should be <6mm in the neonate.

(n) Bone

Fractures are easy to spot, particularly in ribs (where the bone is almost subcutaneous). This is especially useful anteriorly, where the ribs are cartilaginous and invisible on radiographs.

ULTRASOUND SCAN OF OSTEOMYELITIS

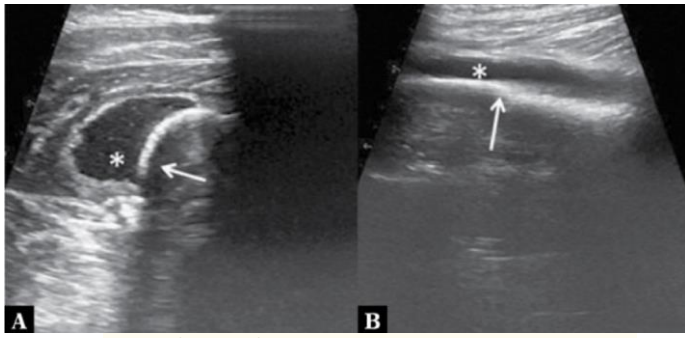


Fig. 38-13 Deep soft tissue fluid collection around bone. A, axial and B, sagittal sections through the distal thigh show deep fluid collection (white star) next to the distal femoral metaphysis (white arrow).
After Paliwal AK, Sahdev RS, Deshwal A, Ram B. Role of ultrasound in the diagnosis of paediatric acute osteomyelitis. J Ultrason 2021; 21(84):34-40.

Fluid around bone is also easily spotted, so can pick up a diagnosis of osteomyelitis after 3 days of infection. You can see adjacent increased vascularity also on Doppler.

(o) Separate tips

Confirming diagnosis of cysts:
(1) Almost round shape
(2) Sharp demarcation
(3) Anechoic content
(4) Posterior acoustic enhancement
(5) Accentuated entrance/exit echo.

To differentiate between a simple cyst and an abscess:
An abscess: whilst immature, an abscess will show internal echoes and sometimes a brighter rim. When the abscess matures, it will become more and more anechoic and at some point you will not be able to differentiate it from a cyst sonographically. If you can press on the cystic structure and elicit pain, it is more likely to be an abscess, but this is not wholly reliable, especially in patients with HIV-neuropathy.

A dilated vessel: scan the structure in both planes and figure out where it comes from and where it goes to. A vessel will always have some connection to another vessel.

(p) Normal values in adults

Appendix:	maximum size >6mm diameter
Gallbladder:	wall thickness <0·4cm before food maximal size 11x4cm
Common bile duct:	transverse diameter (wall to wall) <8mm *(but increases with age 0·1mm/yr)* *N.B. After a cholecystectomy, <10mm*
Common hepatic duct:	transverse diameter <5mm
Portal vein:	diameter 13-15mm
Aorta:	diameter <3cm
Kidney:	maximal size 12x6 cm cortical width >13mm
Prostate:	normal size 2x2x2cm (young man), growing with age
Urinary bladder:	wall thickness <0·4cm filled volume <750ml post-micturition volume <100ml

38.3 Ultrasound in pregnancy

Ultrasound is easily the most useful and reliable diagnostic tool in pregnancy, and you must learn to use it (38.2).

The gestational sac is visible at 4½wks, the yolk sac at 5wks, and the embryo at 5½wks. You can readily discern the position of the placenta (a thickening on the amniotic sac wall), the heartbeat, and the lie.

Dating the gestational age to check on the size and growth of the foetus is possible at 7-13wks by measuring the crown-rump length, from 13wks measuring the biparietal diameter (BPD, 2·4-9·5cm at term), and from 14wks the femur length (FL, 1·5-7·8cm at term). Also measure the abdominal circumference (AC): this gives a clue to polyhydramnios.

Make an estimate of foetal weight by using the formula:
Wt (g) = 1·4 x (BPD) x (FL) x (AC) – 200 (cm)

Multiple pregnancy and intra-uterine death are easy diagnoses.

You can readily diagnose certain foetal malformations, *viz.* hydrocephalus, anencephaly, meningomyelocoele, exomphalos, gastroschisis, duodenal atresia, cleft lip and palate, and cardiac abnormalities. An expert may detect many more features, however.

If there is a dark collection behind the placenta, this suggests an abruption.

A uterine rupture presents as a collapsed amniotic space and free fluid in the abdomen.

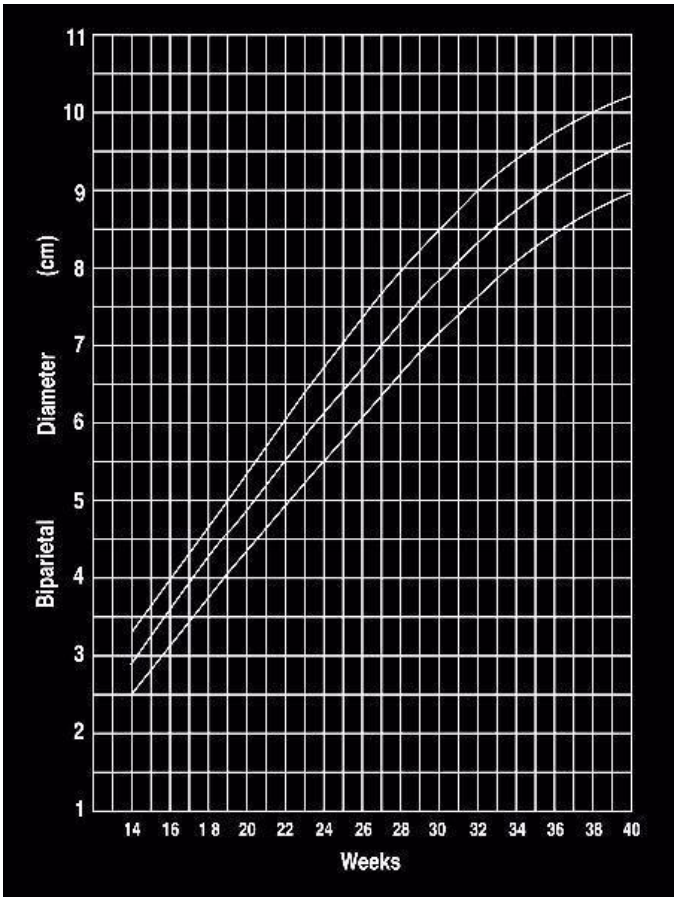


Fig. 38-14 CENTILES OF FOETAL BIPARIETAL DIAMETERS.

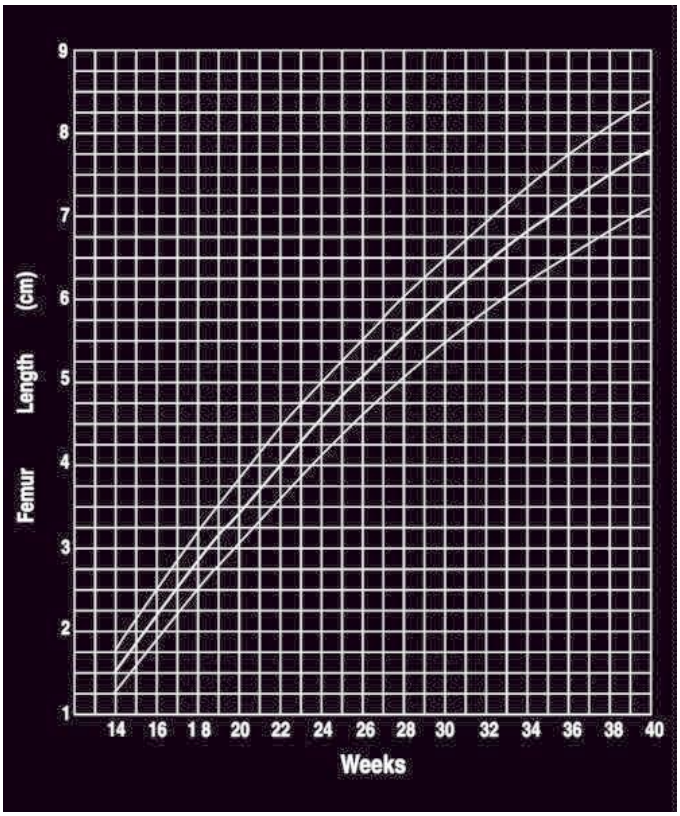


Fig. 38-15 CENTILES OF FOETAL FEMUR LENGTH.

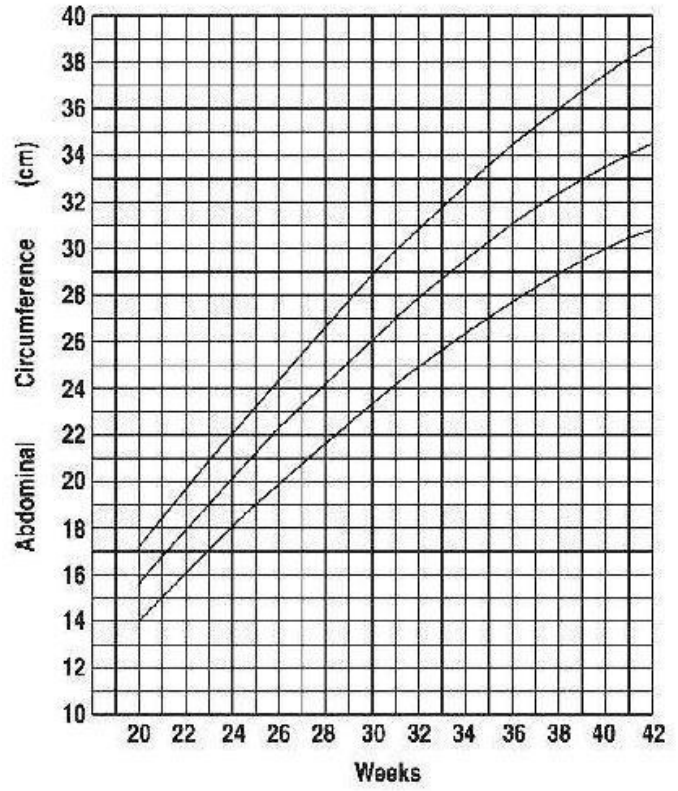


Fig. 38-16 CENTILES OF FOETAL ABDOMINAL CIRCUMFERENCE.

APPENDIX A: GRADES OF OPERATION

You can use this chart to monitor your progress, and to give you an idea of the relative difficulty of operations. Grades 1.1-1.5 describe simple procedures that you will definitely have to master. Grades 2.1-2.5 describe straightforward operations without serious difficulties or complications that would not pose much of a problem for basic surgical trainees. Grade 3 represents more difficult operations, with increasing complexity up to 3.5.

1.1 Suturing wounds (4.8)
Aspiration of pleural effusion (9.1)
Fine needle aspiration (17.2)
Rupturing of membranes (22.2)
Exploration of the cervix (22.11)
Proctoscopy (26.1)
Urethral catheterization (27.2)
Syringeing the ear (29.2)
Nasal packing (29.7)

1.2 Drainage of simple abscess (6.2)
Sigmoidoscopy (12.4)
Balloon tamponade of the uterus & vagina (22.11)
Cone & wedge biopsy of the cervix (23.8)
Trucut biopsy of breast (24.2)
Reduction of paraphimosis (27.30)
Curettage of tarsal cyst (28.12)
Removal of foreign body in the ear (29.6)
Extraction of foreign body in the nose (29.11)
Extraction of lower & upper teeth (31.3)
Anal dilation (33.6)
Excision of sebaceous cyst (34.3)
Excision of basal or squamous carcinoma (34.5)
Sclerotherapy of varicose veins (35.1)

1.3 Drainage of dental abscess (6.9)
Drainage of the hand (8.1)
Posterior colpotomy (10.3)
Microcurettage of the uterus (19.3)
Vacuum foetal extraction (21.6)
Repair fresh 3rd degree vaginal tear (21.15)
External cephalic version (22.7)
Marsupialization of Bartholin's cyst/abscess (23.5)
Lateral anal sphincterotomy (26.5)
Excision of perianal warts (26.6)
Needle prostatic biopsy (27.22)
Eyelid margin splitting (28.13)
Electrocautery for swollen nasal turbinates (29.9)
Drainage of nasal septal haematoma (29.9)
Subcutaneous Achilles tenotomy (32.8, 9)
Excision of melanoma (34.6)

1.4 Drainage peritonsillar abscess (6.7)
Drainage parotid abscess (6.10)
Drainage Ludwig's angina (6.11)
Drainage breast abscess (6.13)
Drainage axillary abscess (6.14)
Drainage anorectal abscess (6.17)
Drainage periurethral abscess (6.18)
Drilling for osteomyelitis (7.4)
Closed drainage for pleural effusion (9.1)
Liver aspiration (15.10)
Liver biopsy (15.11)
Tubal insufflation (19.3)
Dilation & curettage of uterus (23.4)
Urethral meatotomy (27.31)
Drain maxillary sinus (29.8)
Nasal polypectomy (29.10)
Cricothyroidotomy (29.15)
Cutback operation for anocutaneous fistula (33.6)
Full thickness rectal biopsy (33.7)
Z-plasty (34.2)

1.5 Drain retropharyngeal abscess (6.8)
Drain thyroid abscess (6.12)
Rectal drainage of pelvic abscess (10.3)
OGD (13.2)
Neck/axilla/groin lymph node biopsy (17.3)
Assisted breech delivery (22.7)
Manual removal of placenta (22.11)
Cystoscopy (27.3)
Drain frontal sinus (29.8)
Open biopsy nasopharynx (29.16)

2.1 Arthrotomy of minor joint (7.17)
Evacuation of an incomplete miscarriage (20.2)
Symphysiotomy (21.7)
Burns-Marshall manoeuvre (22.7)
Mauriceau-Smellie-Veit manoeuvre (22.7)
Thiersch operation (26.8)
Closed suprapubic cystotomy (27.7)
Eversion of hydrocoele (27.24)
Circumcision (27.29)
Excision of calcaneal spur (32.13)
Zadik removal of hallux nail bed (32.19)

2.2 Drainage penoscrotal abscess (6.21)
Suprapubic drainage pelvic abscess (10.3)
Parotid sialolithotomy (17.5)
Epigastric hernia repair (18.12)
McDonald's cervical suture (20.5)
Løvset manoeuvre (22.7)
Internal cephalic version (22.10)
Cervix cone biopsy (23.8)
Laying open fistula-in-ano (26.3)
Open suprapubic cystotomy (27.8)
Excision of hydrocoele (27.24)
Simple orchidectomy (27.26)
Tarsorrhaphy (28.10)
Release eyelid contracture (28.10)
Amputating toes (35.7)

2.3 Debridement necrotizing fasciitis (6.23)
Aspiration pericardium (9.2)
OGD + dilation oesophagus (13.2)
+ foreign body extraction (13.2)
+ stenting (13.2)
Drainage appendix abscess (14.1)
Minilaparotomy for TB (16.2)
Inguinal herniotomy in children (18.5)
Femoral hernia repair (18.7)
Umbilical hernia repair (18.10)
Laparoscopy (19.3)
Tubal ligation (19.4)
Intra-uterine foetal craniotomy (21.8)
Excision of breast lump (24.2)
Breast microdochectomy (24.3)
Thyroglossal cystectomy (25.3)
Haemorrhoidectomy (26.9)
Orchidopexy for torsion (27.25)
Subcapsular orchidectomy (27.26)

Radical eyelash excision (28.13)
Myringotomy (29.4)
Open extraction of aural foreign body (29.6)
Tracheostomy (29.15)
Carpal tunnel release (32.17)
Release of broad contractures (34.2)

2.4 Drainage iliac abscess (6.16)
Drainage prostatic abscess (6.19)
Drainage seminal vesicles (6.20)
Drainage pyomyositis (7.1)
Arthrotomy of medium joint (7.17)
Insertion Sengstaken tube (13.7)
Open drainage liver abscess (15.10)
Drainage pancreatic pseudocyst (15.14)
Inguinal herniorrhaphy (18.2)
Paraumbilical hernia repair (18.11)
Repair old 3rd degree vaginal tear (21.16)
Excision pilonidal sinus (26.10)
Orchidopexy for maldescended testis (27.27)
Corporo-spongiosum shunt (27.32)
Trabeculectomy & peripheral iridectomy (28.6)
Tarsal eversion (28.13)
Tonsillectomy (29.12)
Balloon dilation for achalasia (30.6)
Marsupialization dental cyst (31.6)
Tendon transfer for claw toes (32.12)

2.5 Sequestrectomy (7.5)
Excision of fibula (7.11)
Arthrotomy of hip (7.18)
Rib resection (9.1)
Submandibular sialadenectomy (17.7)
Incisional hernia repair (18.13)
Intra-uterine foetal decapitation/evisceration (21.8)
Ventrisuspension of uterus (23.12)
Subcutaneous mastectomy (24.6)
Cystolithotomy (27.16, 17)
Radical orchidectomy (27.26)
Cataract extraction (28.4)
Mastoidotomy (29.5)
Rigid bronchoscopy (29.14)
Rigid oesophagoscopy (30.2)
Millard repair cleft lip (31.7)
Saphenous nerve transfer (32.12)
Rotation flap (34.16)
Transposition flap (34.16)
Saphenous vein ligation, strip & avulsions (35.1)
Above/through/below-elbow amputation (35.4)
Transmetatarsal amputation (35.7)

3.1 Appendicectomy (14.1)
Laparotomy for splenic abscess (15.18)
Repair of ruptured uterus (21.17)
Mastectomy (24.5)
Urethrolithotomy (27.18)
Partial penectomy (27.33)
Reduction scrotoplasty (27.34)
Oesophageal dilation (30.3)
Amputation through wrist/carpus/metacarpus (35.4)
Amputation above/through knee (35.5)

3.2 Drainage retroperitoneal abscess (6.15)
Laparotomy for peritonitis (10.1)
Laparotomy for subphrenic abscess (10.2)
Closure small bowel perforation/bowel end (11.3)
Caecostomy (11.6)
Sigmoid colostomy (11.6)
Feeding jejunostomy (11.7)
Gastrostomy (13.9)
Block dissection inguinal nodes (17.8)
Lower segment Caesarean section (21.10)
Laparotomy for PID (23.1)
Oophorectomy (23.9)
Ovarian cystectomy (23.9)
Perineal rectosigmoidectomy (26.8)
Perineal urethrotomy (27.10)
Nephrostomy (27.14)
Ureterolithotomy middle third (27.15)
Evisceration of the eye (28.14)
Ligation of external carotid artery (29.7)
Stenting oesophageal stricture (30.5)
Cervical oesophagostomy (30.7)
Thigh-to-leg flap (34.9)
Amputation below knee (35.6)

3.3 Drainage extradural abscess (6.5)
End-to-end bowel anastomosis (11.3)
End-to-side, side-to-side anastomosis (11.4)
Ileostomy (11.6)
Transverse loop colostomy (11.6)
End colostomy (11.6)
Closing colostomy (11.6)
Closure burst abdomen (11.14)
Mesosigmoidoplasty (12.9)
Laparotomy for perforated duodenal ulcer (13.3)
Ramstedt's pyloromyotomy (13.6)
Cholecystectomy (15.8)
Cholecystojejunostomy (15.9)
Strangulated femoral hernia repair (18.8)
Vesico-vaginal fistula repair (simple) (21.18)
B-Lynch uterine suture (22.11)
Myomectomy (23.7)
Salpingo-oophorectomy (23.9)
Abdominal rectopexy (26.8)
Urethroplasty (23.6, 27.12)
Ureterolithotomy lower third (27.15)
Enucleation of the eye (28.14)
Retrograde oesophageal bouginage (30.3)
Costotransversectomy (32.4)
Closure of omphalocoele (33.4)
Ankle disarticulation (Syme's amputation) (35.7)
Vein graft (35.8)

3.4 Laparotomy for intestinal obstruction (12.4)
Ileocaecal resection (12.7)
Sigmoid colectomy (12.9)
Hartmann's operation (12.9)
Right hemicolectomy (12.11)
Ileotransverse anastomosis (12.11)
Pyloroplasty (13.5)
Gastrojejunostomy (13.8)
Laparotomy for perforated small bowel (14.3)
Laparotomy for enterocolitis (14.4)
Laparotomy for hydatid disease (15.12)
Pancreatic cystojejunostomy (15.14)
Laparotomy for pancreatic abscess (15.15)
Splenectomy (15.17)
Partial hysterectomy for ruptured uterus (21.17)
Ligation of uterine arteries (22.11)
Anterior colporrhaphy (23.13)
Posterior colporrhaphy (23.14)
Hysterectomy (23.15)
Open prostatectomy (27.20)

Bladder neck resection (27.21)
Exentration of the orbit (28.14)
Gastric oesophageal patch (30.7)
Tibialis posterior transfer (32.13)
Correction of hypospadias (1st stage) (33.9)
Ventriculo-peritoneal shunt (33.11)

3.5 Girdlestone excision arthroplasty of hip (7.19)
Exteriorization of bowel (12.9)
Closure Hartmann's operation (12.10)
Left hemicolectomy (12.11)
Laparotomy for bleeding peptic ulcer (13.5)
Oesophageal transection (13.7)
Gastrectomy (13.10)
Laparotomy for amoebiasis (14.5)
Choledochostomy (15.5)
Transduodenal Oddi sphincterotomy (15.5)
Roux-en-Y anastomosis (15.14)
Segmental splenectomy (15.17)
Laparotomy for abdominal gestation (20.9)
Thyroidectomy (25.7)
Complete penectomy (27.33)
Neonatal duodenoduodenostomy (33.3)
Resection of jejunal/ileal atresia (33.3)
Correction of hypospadias (2nd stage) (33.9)
Closure of meningomyelocele (33.10)
Excision of sacrococcygeal teratoma (33.13)

APPENDIX B: DIFFICULTIES WITH THE INDEX

If you have trouble looking things up, this section will probably help you.

Spelling follows the UK English tradition and usage rather than the American, so you will find 'appendicectomy', 'Caesarean', 'Centigrade', 'foetus', 'haemoglobin', 'hiccough', 'labour', 'leucocyte', 'oesophagus', 'operating theatre' and 'titre' rather than 'appendectomy', 'Cesarean', 'Fahrenheit', 'fetus', 'hemoglobin', 'hiccup', 'labor', 'leukocyte', 'esophagus', 'operating room' and 'titer'.

APPENDIX C: ABBREVIATIONS

Å	Ångström (=0·1nm)	(38.1)
AAFB	acid-alcohol fast bacilli (TB)	(5.6)
Abb.	abbreviation	(5.8)
ABC	abacavir	(5.8)
ABO	(system of blood groups)	(3.6)
AC	abdominal circumference	(38.3)
ACG	angle closure glaucoma	(28.6)
ACS	abdominal compartment syndrome	(11.10)
AGS	adrenogenital syndrome	(27.27)
AIDS	acquired immune deficiency syndrome	(5.1)
AIN	anal intra-epithelial neoplasia	(5.8)
am	morning (Latin: *ante meridiem*)	(21.3)
AMBU	artificial mechanical breathing unit	(2.1)
AMO	assistant medical officer	(1.3)
ANC	ante-natal clinic	(19.1)
AO	Association (for) Osteosynthesis	(7.5)
AP	antero-posterior	(7.6)
APH	ante-partum haemorrhage	(20.2)
APV	aprenavir	(5.8)
ARDS	acute respiratory distress syndrome	(3.6)
ARM	artificial rupture (of) membranes	(22.2)
ART	antiretroviral therapy	(5.1)
AZT	zidovudine	(5.8)
AZV	atanazavir	(5.8)
BB	borderline (leprosy)	(32.2)
BC	(year) before Christ	(26.3)
BCG	bacilli Camille Guerin (TB vaccine)	(5.5)
bd	twice daily (Latin: *bis die*)	(2.9)
BFAT	Bilharzia fixation antibody test	(27.36)
ß-HCG	human chorionic gonadotropin	(20.12)
BIO	binocular indirect ophthalmoscopy	(28.1)
BIPP	bismuth iodoform paraffin paste	(4.11)
BL	borderline lepromatous (leprosy)	(32.2)
BMVSS	Hindi: *Bhagwan mahaveer viklang sahavata samiti* (charitable organization providing services free of charge)	(35.6)
BP	blood pressure	(3.4)
BPD	biparietal diameter	(38.3)
BRS	basic radiological system	(1.12)
BSO	bilateral salpingo-oophorectomy	(23.8)
BT	borderline tuberculoid (leprosy)	(32.2)
BXO	balanitis xerotica obliterans	(27.31)
c.	about (Latin: *circa*)	(3.4)
°C	degree Centigrade	(10.1)
CAH	congenital adrenal hyperplasia	(33.13)
cc	cubic centimetre (=ml)	(22.2)
CD4	cluster differentiation (glycoprotein)	(5.3)
CDH	congenital dislocation (of the) hip	(32.14)
CF	count fingers	(28.1)
Ch	Charrière (gauge)	(4.9)
CHOP	cyclophosphamide, hydroxydaunorubicin (doxorubicin), oncovin (vincristine), prednisolone	(17.6)
cm	centimetre	(2.4)
CMV	cytomegalovirus	(5.6)
CNS	central nervous system	(5.7)
CPD	cephalopelvic disproportion	(21.4)
CRL	crown-rump length	(20.1)
CSF	cerebrospinal fluid	(17.6)
CVP	central venous pressure	(10.1)
D&C	dilation and curettage	(10.2)
dB	decibel	(29.2)
DCIS	ductal carcinoma *in situ*	(24.2)
ddC	zalcitabine	(5.8)
ddI	didanosine	(5.8)
DIC	disseminated intravascular coagulation	(2.5)
dip	distal interphalangeal	(7.18)
dL	decilitre	(1.5)
DLV	delavirine	(5.8)
DNA	deoxyribonucleic acid	(5.2)
DOTS	directly observed treatment scheme	(5.7)
DRV	darunavir	(5.8)
d4T	stavudine	(5.8)
DUB	dysfunctional uterine bleeding	(20.6)
DVT	deep vein thrombosis	(6.22)
EBV	Epstein-Barr virus	(17.6)
ECG	electrocardiogram	(2.1)
ECV	external cephalic version	(22.7)
ed	editor, edition	(6.6)
EFV	efavirenz	(5.8)
e.g.	for example (Latin: *exempli gratia*)	(1.11)
ELISA	enzyme-linked immunosorbent assay	(5.1)
EMF	endomyocardial fibrosis	(9.2)
ENL	erythema nodosum leprosum	(32.2)
ENT	ear nose throat	(2.12)

EPI	extended programme (of) immunization (WHO)	(32.7)
ESR	erythrocyte sedimentation rate	(5.6)
ET	endotracheal	(2.1)
et al	and others (Latin: *et alia*)	(5.3)
ETR	etravirine	(5.8)
FDI	World Dental Federation (French: *Fédération dentaire internationale*)	(31.1)
FFP	fresh frozen plasma	(3.1)
FGM	female genital mutilation	(6.21)
Fig.	figure	(1.1)
FL	femur length	(38.3)
FO	fibreoptic	(29.13)
Fr	French (gauge)	(9.1)
FSH	follicle stimulating hormone	(19.1)
FTC	emtricitabine	(5.8)
5FU	5-fluorouracil	(34.5)
g	gram	(2.9)
G	gauge	(4.6)
	pregnant (Latin: *gravidus*)	(20.2)
G6PD	glucose 6-phosphatase dehydrogenase deficiency	(33.8)
GA	general anaesthetic	(1.4)
GI	gastrointestinal	(13.4)
GIST	gastrointestinal stromal tumour	(13.5)
GnRH	gonadotropin-releasing hormone	(24.3)
GTD	gestational trophoblastic disease	(23.10)
GUL	gestation of unknown location	(20.1)
h	hour	(3.4)
Hb	haemoglobin	(10.1)
HBV	hepatitis B virus	(2.6)
HCV	hepatitis C virus	(2.6)
HELLP	haemolysis, elevated liver (enzymes), low platelet (syndrome)	(22.2)
HIV	human immunodeficiency virus	(1.2)
HM	hand movements	(28.1)
HPV	human papilloma virus	(23.8)
HRE	isoniazid, rifampicin, ethambutol	(5.7)
HRZE	isoniazid, rifampicin, pyrazinamide, ethambutol	(5.7)
HSG	hysterosalpingogram	(19.3)
I	(Latin: 1)	(1.4)
ICD-10	international classification (of) diseases (10th version)	(19.1)
ICU	intensive care unit	(1.8)
I&D	incision and drainage	(2.12)
IDV	indinavir	(5.8)
i.e.	that is (Latin: *id est*)	(5.6)
Ig	immunoglobulin	(5.2)
II	(Latin: 2)	(1.4)
III	(Latin: 3)	(1.4)
IM	intramuscular	(2.11)
IOP	intra-ocular pressure	(28.1)
ip	inter-phalangeal	(8.15)
IPD	inter-pupillary distance	(28.1)
IR	infrared radiation	(28.1)
IU	international unit	(22.2)
IUD	intra-uterine device	(5.3)
IUGR	intra-uterine growth retardation	(19.3)
IV	intravenous	(1.8)
	(Latin: 4)	(1.4)
IVC	inferior vena cava	(38.2)
IVU	intravenous urogram	(27.5)
IX	(Latin: 9)	(1.4)
JHPIEGO	Johns Hopkins Programme of International Education in Gynaecology & Obstetrics	(19.5)
K	potassium (German: *kalium*)	(3.6)
	Kirschner (fixation)	(32.14)
Kcal	kilocalories	(12.4)
KCl	potassium (German: *kalium*) chloride	(1.8)
Ket	ketamine	(2.12)
kg	kilogram	(2.5)
kHz	kilohertz	(29.2)
KP	keratitic precipitates	(28.3)
KS	Kaposi sarcoma	(34.10)
kV	kilovolt	(38.1)
kW	kilowatt	(2.5)
L	litre	(1.8)
LA	local anaesthetic	(1.8)
lb	pound (Greek: *libra*)	(2.5)
LED	light-emitting diode	(1.1)
LGV	lymphogranulopma venereum	(23.17)
LH	luteinizing hormone	(19.1)
LL	lepromatous (multibacillary) leprosy	(32.2)
LMICs	low- (&) middle-income countries	(19.1)
LPV	lopinavir	(5.8)
m	metre	(38.1)
mA	milliampere	(38.1)
mcp	metacarpo-phalangeal	(7.18)
mg	milligram	(1.8)
µg	microgram	(13.1)
MHz	megahertz	(3.3)
min	minute	(1.8)
mIU	$^{1}/_{1000}$ of an IU	(22.2)
MJ	megajoule	(12.4)
ml	millilitre	(1.5)
µl	microlitre	(5.7)
M&M	morbidity & mortality	(1.3)
mm	millimetre	(2.5)
mM	millimolar (millimoles per litre)	(1.8)
µm	micrometre	(34.7)
µM	micromolar (micromoles per litre)	(33.8)
mmol	millimole	(10.1)
MMR	maternal mortality ratio	(1.1)
MOH	Ministry of Health	(19.1)
MOPP	mustine, oncovin (vincristine), procarbazine, prednisolone	(17.6)
MRI	magnetic resonance imaging	(7.3)
MSICS	manual small incision cataract surgery	(28.4)
mtp	metatarso-phalangeal	(32.12)
MTX	methotrexate	(23.10)
MU	mega-unit	(2.9)
MUA	manipulation under anaesthetic	(2.12)
MVA	manual vacuum aspiration	(19.1)
NaCl	sodium (German: *natrium*) chloride	(1.8)
N.B.	note well (Latin: *nota bene*)	(2.4)
NFV	nelfinavir	(5.8)
NG	nasogastric	(4.9)
nm	nanometre	(38.1)
nil	nothing (Latin: *nihil*)	(13.3)
NNRTI	non-nuclease reverse transcriptase inhibitor	(5.8)
nocte	at night (Latin)	(37.2)
NPL	no perception (of) light	(28.1)
NRTI	nuclease reverse transcriptase inhibitor	(5.8)
NSAID	non-steroidal anti-inflammatory drug	(13.1)
NVP	nevirapine	(5.8)
OA	occipito-anterior	(22.2)
od	once daily (Latin: *omni die*)	(2.9)
OGD	oesophago-gastro-duodenoscopy	(13.2)
OP	occipito-posterior	(22.2)
p.	page	(6.12)
P	born (Latin: *para*)	(20.2)

PA	postero-anterior	(9.1)
PAIR	puncture, aspiration, injection, re-aspiration	(15.12)
p_aO_2	arterial pressure of oxygen	(32.2)
Pap	Papanicolau	(23.3)
PCR	polymerase chain reaction	(5.7)
PDS	polydioxanone sulphate (suture)	(4.6)
PEP	post-exposure prophylaxis	(5.8)
pH	logarithmic value of acidity	(5.4)
PI	protease inhibitor	(5.8)
PID	pelvic inflammatory disease	(5.6)
pip	proximal interphalangeal	(7.18)
P-L	Proctor-Livingstone	(30.2)
PL	perception (of) light	(28.1)
pm	afternoon (Latin: *post meridiem*)	(21.3)
PMB	postmenstrual bleeding	(19.1)
PMMA	polymethylmethacrylate	(28.4)
PNM	perinatal mortality	(20.1)
PNR	perinatal mortality rate	(20.1)
PO	by mouth (Latin: *per os*)	(1.8)
POAG	primary open-angle glaucoma	(28.6)
POP	plaster of Paris	(7.11)
POR	post-operative radiotherapy	(23.8)
PPH	post-partum haemorrhage	(22.11)
PPI	proton pump inhibitor	(13.1)
ppm	parts per million	(5.4)
PR	*per rectum*	(1.11)
PRN	as required (Latin: *pro re nata*)	(2.12)
PROM	premature rupture of membranes	(22.4)
PSA	prostate specific antigen	(5.6)
psi	pounds (per) square inch	(2.4)
PV	*per vaginam*	(19.5)
qds	4 times daily (Latin: *quater die sumendus*)	(2.9)
Rh	Rhesus (blood type: D)	(3.6)
RPR	rapid plasma reagin (syphilis test)	(24.3)
RNA	ribonucleic acid	(5.2)
RTV	ritonavir	(5.8)
RVF	recto-vaginal fistula	(21.10)
SC	subcutaneous	(2.11)
sec	second	(21.3)
SIV	simian immunodeficiency virus	(5.2)
SQV	saquinavir	(5.8)
stat	immediately (Latin)	(10.4)
STI	sexually transmitted infection	(23.1)
TAH	total abdominal hysterectomy	(23.8)
TB	tuberculosis	(5.7)
TBA	traditional birth attendant	(19.1)
3TC	lamivudine	(5.8)
TDF	tenofovir	(5.8)
tds	3 times daily (Latin: *ter die sumendum*)	(2.9)
TFR	total fertility rate	(19.3)
Thio	thiopentone	(2.12)
TL	tubal ligation	(19.4)
TNM	tumour node metastasis (staging)	(24.4)
TOS	trial of scar	(21.13)
TSH	thyroid-stimulating hormone	(25.6)
TT	tuberculoid (paucibacillary) leprosy	(32.2)
U	unit	(20.4)
US	ultrasound	
USA	United States (of) America	(5.1)
UV	ultraviolet	(28.1)
V	(Latin: 5)	(1.4)
	volt	(28.1)
	living (Latin: *vivo*)	(20.2)
VBAC	vaginal birth after Caesarean	(21.13)
VDRL	venereal disease research laboratory (syphilis test)	(24.3)
-ve	negative	(3.6)
+ve	positive	(3.6)
VF	ventricular fibrillation	(9.2)
VI	(Latin: 6)	(1.4)
VII	(Latin: 7)	(1.4)
VIII	(Latin: 8)	(1.4)
viz	namely (Latin: *videlicet*)	(1.8)
vol	volume	(3.1)
VP	ventriculo-peritoneal	(33.3)
VT	ventricular tachycardia	(9.2)
VVF	vesico-vaginal fistula	(1.1)
WHO	World Health Organization	(1.1)
wk	week	(1.6)
X	(Latin: 10)	(1.4)
XI	(Latin: 11)	(1.4)
XII	(Latin: 12)	(1.4)
XIII	(Latin: 13)	(1.4)
XIV	(Latin: 14)	(1.4)
XIX	(Latin: 19)	(1.4)
X-ray	radiograph *(loosely used)*	(1.7)
XV	(Latin: 15)	(1.4)
XVI	(Latin: 16)	(1.4)
XVII	(Latin: 17)	(1.4)
XVIII	(Latin: 18)	(1.4)
XX	(Latin: 20)	(1.4)
yr	year	(1.3)
Y-V	plasty (changing shape of Y to V)	(13.5)
ZN	Ziehl Neelsen (stain)	(5.7)